

CONTENTS

1.	IMA BATHINDA-2018	1
2.	COMMON PEDIATRIC EMERGENCIES	3-6
3.	ACUTE POISONING – MANAGEMENT GUIDELINES	7-11
4.	FOREIGN BODY IN AERODIGESTIVE PASSAGE	12-16
5.	EMERGENCY AIRWAY MANAGEMENT	17-18
6.	DIABETIC EMERGENCIES	19-21
7.	MANAGEMENT OF SHOCK: HAEMORRHAGIC HYPOVOLEMIC SHOCK	22-27
8.	EMERGENCIES IN CARCINOMA CERVIX	28-29
9.	HYPERTENSIVE EMERGENCIES	30-32
10.	MEDICO-LEGAL ASPECTS OF SUDDEN UNEXPECTED DEATH, DIFFICULT SITUATIONS IN MEDICAL PRACTICE, BROUGHT DEAD, POST MORTEM	31-43
11.	APPROACH TO THE CANCER PATIENT	44
12.	EMERGENCY MANAGEMENT OF ACUTE UPPER GI BLEEDING	45-47
13.	POLY-TRAUMA	48
14.	TRIPPLICATE URETER WITH CONTRALATERAL DUPLICATION ALONG WITH MULLERIAN DUCT ABNORMALITIES IN A CONGENITALLY INCONTINENT GIRL	49
15.	CHALLENGES IN CARDIO-THORACIC SURGERY	50

SCIENTIFIC / ACADEMIC PROGRAM IMA-EMERGE BATHINDA

MORNING SESSION

9.00 - 9.20 am	Common Paediatric Emergencies	Dr. Anjali Bansal
9.25 - 9.45 am	Poisoning an Emergency	Dr. Amit Taneja
9.50 - 10.10 am	Common ENT Emergencies	Dr. Grace Budhiraja
10.15 - 10.45 am	Emergency Airway Management	Dr. Minnu Panditrao
10.50 - 11.30 am	Diabetic Emergencies	Dr. Bharat Bhushan Jindal

INAUGURATION

12.00 - 12.25 pm	Shock Management	Dr. Mridul Panditrao
12.25 - 12.45 pm	Emergencies in Obst. & Gynae	Dr. Arvind Sharma
12.50 - 1.30 pm	Cervical Cancer its Vaccine & Emergencies	Dr. Rupinder Sekhon
1.35 - 1.55 pm	Hypertensive Emergencies	Dr. Aman Salwan

LUNCH

2.30 - 2.55 pm	Medico-legal Aspects of Practice	Dr. Mahesh Baldwa
3.00 - 3.35 pm	Approach to a Cancer Patients	Dr. Anish Maru
3.40 - 4.00 pm	Head Injury	Dr. Dinesh Gupta
4.00 - 4.20 pm	Acute G.I. Bleeding	Dr. Gursewak Singh
4.20 - 4.40 pm	Poly-Trauma	Dr. G.S. Maan
4.40 - 5.00 pm	Financial Aspect of Medical Practice	Mr. Suresh Goyal

IMA BATHINDA-2018

Dear Friends,

Greetings

I take this privilege of presenting activities held by IMA, Bathinda-2018 in last 6 months.

First of all Medical bandh was announced by the Indian Medical Association (IMA) in protest against the provisions in the National Medical Commission Bill on 2nd January, 2018 and IMA Bathinda took active participation with complete caseation of work & presented memorandum to Deputy Commissioner.

On 14th Jan IMA organised Lohri function at Dune's Club which was attended by all IMA members with full zeal.

PIMACON was attended by IMA members Bathinda at Hoshiarpur on 20th and 21st January with full zest and vigour. Annual State council meeting was also attended on the same event on 20th Jan 2018.

Pulse polio booth was organised by IMA Bathinda on 28th Jan 2018, at Bibiwala Chowk, Bathinda in which IMA members took active participation by even going to homes personally for the immunisation program.

IMA Bathinda also hosted a Family HOLI Function on 2'nd March at Teshwar Garden Cricket Academy. Cricket Match Exclusively for ladies, traditional games with softer balls like PITHU SATOLIYA and Tug of war for masculine members and fairer gender too were held and enjoyed by all.

On 20th March 2018 Dr. Ravi Wankhedkar, National President IMA, started his Punjab Yatra from Bathinda as a part of IMA Bharat Yatra, He also addressed gathering of members of all the IMA's of Malwa Region (Bhucho, Rampura, Tappa, Rama Mandi, Goniana, Jaito, Giderbaha, Malout East/West, Abohar & Fazilka) he Elaborated how impact of NMC Bill is going to ruin the practice of Medical Fraternity. It was a Proud moment as history was created because for the first time a National President of IMA visited Malwa region at Bathinda. He was also accompanied by Dr. P. S. Bakshi (IMA National Vice President), Dr. Jatinder Kansal (Punjab IMA President), Dr Navjot Dahiya (Punjab IMA Secretary) and Dr Rajan Sharma (Director, IMA A.K. Sinha Institute). IMA Presidents and Secretaries from Malwa region attended the meeting.

For the cause of IMA to be fought at National level a fund of Rs One Lakh was also given at the IMA Meeting to

National President from IMA Bathinda. IMA RUNNING CRICKET TROPHY was also unveiled by IMA National President Dr. Ravi Wankhedkar on the same day.

IMA MAHAPANCHAYAT at Indira Gandhi Indoor Stadium, New Delhi was attended by office bearers & members of IMA Bathinda on 25th March 2018 against NMC Bill.

Installation of Grievance and Redressal Committee IMA Bathinda after decision of IMA Executive Committee meet at Dunes Club Bathinda.

IMA Cycle awareness rally against NMC Bill to Connect with the society was organised at Bathinda which also included students from Adesh Medical College spreading awareness against the Draconian NMC Bill on 11th March 2018.

IMA Inter District Cricket Match with Team Patiala was held on 11th March, 2018 at Paitiala which was attended with full zest and vigour.

IMA Bathinda Sports Meet started on 14th March and continued upto 1st April 2018 and was inaugurated by our Hon'ble Civil Surgeon Dr. H.N.Singh various games were held which included Cricket at Teshwar Sports Academy and Badminton, Carrom, Chess at Civil Lines Club, Bathinda.

IMA PUNJAB Annual state Council Meeting was held at Jalandhar which was attended by Team IMA.

IMA members specially Physicians and Pediatricians attended "JAGO" organised by District Administration and Hon'ble Civil Surgeon Bathinda on 28th April, 2018.

A cycle rally was organized by members of the Indian Medical Association (Bathinda) in collaboration of the Punjab Medical Representatives' Association and the District Health Department to spread awareness regarding the measles rubella (MR) campaign in the city on 6th May, 2018.

IMA Inter District Cricket match with Team Patiala was held on 2nd June, 2018 at Cops Club, Bathinda.

IMA Bathinda is organising a Conference & Workshop on Emergencies in Medical & Surgical field and Advanced cardiac life support on 23'rd & 24' th June.

Dr. Parneet Singh Brar
Secretary
IMA, Bathinda



IMA-EMERGE



Conference & Workshop BATHINDA

Conference :

**Medical &
Surgical Emergencies**

Workshop :

Advance Cardiac Life Support

Date :

23rd & 24th June, 2018

Venue :

**Hotel Three Palms
Goniana Road, Bathinda**

Chief Guest

S. Manpreet Singh Badal

Hon'ble Finance Minister, Govt. of Punjab

Guest of Honour

Dr. Rishipal Singh

Commissioner, Municipal Corporation, Bathinda

Guest of Honour

Sh. Diprava Lakra (I.A.S.)

Deputy Commissioner, Bathinda

Guest of Honour

Dr. H.N. Singh

Civil Surgeon, Bathinda

Organizing Chairmen

Dr. Vikas Chhabra (President IMA)

Dr. Arvind K. Sharma (Incharge, Acad. Committee)

Dr. Mridul Panditrao, Dean Academics, AIMS, Bathinda

Secretary

Dr. Parneet Singh Brar

Finance Secretary

Dr. Deepak Bansal

Chairman

Dr. Shiv Dutta Gupta

Vice President

Dr. S.K. Verma

Dr. Saurabh Gupta

Dr. Amit Aggarwal

Joint Secretary

Dr. Prem Verma

Dr. Ashwani Goyal



**DONATE
BLOOD
& SAVE LIFE**



COMMON PEDIATRIC EMERGENCIES

Dr. Anjali Bansal
M.D Paediatrics
Civil Hospital, Bathinda.

- Aim and objectives. Examine the unique challenges associated with the provision of emergency services to children and adolescents
- To understand difference in the management between adults and children
- The child's airway is smaller than an adults and can obstruct a lot more easily.
- Children have a smaller circulating blood volume than an adult.
- An infants head is larger in proportion to the rest of the body than an adults. (Heat Loss)!
- Solid abdominal organs are relatively larger in children compared with adults, there is an increased risk of direct organ injury following blunt or penetrating forces.

PRESENTATION OF EMERGENCIES

Upper and lower respiratory which includes bronchiolitis, croup, asthma, pneumonitis.

Cardiac causes includes arrhythmias and CHF.

Neurological causes includes coma which can be due to different causes.

Metabolic causes includes DKA, hypoglycemia , electrolyte imbalances Renal, poisoning ,drowning, surgical emergencies including acute abdomen

Newborn emergencies Six key, generic skills are expected of all health professionals involved with the care of sick children. These are:

1. To recognise the critically sick or injured child;
2. To initiate appropriate immediate treatment;
3. To work as part of a team;
4. To maintain and enhance skills;
5. To be aware of issues around safeguarding children;
6. To communicate effectively with children and carers.

PAEDIATRIC RESPIRATORY EMERGENCIES

Acute respiratory illnesses are one of the most common reasons for presentation of children to hospital. Infants with respiratory illness are at greater risk from rapid deterioration due to their relatively higher oxygen consumption, smaller functional residual capacity and greater airway resistance. The cartilaginous components of their airway are softer and render the airway prone to dynamic obstruction with increased negative pressure as it occurs in respiratory distress Due to unfavourable respiratory mechanics, children develop respiratory failure quickly due to muscle fatigue.

A comprehensive assessment of the degree of respiratory distress with a thorough history and careful examination is must. Evidence of increased respiratory effort includes tachypnea, accessory muscle use, intercostal, subcostal or suprasternal recession, and abnormal sounds such as stridor, wheeze or grunting. Difficulty in talking or feeding is also significant. Diminished breath sounds may be evidence of impending respiratory arrest and can be due to obstruction or exhaustion. Further evidence of hypoxia and respiratory distress should be sought by continuous pulse oximetry and also reduced conscious state, agitation and confusion. Cyanosis is often difficult to detect and its absence does not rule out respiratory failure.

The management of the child with severe respiratory disease should follow a structured approach with sequential attention to airway, breathing and circulation.

STRIDOR

The most common cause of stridor in children is viral croup (acute viral laryngotracheobronchitis). Less common causes include non-infectious (spasmodic) croup and an upper airway foreign body (AFB). Rarer causes of stridor in the absence of trauma or burns include tonsillitis, anaphylaxis, tracheitis, diphtheria and epiglottitis.

CROUP

Croup is a common childhood respiratory illness, with a peak incidence in the age range of 6 months to 3 years. It is estimated to affect 3% of children younger than 6 years annually [1]. Classically, croup presents with a barking cough, that may be preceded by mild fever and runny nose. The cough often appears suddenly and stridor and respiratory distress may follow. Fewer than 5% of patients with croup require admission to hospital and approximately 1% require tracheal intubation.

The management of severe croup is initially supportive. The child should be kept with the parent or carer, unnecessary distress should be avoided, continuous oximetry commenced and oxygen applied as tolerated. A child who requires oxygen with an upper airway obstruction must be monitored carefully for signs of respiratory failure as desaturation is usually a late feature.

Nebulised adrenaline improves symptoms within 30 min, but this effect will wear off by 2 h [2]. Advanced Paediatric Life Support (APLS) teaching recommends a dose of 0.5 ml/kg of 1:1000 adrenaline up to a maximum of 5 ml and this may be repeated [3]. Glucocorticoids improve croup symptoms within 6 h of administration and this effect will last approximately 12 h [4]. Steroids have been shown to reduce the need for tracheal intubation. Oral dexamethasone (0.15 mg/kg) or prednisolone (1 mg/kg) is recommended.

If the child remains in significant respiratory distress, has signs of impending exhaustion and has evidence of hypoxia in room air, then tracheal intubation should be considered.

Tracheal intubation within the first hour of presentation is only required in the minority of cases; thus there may be time for adequate assessment and for steroid therapy to take effect.

AIRWAY FOREIGN BODY

An AFB is a potentially life-threatening event and a very common cause of non-intentional injury in children. The mean age is 3 years and over 50% of cases are younger than 2 years of age. It was previously thought that death is extremely rare once the child with an AFB reaches hospital, but recent analysis of nearly 3000 cases of children admitted to hospital with airway obstruction caused by a AFB showed a hospital mortality rate of 3.4% . The

presenting symptoms vary according to the size, position and mobility of the foreign body and the time of inhalation. Usually there is a history of a choking episode if the incident was witnessed. Cough, wheeze and dyspnoea may also be present. Upper tracheal or glottic foreign bodies may present with stridor and/or voice change. Later presentations may include fever, productive cough and progressive respiratory distress. Auscultation of the chest may reveal localising signs. Stable symptoms and signs usually indicate that the foreign body is not mobile. A history of fluctuating symptoms such as intermittent stridor may be an indicator of a mobile foreign body, which can be life-threatening. Of all the presenting symptoms and signs, a choking episode has the highest sensitivity and specificity for an AFB.

In the absence of acute obstruction and severe symptoms, a chest X-ray may assist in diagnosis, although most foreign bodies are organic and not radiopaque. Comparison of inspiratory and expiratory films may assist the diagnosis of a bronchial foreign body; hyperinflation of the affected side, due to air trapping beyond the foreign body, is seen on the expiratory X-ray.

The initial management of AFB is aimed at determining the severity and this is simply done by assessing the child's ability to cough. The presence of an effective cough indicates mild airway obstruction and coughing may result in clearance of an upper AFB. An ineffective cough indicates severe obstruction and if the child is conscious, with mild airway obstruction, then the child should be encouraged to cough with the aim of expelling the foreign body. If stable, he/she requires close monitoring, but no further treatment is necessary until appropriate surgical assessment can be arranged[3].

The definitive treatment of an AFB is removal via rigid bronchoscopy. If the child presents at a hospital that cannot provide this service, then the child will require transfer to a specialist centre that can. The child should be transported with a medical escort who can provide appropriate resuscitation.

EPIGLOTTITIS

The introduction of the Haemophilus influenzae type-b (Hib) vaccination has seen a 10-fold reduction in epiglottitis presentations [5]. Therefore, epiglottitis is now rare. It is important to differentiate epiglottitis from other causes of stridor because tracheal

intubation in the child with epiglottitis can be difficult due to glottic obstruction.

The presence of stridor and drooling is predictive of epiglottitis whereas stridor and coughing strongly suggests croup. Other reliable signs of epiglottitis are a preference to sit, refusal to swallow and dysphagia.

Airway management of the child with epiglottitis involves preparation for a difficult intubation, with ENT surgical backup, careful inhalational induction, maintenance of spontaneous ventilation and intubation using a tracheal tube one size smaller than usual. Antibiotic treatment with a third-generation cephalosporin, following swabs and blood cultures, is recommended.

STATUS ASTHMATICUS

The presentation of status asthmaticus in children is variable. In most cases, there is a previous history of asthma and evidence of respiratory distress with cough, wheeze and increased work of breathing. The degree of wheeze does not correlate well with the severity of asthma. An absence of breath sounds (a 'silent chest') may indicate minimal air entry and impending respiratory arrest. Other evidence of impending arrest may include restlessness, confusion, inability to speak, inability to lie down, pulsus paradoxus, hypercapnia and hypoxia [6].

Oxygen therapy should be commenced if not already. Inhaled β -adrenoceptor agonists and corticosteroids continue to be the mainstays of treatment for severe asthma. Cochrane review has concluded that the addition of aminophylline does not achieve further bronchodilation and results in more adverse events [7]. Intravenous magnesium sulphate has been shown to be effective in severe asthma.

Intubation and mechanical ventilation should be avoided if possible due to the risk of barotrauma, cardiovascular collapse and aggravation of bronchospasm. Non-invasive positive pressure ventilation has been shown to be effective in children and may reduce the need for intubation [8].

SHOCK

The different types of shock can be hypovolemic shock due to diarrhoea, dehydration, acute blood loss due to surgery or injury, septic shock which can be further of warm or cold type, cardiogenic shock,

anaphylactic shock & neurogenic shock. The primary aim is to identify the type of shock and treat accordingly. Early fluid resuscitation remains the mainstay in each type of shock. The use of vasopressors depends on the type of shock due to their selective actions on various receptors. The clinical diagnosis of septic shock in a child with suspected infection can be made if the child has hypothermia or hyperthermia and clinical signs of inadequate tissue perfusion including any of the following; decreased or altered mental status; prolonged capillary refill (>2 s); diminished pulses; cool peripheries; bounding peripheral pulses; wide pulse pressure; or decreased urine output. Hypotension may not always be present [9].

Both PALS teaching and the American College of Critical Care Medicine (ACCCM) recommend the circulation, airway & breathing (CAB) approach to management of septic shock, with the goals of resuscitation being maintenance of adequate circulation, restoration of airway, oxygenation & ventilation. [3,9]. Boluses of 20 ml/kg isotonic fluid should be given. After the second bolus, if signs of shock persist, inotropic support is required. Central venous access should be attempted, with a femoral line recommended for those not experienced in the internal jugular approach in children. If central access is not possible then inotropes may be administered in dilute solutions via a peripheral or intraosseous line. CVP is the guide for fluid administration.

It has long been established that early aggressive fluid resuscitation is associated with improved outcome in paediatric sepsis [10].

STATE OF UNRESPONSIVENESS / COMA

- Seizures
- Bleeding, Tumour, Abscess, Hydrocephalus
- Drugs / Poisons Infection (meningitis, encephalitis, HUS)
- Metabolic (hypoglycaemia, DKA, Reye) renal failure, hepatic coma

Endocrine (Addison) STATUS EPILEPTICUS

Convulsive status epilepticus (CSE) is defined as a single convulsion lasting more than 30 min or two or more convulsions lasting more than 30 min without recovery of consciousness.

As a single convulsion lasting more than 30 min

or two or more convulsions lasting more than 30 min without recovery of consciousness.

Convulsive status epilepticus is the most common neurological emergency in children and is more common than in adults albeit with a lower risk of death. Common causes of CSE include prolonged febrile seizures, acute central nervous system infection, epilepsy, hypoxia and metabolic abnormalities such as hypoglycaemia. The management of CSE includes airway management and assistance with administration of anticonvulsant medication. The management of CSE, as for all seriously ill children, should follow an ABCDE approach, as recommended by PALS and NHS guidelines [3,11]. NHS guidelines recommend up to two doses of intravenous benzodiazepine, with lorazepam 0.1 ml/kg. If CSE persists, phenytoin 20 mg/kg (over 20 min) should be given, maximum upto 40mg/kg.

The final escalation in the treatment of uncontrolled CSE is rapid sequence induction with thiopental 4mg/kg. Recently, levetiracetam has been shown to be effective in the treatment of CSE in children.

Keywords: Stridor, Croup, Epiglottitis, Status Asthmaticus, Shock, Status Epilepticus, PALS

References:

1. Denny F, Murphy TF, Clyde WA Jr, Cllier AM, Henderson FW et al. Croup: an 11-year study in a pediatric practice. *Pediatrics* 1983; 71: 871–6.
2. Bjornson C, Russell KF, Vandermeer B, Durec T, Klassen TP, Johnson DW. Nebulized epinephrine for croup in children. *Cochrane Database of Systematic Reviews* 2011; 2: CD006619.
3. Advanced Paediatric Life Support Group. *Advanced Paediatric Life Support: The Practical Approach*, 5th edn. New Jersey: Blackwell Publishing, 2011.
4. Kairys SW, Olmstead EM, O'Connor GT. Steroid treatment of laryngotracheitis: a meta-analysis of the evidence from randomized trials. *Pediatrics* 1989; 83: 683–93.
5. Faden H. The dramatic change in the epidemiology of pediatric epiglottitis. *Pediatric Emergency Care* 2006; 22: 443–4.

6. Werner HA. Status asthmaticus in children: a review. *Chest* 2001; 119: 1913–29.
7. Parameswaran K, Belda J, Rowe BH. Addition of intravenous aminophylline to beta2-agonists in adults with acute asthma. *Cochrane Database of Systematic Reviews* 2000; 4: CD002742.
8. Thill PJ, McGuire JK, Baden HP, Green TP, Checchia PA. Noninvasive positive-pressure ventilation in children with lower airway obstruction. *Pediatric Critical Care Medicine* 2004; 5: 337–42.
9. Brierley J, Carcillo JA, Choong K, et al. Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. *Critical Care Medicine* 2009; 37: 666–88.
10. Carcillo JA, Davis AL, Zaritsky A. Role of early fluid resuscitation in pediatric septic shock. *Journal of the American Medical Association* 1991; 266: 1242–5.
11. NICE clinical guideline 137 The epilepsies: the diagnosis and management of the epilepsies in adults and children in primary and secondary care, January 2012. <http://publications.nice.org.uk/the-epilepsies-the-diagnosis-and-management-of-the-epilepsies-in-adults-and-children-in-primary-and-secondary-care-cg137/appendix-f-protocols-for-treating-convulsive-status-epilepticus-in-adults-and-children-adults> (accessed 13/08/2012)



ACUTE POISONING – MANAGEMENT GUIDELINES

Dr. Amit Taneja

M.D Medicine

Chandigarh Clinic & Nursing Home
Bathinda

With the availability of a vast number of chemicals and drugs, acute poisoning is a common medical emergency in any country. The exact incidence of this problem in our country remains uncertain but it is estimated that about 10-15 million cases of poisoning are reported every year, of which, more than 50,000 die. The objective of this article is to familiarize the physicians about various steps required in the effective management of patients with acute poisoning.

For effective management of an acutely poisoned victim, five complementary steps are required. These are :

1. Resuscitation and initial stabilization
2. Diagnosis of type of poison
3. Nonspecific therapy
4. Specific therapy
5. Supportive care

Resuscitation and Initial Stabilization

On arrival of a patient with poisoning, the initial priorities are the maintenance of airway, breathing and circulation. If the patient has an altered level of consciousness, his cervical spine must be immobilized till an injury can be ruled out. If respiratory inadequacy is present, endotracheal intubation is required. Hypotension in poisoned patients is most often due to loss of fluids or toxin-induced vasodilatation. Hence, crystalloids are the first choice of treatment of hypotension. Before infusing fluids, blood should be withdrawn for investigations (including sugar, urea, electrolytes and acid-base status). Rectal temperature should be obtained in all patients with altered sensorium.

After initial resuscitation, all patients with altered sensorium should receive a 'cocktail' of 50% dextrose, naloxone and thiamine. However, recently, empiric administration of dextrose has been questioned. Experiments in animals have shown that administration of dextrose in both pre-and post-cardiac arrest conditions was associated with worse neurologic recovery^{1,2}. At present, it is recommended to check the blood sugar using a reliable bedside test and to administer dextrose only if the blood sugar is below 80 mg/dl. However, if the

sticks are not available, it is still advisable to administer dextrose to all patients with altered sensorium, including those with focal neurologic deficits^{3,4}.

Another component of the 'cocktail' recommended in patients with altered mental status is naloxone. It is able to rapidly counteract the sedation and respiratory depression induced by opiates. The dose is 2 mg in all age groups. However, if the patient is an opioid addict and is not apnoeic, the initial dose may be reduced to avoid withdrawal features⁵. Naloxone can occasionally produce side effects in the form of hypertension, pulmonary oedema, arrhythmias, seizures and cardiac arrest. It can also precipitate the withdrawal reaction.

Diagnosis of Type of Toxin

History : The history should be elicited from the patient as well as his relative. Occupational history and availability of potential poisons at home should also be asked for. However, it is very important not to believe the patient blindly particularly those who have ingested poison with a suicidal intent.

Examination : Once the patient has been stabilized, a thorough head-to-toe examination should be conducted. The objectives of this examination are two-fold : to diagnose the type of poison and to detect any associated trauma.

Based on the examination findings, it may be possible to identify the type of poison involved (Table I)⁶.

Table I : Clinical Features and Associated Poisons⁶

Clinical Features	Poisons
Odour of Breath	Chloroform, Ethanol, Cyanide, Arsenic, Organophosphates, Phosphorus, Kerosene
Hypertension with	Amphetamines, Cocaine, LSD, MAO inhibitors, Marijuana, Phencyclidine, Alcohol
Tachycardia	withdrawal, Nicotine, Antihistamines,

Hypotension with bradycardia	Antipsychotic agents, Antidepressants (severe cases), Barbiturates, Narcotics, Benzodiazepines, Cyanide, Nicotine, Organophosphates	Miosis	(amphetamines, cocaine, phencyclidine) Barbiturates, Phenothiazines, Ethanol, Narcotics, Nicotine, Organophosphates Amphetamines, Caffeine, Cocaine, LSD,
Hypotension with tachycardia	Aluminium phosphide, Antipsychotics, Caffeine, Cyanide, Disulfiram-ethanol interaction, Tricyclic antidepressants	Mydriasis	MAO inhibitors, Nicotine, Antidepressants, Antihistamines, Atropine
Hyperthermia	Amoxapine, Amphetamines, Antidepressants, Cocaine, Lithium, LSD, MAO inhibitors, Phencyclidine, Anticholinergic agents, Salicylates, Antihistamines	Cyanosis	Methaemoglobinaemia-inducing agents, Terminal stages of all poisonings
Hypothermia	Antidepressants, Ethanol, Benzodiazepine, Narcotics, Barbiturates, Phenothiazines		
Tachypnoea	Amphetamines, Atropine, Cocaine, Salicylates, Carbon monoxide, Cyanide, Hepatic		
Bradypnoea	Encephalopathy (paracetamol, amatoxin mushrooms), Metabolic acidosis Antidepressants, Antipsychotic agents, Barbiturates, Ethanol, Benzodiazepines, Chlorinated hydrocarbons, Narcotics, Nicotine, Organophosphates, Cobra bites		
Altered Sensorium	Antidepressants, Antihistamines, Antipsychotics, Atropine, Organophosphates, Barbiturates, Lithium, Cyanide, Benzodiazepines, Ethanol, Narcotics, Carbon monoxide		
Seizures	Antidepressants (amoxapine and maprotiline), Antipsychotic, Antihistamines, Chlorinated hydrocarbons, Organophosphates, Cyanide, Lead and other heavy metals, Lithium, Narcotics, Symptomimetics		

Laboratory Investigations :

A few simple bedside tests are helpful in diagnosing the chemical ingested. A pinkish colour of urine occurs in phenothiazine intoxication, as well as in myoglobinuria and haemoglobinuria. Chocolate-coloured blood is indicative of methaemoglobinaemia. Presence of oxalate crystals in urine is typical of ethylene glycol ingestion. Ketonuria without any metabolic change occurs in isopropyl alcohol and acetone intoxication while ketonuria with metabolic acidosis is suggestive of salicylate poisoning.

Abdominal X-ray may be useful in diagnosing certain radiopaque toxins which include chloral hydrate, heavy metals, iron, iodides, phenothiazines, sustained-release preparations and solvents (chloroform, carbon tetrachloride). However, one must not exclude a poisoning on the basis of absence of radiopaque density on X-ray⁶.

Non-specific Treatment

The next step in the management of a poisoned patient is to remove the unabsorbed poison from the gut and increase the excretion of absorbed poison from the body.

Gastric Decontamination

Removal of unabsorbed poison from the gut can be achieved by several means including induction of emesis, gastric lavage, and use of activated charcoal and cathartics.

Before performing a procedure for gastric emptying, it is important to consider :

- i) Whether the ingestion is potentially dangerous,
- ii) Can the procedure remove a significant amount of toxin, and
- iii) Whether the benefits of a procedure outweigh its risks?

If the patient has ingested a non-toxic agent, non-toxic dose of a toxic agents, or if he is free of symptoms despite passage of time during which the toxin is known to produce features of toxicity, gastric emptying is unnecessary. However, if the patient has ingested a high-risk toxin (cyanide, paracetamol), gastric emptying is indicated even

if he is asymptomatic. Gastric emptying is also not indicated if the patient had prior repeated vomiting or the toxin is absorbed rapidly, or patient presents late after ingestion. However, some toxins (antidepressants, phenothiazines, salicylates, opioids, phenobarbital and anticholinergics) delay gastric emptying. Gastric emptying is also delayed in comatose patients. It is also delayed if the toxin forms a mass in the stomach. In these situations, a delayed gastric emptying may be performed though there is no evidence to support this. If the risks of a procedure outweigh the possible benefits, it should be avoided (e.g., ingestion of volatile hydrocarbons, caustics)⁶.

Syrup of ipecac : Syrup of ipecac is used to induce emesis with the intention to remove the poison from the stomach. It was recommended in all patients with poisoning. The value of syrup of ipecac has been investigated in animal and human studies. The amount of recovery of toxin has been highly variable, and after one hour the amount is insignificant. Thus, presently, ipecac may be considered in an alert conscious patient who has ingested a potentially toxic amount of a poison within the last one hour⁷. It should be avoided in ingestion of hydrocarbons and corrosives. This compound, however, is not available in India.

Gastric lavage : For inserting an orogastric lavage tube, the patient should be placed in left lateral position with the head-end lowered. This will prevent aspiration and also reduce the entry of lavage fluid and poison into duodenum. If patient is unconscious, endotracheal tube must be inserted before lavage tube insertion in order to protect against aspiration into the lungs. A large bore tube (36 F in adults) is inserted into the stomach and its position is checked by injecting air through the tube into stomach and simultaneously auscultating over the epigastrium. The lavage is then performed by using fluid aliquots of 3-4 ml/kg. In adults, tap water at room temperature may be sufficient. However, in young children, isotonic saline at 37°C is preferable in order to prevent chances of hypothermia and hyponatraemia. The lavage is continued till the return is clear. A lavage is

contraindicated following ingestion of strong caustics, non-toxic agents and volatile hydrocarbons.

Gastric lavage is routinely performed in all patients with poisoning. However, studies conducted on animal, volunteer and human poisoning cases do not support the routine use of lavage in all patients with poisoning. Even in comatose patients, where gastric emptying is delayed, studies have shown lavage to be potentially dangerous and of little value in most cases. Therefore, it is recommended that gastric lavage should not be considered unless the patient has ingested a potentially life-threatening amount of poison and the lavage can be undertaken within 60 minutes of ingestion⁸. However, due to non-availability of activated charcoal in India, it may still be considered within 2 hours of ingestion of potentially toxic agents.

Cathartics : Cathartics have been used for several years with the hope of increasing the excretion of the toxins from the gut. Commonly used cathartics are : magnesium sulphate (30 g for adults and 250 mg/kg in children), magnesium citrate (4 ml/ kg up to a maximum of 300 ml) and sorbitol (1 g/kg as 70% solution). Sodium phosphate should not be used as it can lead to phosphate poisoning. Oil-based cathartics are contraindicated as they increase the absorption of several toxins. In addition, repetitive doses of these agents should be avoided. Important complications include electrolyte imbalance, dehydration, and in case of sorbitol, abdominal distension. Cathartics are contraindicated in presence of ileus, intestinal obstruction, renal failure, hypotension, severe diarrhoea and abdominal trauma. Despite theoretical benefits, there is no data to support their efficacy and their use cannot presently be recommended.

Activated Charcoal : Use of activated charcoal has revolutionized the treatment of poisoning. Due to its small particle size and enormous surface area, it can adsorb large amount of toxins. The

usual dose is 1 g/kg body weight or 10 parts of charcoal for every one part of toxin, whichever is greater. Activated charcoal is contraindicated in patients with unprotected airway and caustic ingestion. Although it is not effective in adsorbing lithium, iron, DDT, methanol, ethanol, metals and hydrocarbons, it is not contraindicated in these ingestions and may be given if a co-ingestion is suspected.

Based on the results of the volunteer studies, activated charcoal is likely to be beneficial if administered within 60 minutes of toxin ingestion. Therefore, in such situations, it may be considered beyond 1 hour but the data is insufficient to support or

exclude its use^{9,10}. Unfortunately, activated charcoal is not available in India.

Whole bowel irrigation : In this method, isotonic solution of polyethylene glycol-electrolytes is administered orally in a dose of 2 litres/hour in adults and 0.5 litres/hour in children. The procedure is continued for 4-6 hours or till the rectal effluent is clear. The components of this solution are not absorbed through the intestines. Instead, the solution flushes the gut mechanically. At present, there are no established indications for the use of whole bowel irrigation. Based on the experimental studies, WBI is an option for potentially toxic ingestions of sustained release or enteric coated drugs. It has a theoretical value in the management of iron ingestion and ingestion of drug packets. It is also of theoretical value if the toxin is not adsorbed by activated charcoal¹¹.

Enhancing Excretion

Once the absorption of a toxin has been reduced by various methods, the next logical step is to enhance the elimination of already absorbed toxin from the body. Important methods for this purpose are forced diuresis with alteration in urinary pH, multiple doses of charcoal, peritoneal and haemodialysis, haemoperfusion, haemofiltration and exchange transfusion.

Forced alkaline diuresis : One of the commonly used methods to increase the elimination of a toxin is forced diuresis with alteration in urine pH. Renal excretion of a substance is dependent upon glomerular filtration rate, active renal tubular secretion and passive tubular reabsorption. The glomerular filtration is determined by the molecular weight, the degree of protein-binding and the volume of distribution in the body. A large volume of distribution means that only a small amount of a chemical is available for filtration and therefore, forced diuresis is of little help. Because of these reasons, most of the chemicals (except isoniazid and bromides) are not amenable to removal by forced diuresis alone. The renal tubular epithelium is relatively impermeable to the ionized molecules. If the urinary pH is changed so as to produce more of ionized form of a chemical, it is trapped in the tubular fluid and is excreted in the urine. This is the basis for alkaline diuresis which is useful in salicylates, phenobarbital and lithium intoxication¹².

For alkaline diuresis, 5% dextrose in half-normal saline containing 20-35 mEq/L of bicarbonate is administered at a rate so as to produce a urine output of 3-6 ml/kg/hour and a urine pH 7.5-8.5. Diuretics are often needed to maintain high urine flows. To prevent hypokalaemia, potassium should be added in every

second or third bottle. During forced alkaline diuresis, the vitals of the patient alongwith input/output, electrolytes and acid base status should be closely monitored. This procedure is contraindicated in patients with shock, hypotension, renal failure and congestive heart failure.

Multiple-doses of activated charcoal : Multiple doses of activated charcoal have been recommended in treating certain poisonings. Because of multiple doses, free charcoal is available in the intestines to bind any toxin which has significant enterohepatic circulation. Further, free toxin in the blood tends to diffuse out of the blood into the intestines where it binds the charcoal, thereby maintaining the concentration of free toxin in the intestines near zero. This is termed "gastrointestinal dialysis". Depending upon the severity of poisoning, the doses are : 0.5-1 g/kg body weight every 1-4 hours. Multiple doses of charcoal are indicated in following conditions :

- (a) If the toxin has a long half life;
- (b) If the toxin has a significant enterohepatic circulation (digoxin, phenobarbital, theophylline);
- (c) If continuous release of toxin occurs from a sustained-release preparation;
- (d) If a toxin forms a mass in the gut which is a source of continuous release of toxin; and
- (e) If the ingestion is too massive to be effectively adsorbed by a single dose of charcoal.

However, repeated doses are contraindicated in the presence of ileus. In addition, repeated doses of cathartics must not be administered along with multiple doses of charcoal¹¹.

Dialysis : Peritoneal and haemodialysis are useful for water-soluble compounds of low molecular weight. Dialysis is useful in ethanol, methanol, salicylates, theophylline, ethylene glycol, phenobarbital and lithium intoxications. Peritoneal dialysis is a slow process and it should not be used if facilities for haemodialysis are available^{6,11,13}.

Table II : Antidotes and their use^{6,14,15}

antidotes, which are available in India, have been listed in table II.

Supportive Therapy

Since the antidotes are available only for a few toxins, treatment of most cases of poisoning is largely supportive^{6,11}. It is important not to waste time in locating an antidote; instead supportive therapy should be instituted after which an attempt may be made to get the antidote. The aim of the supportive

treatment is to preserve the vital organ functions till poison is eliminated from the body and the patient resumes normal physiological functions. Therefore, functions of central nervous system, cardiopulmonary system and renal system should be supported with proper care for coma, seizures, hypotension, arrhythmias, hypoxia, and acute renal failure. The fluid, electrolyte and acid-base status should be closely monitored in all patients.

Exposure to a Toxin through Routes other than Ingestion

Besides ingestion, a patient may be exposed to

Antidote Poison Administration Atropine
Cholinesterase inhibitors Initially, administer 2-4 mg for adults and 0.05 mg/kg for children. Repeat it every 5-15 minutes until there is cessation of oral and tracheal secretions. Then lower the dose and give at less frequent intervals to maintain atropinization for 24-48 hrs.

Pralidoxime Organophosphates 1-2 gm (25-40 mg/kg in children) IV over 10-20 minutes. Repeated every 4-8 hours

Naloxone Opiates See under 'Resuscitation'
Methylene blue Methaemoglobinemia 1-2 mg/kg body weight as a 1% solution to be given slowly over 5 minutes intravenously. May be repeated after 1 hour

Ethanol Methanol, Ethylene glycol Loading dose is 0.75 g/kg which is followed by maintenance dose of 0.1 g/kg/hr.

Deferoxamine Iron 90 mg/kg (upto 1 gm) i.m. followed by 90 mg/kg (upto 1 gm) every 4-12 hours. If hypotension is present, give intravenously at a rate not more than 15 mg/kg/hour

Snake antivenin Snake bites Dose varies with the species of snake which has bitten and the severity of envenomation

BAL (Dimercaprol) Lead, Arsenic, Mercury 300 mg/sq. meter/day in 6 divided doses (3-5 mg/kg every 4 hours) for 2 days, then 2.5-3 mg/kg every 6 hours for 2 more days, and then every 12 hours for 7 more days.

Specific Therapy

If the toxin can be identified, specific therapy using antidotes should be administered^{14,15}. Important a toxin via cutaneous or ocular routes. This is quite common with pesticides and insecticides. In case of cutaneous exposure, the initial decontamination consists of removal of all the clothing of the patient and putting them in a plastic bag. Even the shoes and gloves should be removed. While doing so, the physician must protect himself by using aprons, gum boots, masks and gloves. After derobing, the patient should be washed with soap and copious amounts of water. Use of neutralizing agents is strongly contraindicated⁶.

In case of ocular exposure, the eye should be irrigated with water for at least 20 minutes. An

intravenous set tubing with the tip about 3 cm away from the eye may be used to flush the eyes.

Legal Responsibilities

Any physician can treat a victim of poisoning without any fear of legal implications provided he follows set rules. The first sample of gastric lavage and other relevant body fluids like urine and blood, should be collected in clean bottles. It is not mandatory to perform a gastric lavage; it may be omitted if not indicated. The bottles should be sealed using a glue paper. After sealing the bottles, particulars of the patient should be written on the seal and the signatures affixed on the label at the juncture between the cap and the bottle. All the relevant information and observations about the patient should be recorded carefully. After initial management, police should be informed about the case.

With the use of a systematic approach to the poisoned patients, the morbidity and mortality of these patients can be minimized.

References

1. Myers RE, Yamaguchi S. Nervous system effects of cardiac arrest in monkeys. *Arch Neurol* 1977; 34: 65-74.
2. Hattori H, Wasterlain CG. Post glucose supplement reduces hypoxic-ischemic brain damage in the neonatal rat. *Ann Neurol* 1990; 28: 122-8.
3. Doyon S, Roberts JR. Reappraisal of the "coma cocktail". *Emerg Clin North Am* 1994; 12: 301-16.
4. Hoffman RS, Goldfrank LR. The poisoned patient with altered consciousness. Controversies in the use of a 'coma cocktail'. *JAMA* 1995; 274: 562-9.
5. Weismaan RS. Naloxone. In *Toxicologic Emergencies*, Goldfrank LR (eds.), Norwalk Connecticut 1994; pp 784-6.
6. *Diagnosis and Management of Acute Poisoning*. Aggarwal P, Wali JP (eds.), Oxford University Press Delhi 1997; pp 1-38.
7. Anonymous. Position statement : Ipecac syrup. *Clin Toxicol* 1997; 35: 669-709.
8. Anonymous. Position statement : Gastric lavage. *Clin Toxicol* 1997; 35: 711-9.
9. Anonymous. Position statement : Single-dose activated charcoal. *Clin Toxicol* 1997; 35: 721-41.
10. Bateman DN. Gastric decontamination - a view for the millennium. *J Accid Emerg Med* 1999; 16: 84-6.
11. Vernon DD, Gleich MC. Poisoning and drug overdose. *Critical Care Clin* 1997; 13: 647-67.
12. Prescott LF, Balali-Mood M, Critchley JAJH et al. Diuresis or urinary alkalization for salicylate poisoning? *BMJ* 1982; 286: 1383-6.
13. Pond SM. Extracorporeal techniques in the treatment of poisoned patients. *Med J Aust* 1991; 154: 617-22.
14. Jacobsen D, McMartin KE. Antidotes for methanol and ethylene glycol. *Clin Toxicol* 1997; 35: 127-43.
15. Bolgiano EB, Barish RA. Use of new and established antidotes. *Emerg Clin North Am* 1994; 12: 317-

FOREIGN BODY IN AERODIGESTIVE PASSAGE

Dr. Grace Budhiraja
M.S. (ENT)

Associate Professor

Adesh Institute of Medical Sciences and Research (AIMSR)
Bathinda, Punjab, 151001

Foreign body ingestion & aspiration is a common accident that befalls children & adults alike. Significant dilemmas in diagnosis of these problems - between ENT Surgeons & Paediatrician.

Teamwork of ENT Surgeons, Paediatrician & Anaesthetist – adequate understanding results in smooth management.

VARIOUS FOREIGN BODIES

- Coins (75%) • Marbles
- Beads
- Buttons
- Plastic particles
- Pendant
- Rings
- Battery cells

Sharp Objects :

- Safety pin
- Common pin
- Needles • Glass Pieces
- Fish/chicken bone pieces
- Denture
- Metallic wires

Vegetative materials :

- Seeds of Sitafal
- Seeds of Chiku
- Peanut
- Beans
- Betel nut
- Corn
- Meat – large boluses

AETIOLOGY

Age group – most common in < 12 yrs
Highest incidence between 1 to 3 yrs

IN CHILDREN

Lack of molars • Improper grinding of food
Less coordinated swallowing & respiratory ability
Exploring environment by mouth
Running/playing while eating

IN ADULTS

Use of upper denture prevents tactile sensation & foreign body is swallowed undetected.

Pieces of food may be held up in patients with pathological narrowing e.g. oesophageal stricture due to carcinoma, corrosive ingestion, peptic oesophagitis

- Epileptic seizures
- Alcoholic intoxication
- Poorly prepared food & improper mastication
- Psychotics - foreign body may be swallowed with an attempt to commit suicide

FOREIGN BODIES

Food Passage

(Ingestion)

Air Passage

(Inhalation)

Foreign Bodies

In

Food Passage

ANATOMICAL FACTS

The ingested foreign body may lodge in Tonsil

The Base of Tongue / Valleculae

The Pyriform Fossa

The Oesophagus

Sharp foreign bodies like fish bone, chicken bone, needle or denture may lodge in Tonsillar crypts, Valleculae or

Pyriform fossa. But the commonest site of lodgement of foreign body is Oesophagus.

SITES OF ANATOMICAL NARROWING IN OESOPHAGUS

- » Narrowest part of GIT – Cricopharynx
- » Less commonly at mid or distal oesophagus, caused by extraluminal compression by Aortic Arch or left Main Bronchus
- » Above gastro- oesophageal sphincter (at the level of diaphragmatic hiatus)

CLINICAL PRESENTATION

- » Majority of cases clear history of ingestion of foreign object
- » Initial Choking or Coughing.
- » Discomfort / Pain in Throat : on right or left side point to the site of Foreign body.
- » Dysphagia: partial or total. Partial obstruction may become total with time due to oedema.
- » Excessive salivation: if total obstruction
- » Nausea & Vomiting

CLINICAL PRESENTATION

- » In some cases, no definite history.
- » In few cases, no acute symptomatology or presentation occurs in an obscure fashion e.g. respiratory symptoms – stridor, wheezing due to compression over posterior wall of Trachea.
- » In some neglected cases, patients present with atypical complaints of fever, substernal or epigastric pain or hematemesis which are suggestive of complications.

INVESTIGATIONS

RADIOGRAPHIC EVALUATION

- » Plain x ray soft tissue neck with chest including abdomen (posteroanterior & lateral films) for radio opaque foreign body
- » Barium swallow / Fluoroscopy for radiolucent foreign body.
- » Repeat radiograph before attempted removal
- » Children should be x rayed from neck to the rectum as multiple foreign bodies may have been ingested.

BARIUM SWALLOW/FLUOROSCOPY

For radiolucent foreign bodies. Patient is asked to swallow a cotton soaked in barium or barium filled

capsule & its passage is observed through the oesophagus.

MANAGEMENT

- » Frequently resulted in death prior to 20th century
- » The basic principles of foreign body extraction by endoscopic techniques were meticulously developed by Chevalier Jackson in 1936.
- » Fiberoptic flexible oesophagoscope is also used now a days in cases when foreign body has passed cricopharyngeal junction. Sharp foreign bodies like fish bone, chicken bone, needle or denture lodged in Tonsillar crypts, Valleculae or Pyriform fossa can be removed under local anaesthesia by using Macintosh laryngoscope.

The most popular technique for cricopharyngeal & oesophageal foreign body removal is Rigid Direct Laryngoscopy / Oesophagoscopy Other Techniques include:

- » Flexible Fiberoptic Oesophagoscopy
- » Foley's Catheter Technique
- » Oesophageal Bougienage
- » Dormia Basket

All these methods require general anaesthesia with endotracheal intubation.

RIGID DIRECT LARYNGOSCOPY / OESOPHAGOSCOPY

- » The most popular technique.
- » Sharp foreign body can also be removed safely by avoiding open surgical methods.
- » Foreign body, forceps & oesophagoscope removed as single unit.
- » Oesophagoscope reinserted to look for another foreign body, assess mucosa of oesophagus.
- » Smooth foreign bodies do not pose much threat but may cause airway obstruction.
- » Sharp foreign bodies, if not retrieved at the earliest may penetrate oesophageal wall & cause complications. (Amateur efforts for removal can cause perforation because of sharp edges.

- » The sharp ends of the foreign body or entire foreign body itself can be introduced into the lumen of rigid endoscope & removed without any risk of lacerating the mucosa during extraction.
- » No such protection with flexible endoscope.
- » For removal of open safety pins with pointed edge facing up - Rotating it in stomach, engaging the pointed edge into the scope & withdrawing it, or closing it in the lumen of oesophagus.

OESOPHAGEAL BOUGIENAGE

- » Another method is pushing the foreign body into the stomach with a bougie.
- » Certain foreign bodies like marbles or beads which are difficult to hold with forceps are pushed into the stomach or can be removed with Dormia basket. (Watchful observation of stools)

FOLEY'S CATHETER TECHNIQUE

- » Fluoroscopically controlled foley's catheter can be used to remove non opaque oesophageal foreign bodies which are smooth. (After catheter is advanced past the foreign body, the balloon is filled with radio opaque contrast material & catheter is withdrawn & foreign body is usually delivered into a pharynx & removed.

Rigid Direct Laryngoscopy & Oesophagoscopy
Trolley

OPEN SURGICAL METHODS

(Occasionally, sharp foreign bodies do require surgical intervention specially in case of perforation or abscess formation or impaction.

- » **Cervical Oesophagotomy:** For sharp foreign bodies like hooks, dentures located above thoracic inlet - Incision in the neck & opening of cervical oesophagus.
- » **Transthoracic Oesophagotomy:** For impacted foreign bodies of thoracic oesophagus - chest is opened at the appropriate level.

COMPLICATIONS

Technological advances have allowed us to master the techniques of foreign body removal, but still complications do occur. Caused by Object itself By length of time By attempts to retrieve the object

Complications of Foreign body ingestion

- » Respiratory Obstruction–Tracheal compression & laryngeal oedema
- » Perioesophageal cellulitis & retropharyngeal abscess
- » Perforation if object is sharp - Mediastinitis, pericarditis, empyema
- » Tracheo oesophageal fistula
- » Ulceration & stricture in overlooked foreign body

DISK BATTERY INGESTION

- » Emergency
- » 1 hr - Mucosal damage
- » 4 hrs - Erosion of muscular wall
- » 6 or more hrs - Oesophageal perforation or Death The ingested foreign body may lodge in (Larynx)
- » Trachea
- » Bronchi
- » Site depends on size & nature of the foreign body.
- » Small foreign body passes through larynx into Trachea or Bronchi (80-90%).
- » Larger objects & sharp objects impact in larynx or trachea or subglottis.
- » Lodgment in Right main bronchus is more common due to
- » Greater diameter than left
- » Less divergent angle
- » Interbronchial septum projects to the left
- » The effect of inspiratory air currents also determines the site of final impaction

PHASES OF F.B. AIR PASSAGE

I. Initial phase: choking, gagging, paroxysm of coughing airway obstruction

II. Asymptomatic phase: lasting for hours to weeks when foreign body becomes lodged & reflexes fatigue

III. Complication phase: pneumonia, atelectasis, abscess, fever, hemoptysis Clinical Presentation

- » Event of aspiration witnessed by parent.
- » Acute choking
- » Severe coughing followed by respiratory distress
- » Symptoms of asthma, croup, pneumonia
- » Sudden onset of wheezing in healthy child

CLINICAL SIGNS

- » Increased respiratory rate
- » Use of accessory respiratory muscles
- » Chest indrawing
- » Suprasternal retraction
- » Respiratory distress - inspiratory stridor
- » Cyanosis
- » On auscultation, decreased air entry in the lung fields with obstructive sounds. Laryngeal Foreign Bodies
- » Irregular foreign body or foreign body in sagittal plane causes partial obstruction.
- » Laryngeal oedema following aspiration can cause complete obstruction.
- » Symptoms of obstruction, hoarseness & croup
- » Triad of cough, wheezing & decreased breath sounds
- » Swollen foreign body (e.g. seed) or oedema of tissue around the foreign body can cause complete obstruction & lobar collapse.

TRACHEAL FOREIGN BODIES

- » Symptoms of obstruction but without hoarseness
- » Three signs in Tracheal foreign bodies
- » "Asthmatoid wheeze"
- » "Audible slap"
- » "Palpable thud" over trachea

RADIOGRAPHIC EVALUATION

- » X-ray chest PA view
- » X-ray soft tissue neck lateral view
- » Must be before intervention
- » Laryngotracheal foreign body may be seen on lateral x ray as subglottic narrowing
- » Fluoroscopy / Videofluoroscopy for radiolucent foreign bodies

On X Ray Evaluation : Bronchial Foreign Bodies
Early – Check valve effect expiratory obstruction & thus obstructive emphysema.

On X Ray: Hyperinflation on affected side & mediastinal shift to the opposite side

On X Ray Evaluation : Bronchial Foreign Bodies
Intermediate –

Ball valve effect
Inspiratory obstruction
producing atelectasis on
affected side & mediastinal shift on affected side

On X Ray Evaluation : Bronchial Foreign Bodies

Late – Stop valve effect complete obstruction
consolidation of lobe & Lobar collapse

- » X-rays often suggest but do not diagnose foreign body aspiration.
- » Negative radiographs are NOT ENOUGH to rule out presence of foreign body.
- » SUSPECT - Airway foreign body in children with respiratory distress not responding to medical management (e.g. higher antibiotics, steroids & bronchodilators)

MANAGEMENT

Patient at home or Primary Health Centre (PreHospital Care) (< 1 yr - Back bows & chest thrusts (> 1 yr - Gentle abdominal thrusts in supine position

- » Older child / adults – Heimlich Maneuver
Emergency Department Care Initial Supportive Therapy for Unstable Patients
- » Oxygen administration
- » Cardiac monitor
- » Pulse oximetry
- » Intravenous line
- » Definitive airway management – To by pass upper airway – Tracheostomy/ Cricothyrotomy
- » In stridorous patients, racemic epinephrine via a nebulizer.

DEFINITIVE MANAGEMENT

- » Large laryngeal foreign bodies can be removed by Direct Laryngoscopy under General anaesthesia with Tracheostomy sos.
- » Tracheal & Bronchial foreign bodies are best removed using Rigid Bronchoscope.

Rigid Bronchoscopy + FB Removal
Requirements before taking Patient in OT

- » Obtain pertinent history & high risk informed consent
- » Experienced Anaesthetist

- » Volatile Inhalation agents, Mask preoxygenation & “Intermittent Venturi Method” (Equipments : Endoscopes & Forceps. Type & size of Endoscope depends on age of the patient & location of Foreign body (Select forceps according to type of Foreign body.
- » Standby Tracheostomy Team

Rigid Bronchoscopes with Forceps Optical Foreign Body Grasping Forcep Other Techniques include:

- » Rigid Bronchoscopy with Telescopy aid
- » Bronchoscopy with C - arm Fluoroscopy
- » Use of Dormia Basket or Fogarthy’s Balloon for rounded objects
- » Bronchoscopy through Tracheostome
- » Flexible Fiberoptic Bronchoscopy (Thoracotomy & Bronchotomy for peripheral foreign bodies

Fiberoptic Flexible Bronchoscope

Fiberoptic Flexible Bronchoscopy

+ Foreign Body Removal

- » Unless Endoscopist is skilled at Rigid Bronchoscopy, use of Flexible scope for foreign body removal is not recommended.
- » Problems of poor control of airway & of foreign body itself.

Fiberoptic Flexible Bronchoscopy

+ Foreign Body Removal

- » If trial is given by Flexible Bronchoscopy, a back up team of Rigid Bronchoscopy should always be there.

- » Flexible Bronchoscopy is indicated in patients with cervical spine instability and skull & jaw fractures.

POST OPERATIVE CARE

- » Chest physiotherapy for retained secretions
- » Antibiotics only if long time retained Foreign Body
- » Steroids if trauma during procedure

COMPLICATIONS

- » Mostly due to delayed diagnosis & treatment.
- » Pneumonia & atelectasis - most common
- » Laryngeal inflammation & oedema
- » Granulation tissue & stricture formation
- » Bleeding due to granulation tissue or erosion into major vessel
- » Pneumothorax or pneumomediastinum due to airway tear
- » Mortality

SUMMARY & CONCLUSIONS

- » Early identification & referral lead to a quick result
- » Good knowledge of symptomatology should prompt the diagnosis.

SUMMARY & CONCLUSIONS

- » Under expert care & team management one can predict a good outcome.



EMERGENCY AIRWAY MANAGEMENT

Minnu Mridul Panditrao

Dr. Minnu Mridul Panditrao

Professor

drmmprao@gmail.com 8699943250

Department of Anaesthesiology & Intensive Care

Adesh Institute of Medical Sciences and Research (AIMSR)

Bathinda, Punjab, 151001

Address for Correspondence:

Dr. Minnu Mridul Panditrao

Professor, Department of Anaesthesiology & Intensive Care

AIMSR, Adesh University

Bathinda, Punjab, 151001

drmmprao1@gmail.com

Key words:

Airway Management, emergency, various methods

Introduction:

A clear, patent airway is an absolute necessity for proper ventilation in an emergency situation or during resuscitation. In such patients who are not fully conscious and are unable to respond to the simple commands, their upper airways and ventilation get compromised.

In such situations, sooner than later an advanced airway is required to protect the lungs from aspiration of secretions, blood or GI contents. (Basic airway devices are various types of face masks, oropharyngeal/nasopharyngeal airways).

Indications of Advanced airway are:

1. Unconscious patient
2. Upper airway trauma/inflammation/edema
3. Respiratory failure- (hypoxia/hypercarbia, patient not able to cough) of central/peripheral origin
4. Ventilator dependent patients
5. Inhalation injury
6. Severe head injury
7. Severe shock
8. Under general Anaesthesia

The various advanced airway devices used for maintaining a patent/secure airway are:

1. Endotracheal tubes
2. Supraglottic airway devices (LMAs, i-gel, COPA, Slippa, etc.)
3. Combitube
4. Cricothyroidotomy needles or cannulae

5. Tracheostomy tubes The most simple and commonly used advanced airway devices are the cuffed endotracheal tubes. Others are the alternative options when either the intubation is very difficult/ impossible, or as a stop-gap alternative, or as an aid to intubation.

Endotracheal Intubation:

In patients with normal physique and normal anatomy, in majority cases, intubation is a simple procedure and successfully achieved. But there is a small percentage of patients where it becomes difficult or even impossible. These are the cases which become airway emergencies and here it is very important to have a structured protocol which should be followed to properly manage these cases, so as to avoid the high incidence of morbidity and mortality associated with failed intubation.

There are certain important steps to avoid or minimize the chances of failed intubation. These are the proper preparation of the patient and the equipment before starting to intubate. The following things should be ready before attempting intubation.

1. Appropriate sized Facemasks, oral/nasal airways.
2. A properly working laryngoscope set with appropriately sized laryngoscope blades (MAC and Miller)
3. Appropriate sized endotracheal tubes
4. Stylet, Bougies, Syringes
5. Properly working suction apparatus with suction tip and appropriate sized suction catheters.
6. Ambu-resuscitator or Bain circuit connected to oxygen supply source for uninterrupted supply of oxygen
7. Drugs for sedation and muscle relaxation
8. A small pillow
9. Stethoscope, pulse oximeter, ETCO2 monitor, ECG monitor
10. Emergency drugs and crash cart
11. Experienced help

For smooth and successful intubation one should:-

1. Always pre-oxygenate the patient with facemask for at least 2-3 minutes.
2. Give sedation (midazolam, propofol or ketamine), depending upon GC of patient.
3. Give muscle relaxant i.e. either succinyl choline, or rocuronium or atracurium.

4. Attempt laryngoscopy only after completely paralyzing the patient i.e. after patient becomes apneic.
5. Throat should be cleared of secretions, blood or vomitus with the help of suction. Insert laryngoscope in the right angle of mouth, displacing the tongue to the left.
6. Visualize the epiglottis as a first step.
7. Then position the laryngoscope blade at the base of epiglottis and lift the blade up, thus lifting the epiglottis indirectly and bringing the glottis in your view.
8. If the glottis is not clearly visible at this stage, then give slight pressure on the thyroid cartilage in the neck with your freehand. This will bring the glottis in your view.
9. In some patients with short/stiff neck, even this may not help to visualize the whole of the glottis. At this stage, if you can see even the posterior-most small part of glottis, you can try passing a bougie into that, directing the bougie under the epiglottis to go into the larynx. This works in >90% of the patients.
10. It is advised to try only 3+1 intubation attempts with different laryngoscope blades/ endotracheal tubes and different laryngoscopists, giving intermittent bag and mask ventilation and maintaining Oxygen saturation > 90-95% at all times.

If this does not work we go for next options, which are failed intubation protocols.

These are:-

1. Use of LMA/i-gel
 2. Use of video laryngoscopes
 3. Fiber optic bronchoscopy and intubation (may not be feasible in emergency situations)
 4. Needle/Scalpel Cricothyroidotomy
 5. Percutaneous or Surgical Tracheostomy
- During all these situations, it is very important to do intermittent bag-valve-mask ventilation and not let the O₂ saturation fall below 90% at any stage.

Supraglottic Devices:

SGA/LMA/i-gel insertion:-

The key to success here is adequate sedation and blunting of pharyngeal and laryngeal reflexes. Propofol is suitable for this (if not contraindicated).

Steps are:

1. Pre-oxygenate.
2. Clear the throat by thorough suction.
3. Achieve adequate depth of sedation.

4. Open the mouth wide and insert SGA taking care to remain clear of tongue.

Tongue should not get pushed in and get folded. It should go over the tongue. Once the SGA has gone deep enough and you encounter resistance to further insertion, stop and inflate the cuff. Attach to Ambu-bag or Bain circuit and try to give IPPV → Chest should inflate easily, without significantly large leak. Or you can see spontaneous respiratory efforts transmitted to the reservoir bag. Confirm the placement with chest auscultation and/or ETCO₂ monitor before fixing the SGA in position. Size is chosen according to body weight which is written on the device. You can even try intubating through SGA with a smaller ETT, especially with fast track LMA.

But if SGA also fails, you should try only up to 3 times with different sizes, or use of another type of SGA.

If it is still unsuccessful, go for the next alternative option, which is:-

- Use of video laryngoscope (GlideScope, TruScope, TruView, etc.).

These are very good devices, very useful as you can directly visualize the oral/oropharyngeal structures and can intubate under vision. However, cost and expertise are the factors. Practice is required for fast, successful results. Same is true with fiber-optic bronchoscope. If all these methods fail then the last option is:

- Cricothyroidotomy or Tracheostomy:
 - a. In Cricothyroidotomy, a wide bore needle or cannula is passed through the cricothyroid ligament in the midline in the front of the neck and once it is inside the airway (you can see the bubbling of air), it is connected to O₂ supply and IPPV device.
 - b. In Tracheostomy, a stoma is made either with percutaneous needle and dilators or by surgical scalpel, and a tracheostomy tube is inserted in the trachea in the midline in the front of the neck, which is fixed in place and connected to O₂ and IPPV machine.

But these two approaches are invasive techniques and involve certain amount of morbidity for the patient.

DIABETIC EMERGENCIES

Dr. Bharat Bhushan Jindal
M.D. (Medicine)

Dr. Bharat Bhushan Jindal's Complete Medical Centre
Opp. St No-8/9, Power House Road, Civil Lines,
Bathinda, Punjab 151001

The primary aim of management of diabetes associated emergencies includes rapid evaluation and stabilization of the patient. In such cases, a thorough clinical history, physical and urine analysis) is conducted. Critically ill patients undergo additional testing including complete metabolic panel, serum osmolarity, phosphate levels, and cardiac markers. It is recommended that the patients with high risk should be identified soon after the hospitalization to avoid further complications. This article discusses various aspects of dealing with diabetes associated emergencies in the hospital setting.

Diabetes is frequently associated with various comorbidities such as dyslipidemia, hypertension, chronic kidney disease, cardiovascular disease, nonalcoholic fatty liver disease, lower extremity amputations, higher risks of infections etc. Additionally, the occurrence of hyperglycemia, and associated conditions like ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS), and hypoglycemia makes patients vulnerable to frequent hospitalization. This article discusses the management of diabetes associated emergencies based on the underlying causes.

MANAGING HYPOGLYCEMIA

Hypoglycemia is one of the most frequent complaints reported in the emergency department. Hypoglycemia in hospitalized patients can be defined as blood glucose levels of $< 70\text{mg/dl}$ and severe hypoglycemia being $< 40\text{mg/dl}$. Patients may experience hypoglycemia due to a sudden interruption in the nutrition, infection, sepsis or due to iatrogenic factors like inappropriate timing of insulin in relation to meals, sudden reduction in the corticosteroid dose, reduced rate of intravenous dextrose infusion etc. Although the recurrent hypoglycemia due to concurrent medications is still largely unknown, experts recommend that the patients consuming various medications are at higher risk of hypoglycemic episodes.

Once bedside hypoglycemia is confirmed, immediate management includes either intramuscular glucagon or intravenous 25% dextrose. In case of decreased sensorial state and when oral intake is not feasible, IV dextrose should be considered as the first line therapy. In cases where blood sugar levels do not rise to normal range, then the initial dose should be repeated. The treatment of hypoglycemia may have a

shorter duration of action depending on the underlying cause. Hence it is recommended that the blood glucose levels are monitored hourly to prevent recurrent episodes of hypoglycemia. Other aspect includes addressing iatrogenic causes and ensure preventing improper prescribing of medications, nutrition, insulin, mismatch etc. A proactive surveillance of causes can reduce the hypoglycemic episodes. In this regard, patients should be evaluated before hospital discharge to avoid recurrent hypoglycemic episodes. Following criteria can be considered for the evaluation.

Identifying cause and nature of hypoglycemia

- * Nature of hypoglycemia (isolated, accidental, caused by an oral medication or long-acting insulin)
- * The identified cause that is unlikely to happen again.
- * Not due to medication error or long-action insulin. It has been observed that patients taking oral hypoglycemic agents or long-acting insulin are susceptible to recurrent hypoglycemic episodes.
- * Whether it was managed with rapid and continuous dextrose infusion.

Patient Characteristics

- * Has completed an uneventful 4-hour observation and blood glucose is in the normal range and not trending down.
- * Has eaten a full meal during the observation.
- * Has no comorbid conditions that can interfere with the administration of other medications.
- * Can accurately monitor blood glucose levels at home.
- * It is recommended that their insulin dose is reduced by 25% at least for the 24 hours to reduce the recurrent hypoglycemia risk.

Patient Counseling

- * Making patients aware that hypoglycemia is a true medical emergency and it should be attended and treated immediately to prevent organ and brain damage.
- * Patients should ensure adequate oral intake and maintain a good glycemic control with successful self-administration of the insulin and self-monitoring of the blood glucose levels.

Complication	Management	Important points
Hyperglycemia (Critical ill patients)	IV Insulin	Monitor blood sugar every after 1-2 hours
	IM Basal Insulin	Basal insulin dose= 50% of TDD
	Bolus Insulin	Bolus insulin per meal = (50%TDD)/3 Correctional scale estimation as per insulin sensitivity
DKA	Hydration	0.9% Saline
	Insulin Therapy	IV Insulin. Alternative routes: IM or subcutaneous.
	Electrolyte therapy (Potassium management And/ or Bicarbonate therapy)	Serum Potassium > 3.5 mEq/L Bicarbonates are recommended only when pH < 6.9
HHS	Hydration	0.9% Saline
	Insulin Therapy	IV insulin is recommended
	Potassium Management	

Managing Hyperglycemia

In patient or hospital related hyperglycemia is defined as any blood glucose concentration of > 140 mg/dl. When patients miss an insulin injection, oral hypoglycemic agent, or ingest a large number of carbohydrates can trigger hyperglycemic events. Hyperglycemia can further lead to various physiological changes resulting into life-threatening conditions like DKA and HHS. Hence the management of hyperglycemic patients can be divided into three categories as critically ill patients, and patients with DKA or HHS.

Management of Hyperglycemic Patients

Once the diagnosis of hyperglycemia is confirmed, blood glucose levels before all meals and bedtime should be monitored. Continuous IV insulin should be initiated in critically ill patients whereas subcutaneous basal/bolus insulin is advised in non-critical care settings. Patients with higher blood glucose should receive correctional insulin doses along with bolus insulin.

Additionally, hyperglycemia can also be associated with conditions like DKA and HHS. DKA is characterized by uncontrolled hyperglycemia, metabolic acidosis, and increased total body ketone

concentration. Hence, usual features of DKA include,

- * Usually hyperglycemia (>250mg/dl)
- * Metabolic acidosis (pH < 7.35) and bicarbonate < 15mM
- * High anion gap (>20mM)
- * Ketonaemia/heavy (3+) ketonuria

Whereas, HHS is related to altered mental status due to hyperosmolarity, dehydration and severe hyperglycemia without significant ketoacidosis. DKA commonly occurs in type 1 diabetes patients but occasionally occurs in type 2 diabetes patients.

Table 1: Management Of Hyperglycemia And DKA /HHS

Once DKA or HHS are resolved, the transition of IV insulin to subcutaneous basal insulin can be initiated. However, abrupt cessation of IV insulin can result in hyperglycemia and ketogenesis. It is recommended that subcutaneous basal insulin is given at least 2 hours before discontinuing the IV infusion. Generally, DKA and HHS are associated with medication non-compliance and hence patient assistance programs or counseling sessions are recommended to avoid such life-threatening recurrent episodes.

Additionally, **sliding scale insulin (SSI) therapy** is quite popular but has shown inferior results with respect to bolus insulin. SSI promotes blood glucose to rise, and then the retrospective administration of insulin is initiated. This therapy causes high variation in the blood glucose to rise, and then the retrospective administration of insulin is initiated. this therapy causes high variations in the blood glucose levels and thus it is difficult to predict insulin dosage requirements. Additionally, SSI therapy increases the rate of hyperglycemic episodes. Hence, using **sliding scale insulin therapy** as a sole therapy for hyperglycemia management should be avoided.

Diabetes patients are susceptible to hyper- and hypo-glycemia or comorbidities frequently visit emergency department and hospital. In such cases, emergency physician must be in a position to diagnose and stabilize a patient with appropriate management to attain glycemic control.

References

1. Rowden AK, Fasano CJ. Emergency management of oral hypoglycemic drug toxicity. *Emerg Med Clin North Am.* 2007; 25:347-56. abstract viii.
2. Ford, William, et al. "Diabetes in the emergency department and hospital: acute care of diabetes patients." *Current emergency and hospital medicine report* 1.1(2013): 1-9.
3. Maynard, Greg, et al. "Impact of a hypoglycemia reduction bundle and a systems approach to inpatient glycemic management." *Endocrine practice*4 (2014): 355-367
4. Diabetes Care in the Hospital, *Diabetes Care* 2016;39(Suppl. 1):S99-S104.
5. Queale, William S., Alexander j. Seidler, and Frederick L.Brancti. "Glycemic control and sliding scale insulin use in medical inpatients with diabetes mellitus." *Archives of internal medicine*5 (1997): 545-1450.
6. Udwadia, Farokh, et al. "Intravenous insulin as part in a hospital setting: results from an observational study examining patient outcomes and physician preferences." *Diabetes Management*2 (2012): 103.
7. Avanzini,Fausto, et al."Transion from intravenous to subcutaneous insulin." *Diabetes Care*7 (2011):
8. Umpierrez, guillermo E., et al."Randomized study comparing a basel-bosel with a basel plus correction insulin regimen for the hospitel management of medical and surgical patients with type 2 diabetes." *Diabetes care*8 (2013): 2169-2174.
9. Morris, Lawrence R., MARY BETH murphy, and Abbas E. Kitabchi."Bicarbonate therapy in severe diabetic ketoacidosis." *Ann Intern Med*6 (1986):836-40.
10. Gangopadhyay, kalyan kumar, et al."Consensus evidence-based guidelines for in-patient management of hyperglycaemia in non-critical care setting as per Indian clinical practice." *JAPI Suppl*62 (2014).
11. Hardern, R.D., and N.D.Quinn. "Emergency management of diabetic ketoacidosis in adults," *Emergency medicine journal*3 (2003): 210-213.
12. Fayfman, Maya, Francisco J. Pasquel, and Guillermo E. Umpierrez. "Management of Hyperglycemic crises." *Medical Clinics*3 (2017): 587-606.



MANAGEMENT OF SHOCK: HAEMORRHAGIC HYPOVOLEMIC SHOCK

Mridul Madhav Panditrao

Professor & Head

Department of Anaesthesiology & Intensive Care
Adesh Institute of Medical Sciences and Research (AIMSR)
Bathinda, Punjab, 151001

Abstract: Shock as an entity is enigmatic, dynamic and very challenging. Out of the various types of shock, concentration has been given to the hypovolemic, haemorrhagic shock. Traumatically injured patients should be transported as quickly as possible, to be treated by a specialized trauma center whenever possible. Measures to monitor and support coagulation should be initiated as early as possible and used to guide resuscitation. A damage control approach to surgical intervention should guide patient management. Awareness of potential thrombotic risk and pre-treatment with anticoagulant agents, particularly in older patients, should be part of routine clinical management. Adherence to a multidisciplinary, evidence-based treatment protocol as the cornerstone of patient management

Introduction:

Of all the emergency situations a clinician may face in their day to day practice, none is so diabolically horrifying and dangerous as 'Shock'. Irrespective of the specialty one may belong to, facing a patient in shock/impending shock is not an unusual occurrence. In fact, many a times it is better to be forewarned and be prepared to anticipate the shock than actually to face it, as the dynamism and emergent nature of the pathological conditions involved in development of shock, make it the most unpredictable event in the clinical practice.

Shock can be defined as [1] "An Altered Physiologic state characterized by significant reduction of systemic tissue perfusion, resulting in the life-threatening failure of adequate oxygen delivery to the tissues." Basically:

It is the imbalance between oxygen delivery and oxygen consumption! It may be due to • decreased blood perfusion of tissues, • inadequate blood oxygen saturation, &/or • increased oxygen demand from the tissues

Ultimately culminating in to [2]

- Decreased end-organ oxygenation and dysfunction • generalized cellular hypoxia (starvation)
- If left untreated, results in sustained multiple organ dysfunction
- end-organ damage with possible death
- Tissue hypoperfusion may be present without systemic hypotension
- At the bedside: shock is commonly diagnosed

when both are present (arterial hypotension and organ dysfunction).

General Pathophysiology:

The cells switch from aerobic to anaerobic metabolism, leading to lactic acid production. As a result of the failure of membrane bound Na⁺/K⁺ ATPase mechanism, the membrane becomes more permeable, electrolytes and water starts to seep in, leading to swelling up of cells, release of proteolytic enzymes. Impairment of mitochondrial function occurs and cell death is imminent.

Types of Shock:

On the basis of aetio-pathogenesis, pathophysiology and clinical features, one can broadly classify the shock in to mainly three types, which are further sub typed as follows viz;

- Hypovolemic shock
- Hemorrhagic
- Surgical
- Non surgical
- Non-hemorrhagic
- Distributive Shock
- Septic shock
- Anaphylactic shock
- Neurogenic shock
- Cardiogenic Shock

For all practical purposes, it would be beyond the scope of present discussion to deal with all the types/ sub types of shock. It would be pertinent to concentrate on Hypovolemic, Haemorrhagic shock, which in true sense is a real emergency.

Hypovolemic shock: Refers to a medical or surgical condition in which rapid fluid loss results in multiple organ failure due to inadequate circulating volume and subsequent inadequate perfusion

Hemorrhagic shock: Refers to a condition of reduced tissue perfusion, resulting in the inadequate delivery of oxygen and nutrients that are necessary for cellular function. Whenever cellular oxygen demand outweighs supply, both the cell and the organism are in a state of shock. This occurs generally secondary to excessive/ uncontrolled blood loss, either surgical or Non surgical!!

For the convenience of understanding and remembering, American College of Surgeons in the Advanced Trauma Life support (ATLS) has given various classes of hemorrhage as follows:

	Class I	Class II	Class III	Class IV
Blood loss (ml)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15 %	15–30 %	30–40 %	>40 %
Pulse rate (bpm)	<100	100–120	120–140	>140
Systolic blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (ml/h)	>30	20–30	5–15	Negligible
CNS/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Initial fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

*For a 70 kg man

Table 1: ATLS classification of Haemorrhagic shock
Early Acute Coagulopathy associated with Trauma:[4-8]

Recently there has been growing evidence about a clinical entity, which is evident on admission, about 30% of all bleeding trauma patients already show signs of coagulopathy. It has been noted that in such patients, there is a

- significant increase in the occurrence of multiple organ failure and death

- compared to patients with similar injury patterns in the absence of a coagulopathy
- This has recently been recognized as a multifactorial primary condition that results from a combination of
 - bleeding-induced shock,
 - tissue injury-related thrombin-thrombomodulin-complex generation
 - the activation of anticoagulant and fibrinolytic pathways



A number of terms have been proposed to describe the specific trauma-associated coagulopathic physiology, including:

- Acute Traumatic Coagulopathy
- Early Coagulopathy of Trauma
- Acute Coagulopathy of Trauma-Shock
- Trauma-Induced Coagulopathy
- Trauma-Associated **Coagulopathy**

The exact aetiological factors and pathogenesis is still under consideration but, multiple factors have been postulated to be involved. The factors like age, genetic makeup, pre-existing co-morbidities and any pre-medication have also been implicated to play a major role. Schematic drawing of the factors, both pre-existing and trauma-related, that contribute to traumatic coagulopathy

Principles of Management of hemorrhagic shock

It is directed toward optimizing perfusion of the vital organs & thereby optimizing oxygen delivery. Rapid diagnosis and treatment of the underlying hemorrhage is a must with concurrent management of shock.

Supportive therapy:

- oxygen administration,
- monitoring, and
- establishment of intravenous access (2 large-bore cannulae in peripheral lines, central venous access) Intravascular volume and oxygen-carrying capacity should be optimized. In addition to crystalloids, some colloid solutions, hypertonic solutions, and oxygen-carrying solutions (hemoglobin-based and perfluorocarbon emulsions) may be administered.
- Blood products in severe hemorrhagic shock.
- Replacement of lost components using red blood cells (RBCs), fresh frozen plasma (FFP), and platelets
- Recent combat experience has suggested that aggressive use of FFP may reduce coagulopathies and improve outcomes
- Determination of the site and etiology of hemorrhage is must
- Control of hemorrhage may be achieved in the ED,
- Control may require multidisciplinary approach and special interventions.

Recently there has been rekindled interest in the management of Hemorrhagic hypovolemic shock Pan-European, multidisciplinary Task Force for

Advanced Bleeding Care in Trauma was founded in 2004. This included representatives of six relevant European professional societies. The group used a structured, evidence-based consensus approach to address scientific queries, that served as the basis for each recommendation and supporting rationale. Existing recommendations were reconsidered and revised based on new scientific evidence and observed shifts in clinical practice; new recommendations were formulated to reflect current clinical concerns and areas in which new research data have been generated. This guideline represents the fourth edition of a document first published in 2007 and updated in 2010 and 2013.[9]

The guidelines take in to consideration multiple factors and their relevance as at the level of relevance.

- Severely injured patients be transported directly to an appropriate trauma facility. (Grade 1B)
- Time elapsed between injury and bleeding control be minimized (Grade 1A)
- adjunct tourniquet use to stop life-threatening bleeding from open extremity injuries in the pre-surgical setting. (Grade 1B)
- the avoidance of hypoxaemia. (Grade 1A)
- normoventilation of trauma patients. (Grade 1B)
- hyperventilation in the presence of signs of imminent cerebral herniation. (Grade 2C)

Initial Survey:

- The physician clinically assess the extent of traumatic hemorrhage using a combination of patient physiology, anatomical injury pattern, mechanism of injury and the patient's response to initial resuscitation. (Grade 1C)
- patients presenting with haemorrhagic shock
- an identified source of bleeding undergo an immediate bleeding control procedure unless initial resuscitation measures are successful. (Grade 1B)
- an unidentified source of bleeding undergo immediate further investigation. (Grade 1B)
- early imaging (ultrasonography or contrast-enhanced CT) for the detection of free fluid in patients with suspected torso trauma. (Grade 1B)
- patients with significant intra-thoracic, intra-abdominal or retroperitoneal bleeding and haemodynamic instability undergo urgent intervention. (Grade 1A)
- CT assessment for haemodynamically stable patients.

High degree of suspicion of coagulopathy:

- a low initial Hb be considered an indicator for

severe bleeding associated with coagulopathy. (Grade 1B)

- use of repeated Hb measurements as a laboratory marker for bleeding, as an initial Hb value in the normal range may mask bleeding. (Grade 1B)
- serum lactate and/or base deficit measurements as sensitive tests to estimate and monitor the extent of bleeding and shock. (Grade 1B)
- routine practice include the early and repeated monitoring of coagulation,
- prothrombin time (PT), PTI, INR, activated partial thromboplastin time (APTT), platelet counts and fibrinogen (Grade 1A) and/or a viscoelastic method (Thromboelastogram) (Grade 1C)
- a target systolic blood pressure (SBP) of 80–90 mmHg until major bleeding has been stopped in the initial phase following trauma without brain injury. (Grade 1C)
- In patients with severe Traumatic Brain Injury (GCS \leq 8), a mean arterial pressure (MAP) \geq 80 mmHg be maintained. (Grade 1C)

Volume Therapy:

- In the presence of life-threatening hypotension, administration of vasopressors in addition to fluids to maintain target arterial pressure. (Grade 1C)
- infusion of an inotropic agent in the presence of myocardial dysfunction. (Grade 1C)
- fluid therapy using isotonic crystalloid solutions be initiated in the hypotensive bleeding trauma patient. (Grade 1A)
- “3:1 rule” 3cc crystalloid for every 1cc of blood loss
- excessive use of 0.9 % NaCl solution be avoided. (Grade 2C)
- hypotonic solutions such as Ringer’s lactate be avoided in patients with severe head trauma. (Grade 1C)
- the use of colloids be restricted due to the adverse effects on haemostasis. (Grade 2C)
- a target Hb of 7 to 9 g/dl. (Grade 1C)
- early application of measures to reduce heat loss and warm the hypothermic patient in order to achieve and maintain normothermia. (Grade 1C)

Damage Control Management:

- Urgent Emergency surgery in the severely injured patient presenting with deep haemorrhagic shock, signs of ongoing bleeding and coagulopathy. (Grade 1B)

- Primary definitive surgical management in the haemodynamically stable patient and in the absence of any of the factors above. (Grade 1C)
- Patients with pelvic ring disruption in haemorrhagic shock to undergo immediate pelvic ring closure and stabilization. (Grade 1B)
- patients with ongoing haemodynamic instability despite adequate pelvic ring stabilisation receive
- early pre-peritoneal packing
- angiographic embolisation and/or surgical bleeding control. (Grade 1B)
- the use of topical hemostatic agents in combination with other surgical measures
- with packing for venous or moderate arterial bleeding associated with parenchymal injuries. (Grade 1B)

Pharmacological control over bleeding:

- monitoring and measures to support coagulation be initiated immediately upon hospital admission. (Grade 1B)
- In the initial management of patients with expected massive hemorrhage, one of the two following strategies, to maintain PT and APTT $<$ 1.5 times the normal control. (Grade 1C)
- Plasma (FFP) in a plasma–RBC ratio of at least 1:2 as needed. (Grade 1B)
- Fibrinogen concentrate and RBC according to Hb level. (Grade 1C)
- Tranexamic acid (TXA) be administered as early as possible (within 3 hrs.) at a loading dose of 1 g infused over 10 min, followed by an i.v. infusion of 1 g over 8 h. (Grade 1A)
- resuscitation measures be continued using a goal-directed strategy guided by standard laboratory coagulation values and/or viscoelastic tests. (Grade 1C)

Component Therapy:

- plasma transfusion be avoided in patients without substantial bleeding.
- fibrinogen concentrate or cryoprecipitate if significant bleeding (Grade 1B)
- viscoelastic signs of a functional fibrinogen deficit
- plasma fibrinogen level of less than 1.5–2.0 g/l. (Grade 1C)
- initial fibrinogen supplementation of 3–4 g.
- This is equivalent to 15–20 single donor units of cryoprecipitate or

- Repeat doses must be guided by viscoelastic monitoring and laboratory assessment of fibrinogen levels. (Grade 2C)
- platelets be administered to maintain a platelet count above $50 \times 10^9/l$. (Grade 1C)
- maintenance of a platelet count above $100 \times 10^9/l$ in patients with ongoing bleeding and/or TBI. (Grade 2C)
- If administered, an initial dose of four to eight single platelet units (Grade 2C)

Other Modalities to Control the Bleeding:

- Ionized calcium levels be monitored and maintained within the normal range during massive transfusion. (Grade 1C)
- administration of platelets in patients on antiplatelet agents and
- substantial bleeding or intracranial hemorrhage (Grade 2C)
- continued microvascular bleeding. (Grade 2C)
- desmopressin is not to be used routinely in the bleeding trauma patient. (Grade 2C)
- desmopressin ($0.3 \mu g/kg$) be administered in patients treated with platelet-inhibiting drugs or with von Willebrand disease. (Grade 2C)
- the early use of prothrombin complex concentrate (PCC)
- for the emergency reversal of vitamin K-dependent oral anticoagulants. (Grade 1A)
- life-threatening post-traumatic bleeding in patients treated with novel oral anticoagulants. (Grade 2C)

Miscellaneous

- measurement of plasma levels of oral anti-factor Xa agents such as rivaroxaban, apixaban or edoxaban in patients treated or suspected of being treated with one of these agents. (Grade 2C)
- If bleeding is life-threatening, treatment with TXA 15 mg/kg (or 1 g) intravenously and high-dose ($25\text{-}50 \text{ U/kg}$) PCC/aPCC until specific antidotes are available. (Grade 2C)
- Off-label use of rFVIIa be considered (Grade 2C)
- pharmacological thromboprophylaxis within 24 h after bleeding has been controlled. (Grade 1B)
- early mechanical thromboprophylaxis with intermittent pneumatic compression (IPC) (Grade 1C) and anti-embolic stockings. (Grade 2C)
- No routine use of inferior vena cava filters as

thromboprophylaxis. (Grade 1C)

Role of rFVIIa (Activated Recombinant Factor VII).

A very lucid review [10], describes in detail the role of rFVIIa, in various bleeding disorders as well as, acute episodes during the surgical and traumatic conditions.

The recommendations are:

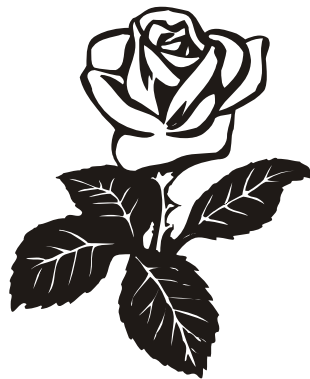
- Replace lost / consumed hemostatic factors with FFP, cryoprecipitate, platelets and red blood cells
- Full blood count, PT,APTT and fibrinogen
- The use of rFVIIa should be considered
- if bleeding continues when more than one blood volume has been transfused (approximately 10 units of red cells in an adult)
- No identifiable surgical source of bleeding has been found.
- If it is felt that rFVIIa may be of benefit
- It should normally only be requested by a consultant anesthetist.
- It should normally be used only following discussion with a consultant hematologist
- rFVIIa should be given in the dose of $50 - 100 \mu g/kg$ for a $50 - 100 \text{ kg}$ patient)
- If bleeding does not diminish in 30 - 60 minutes, then a further 4.8 mg vial can be given

Conclusion: The take home message is

- Replace lost / consumed hemostatic factors with FFP, cryoprecipitate, platelets and red blood cells
- Full blood count, PT,APTT and fibrinogen
- The use of rFVIIa should be considered
- if bleeding continues when more than one blood volume has been transfused (approximately 10 units of red cells in an adult)
- No identifiable surgical source of bleeding has been found.
- If it is felt that rFVIIa may be of benefit
- It should normally only be requested by a consultant anesthetist.
- It should normally be used only following discussion with a consultant hematologist
- rFVIIa should be given in the dose of $50 - 100 \mu g/kg$ for a $50 - 100 \text{ kg}$ patient)
- If bleeding does not diminish in 30 - 60 minutes, then a further 4.8 mg vial can be given

References:

1. Gaieski et al. 2009 (Online accessed 22 August 2013)
URL <http://ijhs.sandi.net/faculty/rtenenbaum/ap-biology-folder/Links/Shock.utd.pdf>
2. <http://bestpractice.bmj.com/topics/en-us/1013> accessed on 2018.06.15
3. <https://www.facs.org/quality-programs/trauma/atls>. accessed on 2018.06.15
4. Celso B, Tepas J, Langland-Orban B, Pracht E, Papa L, Lottenberg L, et al. A systematic review and meta-analysis comparing outcome of severely injured patients treated in trauma centers following the establishment of trauma systems. *J Trauma.* 2006; 60(2): 371–378. doi: 10.1097/01.ta.0000197916.99629.eb. [PubMed] [Cross Ref]
5. Hill AD, Fowler RA, Nathens AB. Impact of interhospital transfer on outcomes for trauma patients: a systematic review. *J Trauma.* 2011; 71(6): 1885–1900. doi: 10.1097/TA.0b013e31823ac642. [PubMed] [Cross Ref]
6. Hess JR, Brohi K, Dutton RP, Hauser CJ, Holcomb JB, Kluger Y, et al. The coagulopathy of trauma: a review of mechanisms. *J Trauma.* 2008; 65(4): 748–754. doi: 10.1097/TA.0b013e3181877a9c. [PubMed] [Cross Ref]
7. MacLeod JB, Lynn M, McKenney MG, Cohn SM, Murtha M. Early coagulopathy predicts mortality in trauma. *J Trauma.* 2003;55(1):39–44. doi: 10.1097/01.TA.0000075338.21177.EF. [PubMed] [Cross Ref]
8. Brohi K, Singh J, Heron M, Coats T. Acute traumatic coagulopathy. *J Trauma.* 2003;54(6):1127–1130. doi: 10.1097/01.TA.0000069184.82147.06. [PubMed] [Cross Ref]
9. Rolf Rossaint, Bertil Bouillon, Vladimir Cerny, Timothy J. Coats, Jacques Duranteau, Enrique Fernández-Mondéjar, Daniela Filipescu, Beverley J. Hunt, Radko Komadina, Giuseppe Nardi, Edmund A. M. Neugebauer, Yves Ozier, Louis Riddez, Arthur Schultz, Jean-Louis Vincent and Donat R. Spahn *Critical Care* (2016) 20:100 DOI .1186/s13054-016-1265-x.
10. Mridul Panditrao, Minnu Panditrao, Mohammed Shamsah. Massive bleeding in Trauma and Surgery: Role of rFVIIa. *Kuwait Medical Journal* 2011,43(3), 176-188.



EMERGENCIES IN CARCINOMA CERVIX

Dr. Rupinder Sekhon

Sr. Consultant and Chief of Gynae Oncology
Rajiv Gandhi Cancer Institute and Research Centre
Rohini, Delhi

Incidence

The current estimates indicate approximately 132,000 new cases diagnosed and 74,000 deaths annually in India, accounting to nearly 1/3rd of the global cervical cancer deaths. Indian women face a 2.5% cumulative lifetime risk and 1.4% cumulative death risk from cervical cancer.

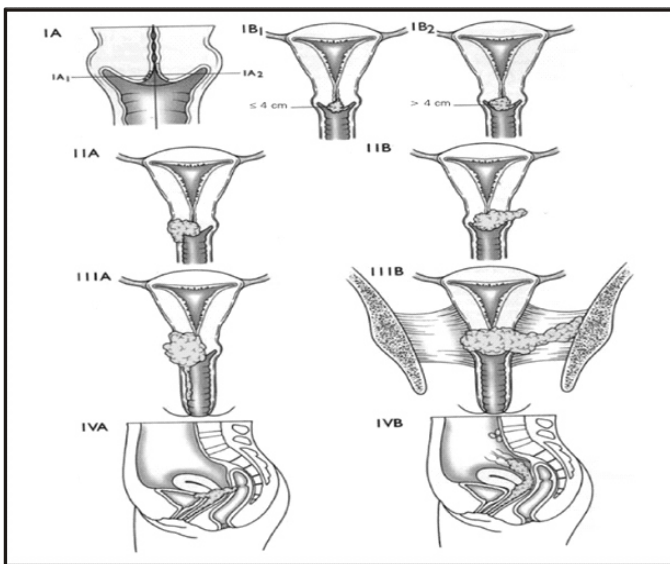


Figure 1. Staging of uterine cervix carcinoma according to FIGO⁽³⁾.

FIGO Staging for Carcinoma Cervix

Revised FIGO Staging for Carcinoma Cervix

Stage I

- ★ The carcinoma is strictly confined to the cervix (extension to the corpus would be disregarded)
- ★ IA Invasive carcinoma which can be diagnosed only by microscopy, with deepest invasion ≤ 5 mm and largest extension ≥ 7 mm
- ★ IA1 Measured stromal invasion of ≤ 3.0 mm in depth and extension of ≤ 7.0 mm
- ★ IA2 Measured stromal invasion of ≥ 3.0 mm and not ≥ 5.0 mm with an extension of not ≥ 7.0 mm

- ★ IB Clinically visible lesions limited to the cervix uteri or pre-clinical cancers greater than stage IA
- ★ IB1 Clinically visible lesion ≤ 4.0 cm in greatest dimension
- ★ IB2 Clinically visible lesion ≥ 4.0 cm in greatest dimension

Stage II

- ★ Cervical carcinoma invades beyond the uterus, but not to the pelvic wall or to the lower third of the vagina IIA Without parametrial invasion
- ★ IIA1 Clinically visible lesion ≤ 4.0 cm in greatest dimension
- ★ IIA2 Clinically visible lesion ≥ 4 cm in greatest dimension
- ★ IIB With obvious parametrial invasion

Stage III

- ★ The tumor extends to the pelvic wall and/or involves lower third of the vagina and/or causes hydronephrosis or non-functioning kidney
- ★ IIIA Tumor involves lower third of the vagina, with no extension to the pelvic wall
- ★ IIIB Extension to the pelvic wall and/or hydronephrosis or non-functioning kidney

Stage IV

- ★ The carcinoma has extended beyond the true pelvis or has involved (biopsy proven) the mucosa of the bladder or rectum. A bullous edema, as such, does not permit a case to be allotted to Stage IV

★ IVA Spread of the growth to adjacent organs

★ IVB Spread to distant organs
Emergencies in Ca Cervix

Haemorrhage / Bleeding

★ Acute: Excessive bleeding PV
Hypotension
Shock

★ Chronic: Hematuria & Bleeding PR in locally advanced cases
Severe Anemia
Intractable pain

Cancer spreads to nerve endings, bones or muscles, it can often cause severe pain, which can usually be controlled with painkilling medications.

DVT

Large tumors can press on the veins in the pelvis. This slows the flow of blood and can lead to a blood clot developing in the legs.
Pulmonary Thromboembolism

Kidney Failure Obstructive uropathy

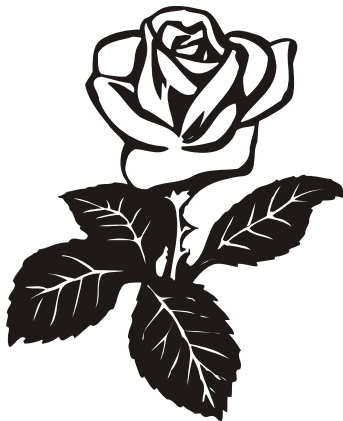
Excessive nausea / vomiting
Severe pain in loin
Oliguria / Anuria

Fistulae: A fistula is a rare but distressing complication of advanced cervical cancer.

★ Vesico-vaginal Fistula

Fistula is a channel that develops between the bladder and the vagina. This can lead to a persistent discharge of urine from the vagina.

★ Recto-vaginal Fistula A fistula can sometimes develop between the vagina and rectum.



HYPERTENSIVE EMERGENCIES

Dr. Aman Salwan

M.D. DNB (Cardiology)

FICS (Singapore) FACC (U.S.A)

Director, Cardiac Cath Lab, Sr. Consultant Cardiologist
Delhi Heart Institute & Multispecialty Hospital Bathinda

HYPERTENSIVE EMERGENCIES : DEFINITION

A rapid decompensation of vital organ function secondary to an inappropriately elevated BP

Require lowering of BP within 1 hour to decrease morbidity

Not determined by a BP level, but rather the imminent compromise of vital organ function Signs:

- Retinal hemorrhages, exudates, or papilledema
- Renal involvement (malignant nephrosclerosis) with AKI, proteinuria, hematuria
- Cerebral edema à seizures and coma
- Pulmonary Edema
- Myocardial Infarction
- Hemorrhagic Stroke, lacunar infarcts
- Hypertensive Emergencies
- CNS - Hypertensive encephalopathy
- CVS
- Acute myocardial ischemia
- Acute cardiogenic pulmonary edema
- Acute aortic dissection
- Post-op vascular surgery
- Renal - Acute renal failure
- Eclampsia Catechol excess- Pheochrom, Drugs

High BP WITHOUT acute end-organ dysfunction IS NOT a hypertensive emergency
“Hypertensive Pseudoemergency”

HYPERTENSIVE EMERGENCIES : OBJECTIVES

- Distinguish which hypertensive presentations require immediate therapy
- Describe appropriate therapies for each presentation
- Describe the risks of treatment
- Discuss the advantages and disadvantages of currently available antihypertensive drugs

CASES

- Asymptomatic 65 year-old, BP 200/115 (143)
- Embolic CVA, BP 215/105 (142)
- Hemorrhagic CVA, BP 200/100 (133)
- SAH, BP 180/100 (127)
- Aortic dissection, BP 175/105 (128)

- Pregnant female, BP 150/100
- Encephalopathy, BP 260/160 (194)
- Acute pulmonary edema, BP 220/120 (153)

SIGNS AND SYMPTOMS

- Hypertensive Urgency:
- Can be completely asymptomatic
- Some symptoms include:
- Severe headache
- Shortness of breath
- Nosebleeds
- Severe anxiety
- Elevated BP on consecutive readings S&S Continued
- Hypertensive Emergencies

Symptoms:

- Inausea, vomiting (cerebral edema)
- Chest Pain
- SOB
- Blurry vision
- Confusion
- Loss of consciousness Pathophysiology of Hypertensive Emergencies
- Rate of change of BP determines likelihood
- Chronic HTN lowers probability adaptive vascular changes protect end-organs from acute changes in BP
- Previous normotensives (eclampsia, acute GN) develop signs and symptoms at lower BP's
- Pathophysiology of Hypertensive Emergencies
- Endothelial Role in BP Homeostasis
- Sudden increased vasoreactivity
- Inflammatory vasculopathy Loss of endothelial function
- Therapeutic considerations in hypertensive emergencies
- Need for rapid reduction of BP
- Potential complications of therapy
- Prevalence of cerebrovascular disease and coronary artery disease (Stenotic lesions)
- Altered cerebral autoregulation
- Impaired baroreflexes o Blood viscosity

- Ability to increase oxygen extraction
- How far can BP be safely lowered?
- Lower limit usually 25% below MAP 50% of chronic hypertensives reached lower autoregulation limit with 11 to 20% reduction in MAP
- 50% had lower limit above usual mean
- Kanaeko et al; J Cereb Blood Flow Metab 3:S51,1983
- Most ischemic complications develop with reductions greater than 20 - 30 % (over 24 to 48 hours)
- Blindness, paralysis, coma, death, MI
- Initial Lowering of BP : Therapeutic Guidelines
- Do not lower BP more than 20% over the first 1 to 2 hours unless necessary to protect other organs
- Decreasing to DBP of 110 or patients "normal" levels may not be safe
- Further reductions should be very gradual (days)
- Follow neuro status closely
- Concept of Hypertensive Urgencies
- Potentially dangerous BP elevation without acute, life-threatening end-organ damage
- Examples (controversial!)
- Retinal changes without encephalopathy or acute visual symptoms
- High BP with nonspecific Sx (headache, dizziness, weakness)
- Very high BP without symptoms

HYPERTENSIVE URGENCIES

- Severe elevation of BP (DBP > 115)
- No progressive end-organ disease
- Joint National Committee on Detection, Evaluation, and Treatment of HBP
- 1984 - lower BP within 24 hours
- 1988 - urgent therapy rarely required
- 1993 - Gradual lowering of BP

Risks of rapid reduction (cerebral and myocardial ischemia) Management of Specific Hypertensive Emergencies

HYPERTENSIVE ENCEPHALOPATHY

- Abrupt, sustained increased BP exceeds limits of cerebral autoregulation
- **MAP 150 -200**
- Variable vasospasm, edema, hemorrhages

- Headaches, nausea, vomiting, confusion
- Patchy focal neuro deficits
- Papilledema, retinopathy
- Signs + symptoms resolve with reduction of BP
- Hypertensive Encephalopathy: Differential Dx
- Stroke (Ischemic)
- Intracranial (intracerebral or subarachnoid) hemorrhage
- Intracranial mass
- Encephalopathy due to drug ingestion, CNS infection, uremia

HYPERTENSION WITH STROKE SYNDROMES

- Need for BP therapy controversial rebleed, hemorrhagic transformation increased edema and ICP
- Hypertension often transient, physiologic response which resolves spontaneously
- BP reduction may cause ischemic neurologic deterioration
- Ischemic penumbra
- Cerebral autoregulation (right shift)

CAUTIOUS REDUCTION OF VERY HIGH BP SUBARACHNOID HEMORRHAGES

- 20% rebleed within 2 weeks (24 hrs)
- Increased risk if SBP >160 or MAP > 110
- No study has shown that treatment of BP reduces risk of rebleeding
- Acute right shift of curve (ICH,hydrocephalus)
- Nimodipine for cerebro-protection (vasospasm)
- Cautious decrease in BP by 20% initially, then below SBP of 160 (if not yet clipped)

INTRACEREBRAL HEMORRHAGE

- HTN associated with increased mortality
- HTN may be a marker for more advanced chronic arterial compromise
- Physiologic response to increased ICP from clot
- Decrease in BP may raise ICP
- Ischemic penumbra may exist in ICH
- No evidence that acute lowering of BP reduces risk of hematoma expansion, rebleed rare after 12 hrs
- Rate of 24-hour BP decline and mortality after spontaneous ICH
- Qureshi et al; CCM 1999, 27: 480 – 485

INTRACEREBRAL HEMORRHAGE

- NSA recommendations
- SBP > 220 or DBP >120

- NINDS recommendations
- SBP > 180, MAP > 130
- Lower BP to MAP 100 - 130
- Control of BP not been demonstrated to decrease ongoing or recurrent bleeding

THROMBOEMBOLIC (ISCHEMIC) CVA'S

- NSA recommendations
- SBP > 220, DBP > 120
- NINDS recommendations
- DBP > 140 - NTP
- SBP > 220, DBP > 120, MAP > 130
- Labetalol, Enalapril, esmolol, Nitropaste
- Ischemic penumbra
- Thrombolytic therapy

AORTIC DISSECTION

- Tear in intima @ separation or "dissection" of wall longitudinally
- 50% mortality in first 48 hours; begin treatment based on suspicion of Dx
- Decrease pulse wave contour (dP/dT)
- Therapeutic regimens: (SBP 100 - 120, HR < 80) propranolol plus nitroprusside labetalol trimethaphan
- Definitive diagnosis (CT, TEE, aortography, MRI) after control of BP, contractility, pain
- Acute LV failure / Acute cardiac ischemia
- HTN @ increased afterload; may precipitate LV failure or ischemia
- Dyspnea, pain, anxiety may cause HTN
- Specific BP therapy indicated if patient remains hypertensive after conventional measures for CHF or ischemia
- NTG, NTP, ACEI
- BB, CCB

PRE-ECLAMPSIA/ECLAMPSIA

- Preeclampsia
- Mild = 140/90 with proteinuria
- Severe = 160/110, 5 gm protein, Sx
- "Standard" therapy is hydralazine
- Other agents: Nifedipine, labetalol, diazoxide (small doses), methyldopa
- Nitroprusside (risk of fetal CN toxicity)
- Additional measures: MgSO₄; Delivery

DRUG ASSOCIATED HYPERTENSION "HYPERCATECHOLAMINE STATE"

CASES

- Asymptomatic 65 year-old, BP 200/115 (143)

- Embolic CVA, BP 215/105 (142)
- Hemorrhagic CVA, BP 200/100 (133)
- SAH, BP 180/100 (127)
- Aortic dissection, BP 175/105 (128)
- Pregnant female, BP 150/100
- Encephalopathy, BP 260/160 (194)
- Acute pulmonary edema, BP 220/120 (153)

CLINICAL CHARACTERISTICS OF HYPERTENSIVE CRISIS

- BP: Usually > 140 mm Hg diastolic
- Funduscopic findings:
- Hemorrhage, exudate, papilledema
- Neurological status:
- Headache, confusion, somnolence, stupor, visual loss, focal deficits, seizures, coma
- Cardiac findings:
- Prominent apical impulse, cardiac enlargement, congestive failure
- Renal: Oliguria, azotemia
- Gastrointestinal: Nausea, vomiting

CONDITIONS TO BE DIFFERENTIATED FROM A HYPERTENSIVE CRISIS

- Acute left ventricular failure
- Uremia from any cause, particularly with volume overload
- Cerebrovascular accident, Subarachnoid hemorrhage
- Brain tumor, Head injury
- Epilepsy (postictal)
- Collagen diseases (i.e., lupus), with cerebral vasculitis
- Encephalitis
- Overdose and withdrawal from narcotics, amphetamines
- Hypercalcemia
- Acute anxiety with hyperventilation syndrome

Summary

- Hypertensive Crises are common
- Differentiate Hypertensive Urgency from Emergency on the basis of end-organ damage
- Can treat hypertensive urgency with oral antihypertensives, but parenteral medications required for hypertensive emergencies
- 25% reduction in diastolic BP over 2-6 hours for hypertensive emergencies
- Don't forget to start Oral antihypertensives and follow-up closely!

Thank You.

MEDICO-LEGAL ASPECTS OF SUDDEN UNEXPECTED DEATH, DIFFICULT SITUATIONS IN MEDICAL PRACTICE, BROUGHT DEAD, POST MORTEM

Dr. (Prof.) Mahesh Baldwa

M.D, D.C.H, FIAP, MBA, LL.B, LL.M, PhD (law)

SENIOR PAEDIATRICIAN & SENIOR MEDICO-LEGAL ADVISOR

Author of following books

1. legal issues in medical practice [2018]- CBS publisher
2. Textbook of Medicolegal issues” [2012]- Jaypee publisher
3. “Legal Problems in day-to-day Pediatric Practice”[2005,2010]- paras publisher
4. “Desktop reference book on medicolegal issues”[2007]” Cipla publisher
5. “Doctors know your Rights and Responsibilities under Consumer Act” [1994,1995] Formerly Assistant Professor of Pediatrics at T.N. Medical College and Nair Hospital, Mumbai-400008

Ex. Asst. Professor JJ Hosp, Grant medical college

Ex. Professor, paper setter & examiner of law to postgraduate students of University Department of Law, University of Mumbai-400032

Baldwa Hospital, Sumer Nagar, S.V. Road, Borivali (West) Mumbai-400092

drbaldwa@gmail.com

9322990138

KEY WORDS

Sudden unexpected death (SUD)

Medico-legal (ML)

medical negligence (MN)

critical care (CC)

emergency treatment (ET)

intensive care units (ICU)

Indian penal Code (IPC)

Consumer protection Act-1986(CPA)

Introduction

It is rather impossible to find a medical professional who would say that they never faced a medico-legal (ML) situation called “Sudden unexpected death” (SUD) and world at large gazing in their face as if they were responsible for death. This type of scenario where treating team of doctor is made to feel guilty about SUD by relatives is not uncommon. All hospitals, one or the other day face difficult situations anticipated or unexpected. The difficult situations can diffuse easily or may take turn for bad. The difficult situations one can face are:

- A. Sudden death and mob violence
- B. Brought dead
- C. Settlement of bills or recovery of dues.

When these problems arise in small individual proprietary hospitals or small to medium multispecialty hospital, the situation is very different and let us concentrate more on this from point of view of small nursing homes. The big or corporate hospitals have a different and faceless channel to settle these matters and no one is personally involved. However, in a small proprietary hospital, the consultant has to deal directly with such situations and may face problems.

What is the definition of MN in SUD:

Heavy cost of CC & ET has made this emerging medical specialty, a hotspot of prosecution in alleged MN cases. In today’s scenario medical professionals are health risk managers of the critically sick patient, who need vigilant monitoring with various gadgets and timely treatment to maintain vital parameters to avert any further crisis due to common & foreseeable complications. So corollary is that medical professionals are expected to chart the course of the health of their critically ill patients with minimal health hazards by use of state of art and costly monitoring equipment. Any action or inaction of medical professional in emergency room that accelerates or increases the health risks may result in allegation of breach of duty of medical professional.

Sudden death and small nursing homes

Sudden death itself constitutes a shock to the family. It is equally shocking to the treating doctor. Intense efforts are usually made by treating doctor or nursing staff on the site in the ward with whatever available resources. In small nursing homes, this is the time when certain deficiencies are noticed by the

bystanders or the relatives of the deceased. Usually, there is shouting, loud orders by the doctors, commotion and lot of hurry and worry in the body language of all those who are trying to resuscitate the ailing patient. This all is witnessed by relatives of the patient and when death is declared, the efforts go unnoticed and it is only the deficiencies that are pointed out. Spectrum of ML reactions from patient party A spectrum of ML reactions from patient party affecting medical professional may be

(a) sometimes in the event of SUD occurring in front of lot of accompanying relatives then at slightest provocation they may take law in their own hands and bash doctors and other staff members along with destroying medical equipment & hospital property. (b) Some other times, no sooner than expected instead relatives asking you explanations about SUD, you have a policemen coming to make ML questioning to you and your staff as to what happened to deceased patient as they have received a ML complaint from relatives. (c) So often in the event of SUD, you may receive a politicians telephone to resolve the issue amicably or some social worker actually walking in your office to pay for SUD or a local goon threatening you to cough up money immediately for SUD without asking your explanation (d) So often in the event of SUD, you may find media and press people gathering around you & your staff members to speak details of SUD, which are flashed in defamatory way on TV or newspapers leaving you disgusted. (e) In the event of ML SUD, you should feel lucky and blessed if police does not walk to make inquiry about SUD but so often your comfort is disturbed weeks later or sometimes months later by a lawyers notice probing in SUD and asking for case papers related to medical treatment of deceased along with compensation. (f) As per the media projection one out of ten doctors are receiving dragged in unnecessary prosecutions for medical negligence (MN). Court summons for SUD and complains narrating absurd allegations and asking astronomical sum of money as compensation is going to be on rise coming days.

Preventive steps for emergency of sudden death

Keep crisis trolley along with designated team member to communicate to relatives.

1. Keeping all resuscitative measures ready all 24 hours. They include emergency drug tray with drugs within expiry date. Resuscitation equipments, oxygen cylinder if there is no central oxygen, tubes, etc.
2. One person specially trained in communication skills be designated team member to talk to relatives during crisis in separate room.

3. Moment sudden unexpected cardiac arrest or respiratory difficulty arises then immediately tell politely all the relatives and bystanders to leave the ward or resuscitation room or else wheel the patient to resuscitation room.

4. Keep one of designated team member during "sudden death crisis" for informing the seriousness of the health and assurance that all measures are being taken to revive the patient.

5. Designated team member should also take additional high-risk consents, informed consents and dissents which should be in writing and need to record in writing on case papers and signed by relatives and a witness. This exercise is presumably for police and law courts to evaluate whether relatives were informed time to time without confusion.

Breaking news of sudden and unexpected death

Breaking the sad news is an art which needs to be mastered. Demeanor of Doctor has not to be polite but also dignified. This means politeness should not be construed as weakness nor doctor should show extreme emotions anger, shouting, humiliating the patient party. At least avoid crying in front the relatives. The doctor must keep his composure and show confidence in explaining the sudden death. Doctor must share empathy, sympathy and sorrow with the patient party at professional level. This may appear odd but one must remember, the more extreme emotions [either way i.e. hypo or hyper] one shows the relatives become more suspicious.

After the death is declared, all the responsible relatives must be called upon for sharing information. At this point remember to call out only responsible relatives and talk to them in a separate room preferably your consulting room. The relatives must be taken into confidence, the situation explained and if cause of death is obvious, it must be explained properly. Can we prevent allegations of MN?

It is surprisingly true that inspite of wearing good, empathetic, sympathetic attitude & observing courteousness in communications allegations of MN in SUD may put medical professional in a ML maze of alleged MN and leave them disgusted. Medical professional s feel they are framed in alleged MN even though there is no ML issue in SUD. A new breed of legal advisors in ML issues of MN on internet are on rise who lure relatives of SUD and show them big money in prosecuting doctor which cannot be traded off by medical professional wearing good, empathetic, sympathetic attitude & observing courteousness in communications in event of SUD.

WHY ML PROBLEMS ARE ON RISE IN THE EVENT OF SUD:

As such two decades ago a SUD in a nursing home or hospital would not invite much hue and cry. Ever since branch of medicine has matured & ushered in new era of critical care (CC) and emergency treatment (ET) with advent of better patient monitoring facilities and advancement in knowledge and skills CC & ET. A number of small to big tertiary care centers in the form of neonatal / premature intensive care units (NICU), intensive care units (PICU) with huge investment in infrastructure have come up, even in small towns and talukas apart from big cities in India. State of art infrastructure costs a fortune and escalates cost of quality care in treatment. Understanding ML mindset of courts in case of SUD in alleged MN

In today's scenario medical professionals are assuming role of health risk managers of ill patients. So often, patients are prone to get in to acute crisis for various hidden, unexplained, dormant reasons not predictable or detectable to ordinary examination and investigations. Law differentiates between what was the original disease, which pushed the patient in to acute crisis. Law also probes in, why original disease pushed patient in to acute crisis. Law finds out whether it was it lack of treatment or delay in treatment, which pushed the patient in to acute crisis or as such nature of disease was such that it galloped its way to acute crisis leaving no time for medical professional to treat it. Law also finds out who was responsible for delay in treatment or for lack of treatment pushing patient in acute crisis. Sometimes under treatment and apparently well patient is pushed in acute crisis due to anaphylaxis caused by a drug. Sometimes post surgical complication of internal bleeding or severe sepsis puts the patient in to crisis. In the event of SUD in such situations if there is alleged MN and it comes up for hearing in the law courts then courts find out after developing of complication what remedial measures were taken in the interest of patient. Was patient referred to proper specialist and proper medical care was made available to patient in acute crisis as soon as possible. Medical professional should keep this in mindset of courts and accordingly doctors should be careful enough to reflect this mind set in case paper writings. Case paper writings showing abovementioned details as required by courts are helpful in pleading and proving no MN of doctors in courts. Any action or inaction (act of omission or commission) of medical professional accelerates or increases the health risks of a critically ill patient may result in an allegation of breach of duty of medical professional. Amount of money asked is mind-boggling. Patients may sue a

medical professional for compensation by asking usually lakhs of rupees and sometimes in crores.

How to tackle situation of SUD when it occurs in presence of lot many relatives:

If there are lots many relatives when SUD then immediately divide medical professionals working team in to two parts. One team shall continue "So called" sham treatment in SUD till relatives are explained about SUD and are satisfied and then only death is declared. Second team tackles the leader amongst relatives about SUD by telling that our team of other medical professionals is trying hard. Try and make relatives understand and differentiate between the basic disease leading to acute crisis and explain that monitoring vitals in not treatment but guiding doctors in treating illness and maintaining vital parameters. Medical professional should wear empathetic, sympathetic attitude & observing courteousness in communications while explaining SUD to relatives.

Making relatives of patient party understand SUD:

ML problems related to SUD in patient has become commonplace with the advent of better patient monitoring facilities and advancement in knowledge in maintaining vital parameters till basic pathology causing havoc in critically ill patient is managed of by appropriate specific treatment. Doctor's perspective in treating fervently acute conditions causing destabilization of vitals and not so vigorously addressing basic pathology in tandem creates confusion in the minds of relatives of patient. So often just monitoring and maintaining vitals cost a fortune, where as relatives perceive as if specific treatment for basic disease was going on. e.g. Septicemia due to a large abscess destabilizing vitals where abscess is not drained because of unstable vitals withstanding anesthesia. Sometimes vitals remain unstable and original disease pathology dominates so much that it reaches to point of no return and patient develops SUD. This misunderstanding of relatives in not able to differentiate between maintaining vitals by monitoring costs are different from medical treatment cost for treating basic pathology. So often SUD occurs causing confusion of relatives as to what needed to be done by doctor was not done and doctor wasted time in monitoring vitals and did nothing for basic disease. This mix up of laymen thinking & loose talk by hospital staff gets jumbled up with misunderstanding and misinterpretation of unpalatable scientific explanations given by treating doctor regarding treatment of basic disease and CC & ET for maintaining vitals pave its way to ML cases.

Even though CC & ET has significantly advanced in field of medicine, which has saved unsalvageable, lives but ushered every one of us in high cost treatment arena. High cost of critical care most often results in cure but when it leads to SUD, as outcome then it becomes source of ML problems. It is not uncommon to see or hear or read in news papers that relatives took law in their own hand e.g. Sinhania Hospital Thane was turned to ashes after SUD of local shiv sena politician Shri Anant Dighe, It is not true for high profile SUD, but also true for common people facing SUD culminating in abuse of medical person & their property. We see alleged ML cases being registered as first information report (FIR) under S. 154 of Criminal procedure code (Cr. PC) for SUD at local police stations all over the India and police arresting doctors under Section 304 A of Indian penal Code (IPC). This tendency of arresting doctors for SUD is brought under control by supreme court decision of Jacob Mathews versus State of Punjab Even the fourth estate is not much behind in reporting SUD in TV, press every day in defamatory way against doctors. Each such news articles shakes medical professional to core, who wishfully thinks, god, let this not happen to me. Let me practice defensively. This has eroded confidence of innovative and motivated dedicated doctors. It is not a good signal. Already existing S. 304 A of IPC has got a boost in prosecution of doctors by enactment of Consumer protection Act-1986 (CPA) and it has accelerated prosecutions related to alleged ML. Both criminal and civil remedies are on fast tract of judicial remedy in alleged ML cases. Already existing criminal courts along with establishment of consumer courts has put the cases of alleged MN on fast tract remedy. There is no limit set by legislature & judiciary for asking of compensation by prosecution for alleged MN cases. Patient party approaching police stations and law courts is better than they taking law in their own hands.

What to do before declaring death in SUD?

“So called” sham treatment should continue in SUD till relatives are completely satisfied and disperse. At the same time see that all documentation of clinical notes is complete. Preserve all empty vials of injection and injection syringes. Collect 10 ml of blood in plain bulb, EDTA bulb and Sugar bulb and label them. This is to meet the allegation of relatives that either a poison or overdose of medicine was given.

When to declare death in SUD:

Where number relatives slowly increase as the time passes in hospital premises when “so called”

treatment in SUD is going on. If number increases then one should surely inform police for self protection as well as safety of property. Declare finally death in SUD cases in front of police and hand over body of patient with collected blood samples and empty injection vials and ampoules and syringes. Do not move the body of SUD from OT to ICU if patient dies in OT.

No loose talk in SUD

Warn doctors and staff members to be careful of loose talk about SUD as it may spark physical abuse of medical team or destruction of hospital property.

Reason for “so called sham treatment”

Declaration of death in SUD should be essentially preceded by “so called sham treatment”, even putting in intravenous line or starting nasal oxygen or even putting on respirator is a good trick. This will buy time and wisdom for medical team to declare death in SUD at the terms and conditions desired by medical team rather than getting swept away by unruly behaviour of relatives by untimely declaration of death in absence of police. Kindly do not charge for treatment as it may spark another fury of relatives.

Sudden death may lead to some odd doctor and patient party interactions:

1. Run away from the situation, go underground or hide somewhere is practiced by some doctors in north India till situation cools down. It is double edged sword in longer run. Never do this. It will only make the things worse.

2. Immediate cover up and unnecessary explanation should be avoided skillfully. The doctors should not face multiple relatives immediately and start giving explanations. The paramedical staff should also be told to keep shut so as to avoid different versions. One should keep nerves under control, take a break for few minutes after exhaustive resuscitative efforts have failed, organize the things in OT, pool all the empty vials used during resuscitative measures, take a look at the papers and take them into your custody, (this is to prevent unauthorized copying of the papers without knowledge of anybody). Later on, one should call only the responsible relatives separately into the counseling or consulting room and explain the situation.

3. Most doctors in small towns in small nursing homes are forced to settle the matter as early as possible by political pressure by yielding to financial demands. This is the most common practice adopted by doctors but this should be as far as possible avoided. Such setting of precedence of coughing up money paves

way in future to mischief mongers and political leaders.

4. One should avoid fighting the situation overconfidently by angry speech or with angry body language.

5. Dismissing the relatives and not giving proper explanation further aggravates the situation.

6. Retaliate the patient party by replying each abuse by counter abusive language will lead to the worst situation since it is not expected of a doctor. The dictum is the relatives may abuse in any language, you cannot reply in the same language as your abusive language shall be capitalized immediately and more and more abuses start getting hurled at you. This also increases the tension in the atmosphere and temperatures start running very high. Sometimes in the heat of the moment patient party is threatened that police, politicians and judiciary is in your pocket so you brush aside patient party by telling [yelling] to do what they want. This angers the patient party and makes them revengeful.

For documentation read chapter no. 8

Unacceptable and ugly behavior of patient party in small nursing homes

- Manhandle the doctor, destruction of the property and hospital equipments
- Threatening the doctor.
- Lodge a criminal complaint.
- Insist on arrest of the doctor.
- Invite the media to spread the news.
- Harassment of doctor through local politicians and mischief mongers.
- Demand for money.

Sudden Death not due to negligence

Sudden is defined as happening quickly and without any warning while, sudden death is defined as instantaneous, unexpected death not caused by negligence. The important ingredient here is “not caused by negligence”. Needless to say, death caused during the course of treatment when it is most unexpected is sudden death. The sudden death could be caused during ward treatment or on operation table. We have to deal with these two types of deaths differently.

Death in Ward or Room during the Course of Treatment just to enumerate the causes of sudden and unexpected death are:

1. Anaphylactic shock
2. Embolism due
 - i. Thrombus
 - ii. amniotic fluid
 - iii. air
3. Endotoxic shock
4. Hemorrhagic shock
5. Neurogenic shock
6. Cardiogenic shock
7. Anesthetic complications: as Mendelson’s syndrome.

Such death is usually postoperative or due to unexpected turn in the health of a patient being treated in ward or room. Such death usually happens to be unattended by treating physician and non-availability of paramedical staff creates more problems

Intra-operative Death in OT or Immediate Postoperative Death in Recovery Room and Mob Violence

This again is a very difficult situation particularly in elective operation are performed on otherwise healthy patients with surgical and anesthetic fitness. Majority of the times the patient enters OT in stable physical and mental condition and a lifeless body is brought out on a gurney. This is really shocking to relatives who are not prepared for such eventuality. The story is different in high-risk cases, accidents, and complicated surgeries wherein the relatives are prepared for eventuality one way or the other.

The reaction of the relatives to such sudden death is:

- Anger and anguish.
- Immediate explanation—relatives demand immediate explanation on the spot which is not possible for the doctors and the agitation and suspicion in the minds of relatives aggravates.
- Financial waver and immediate compensation.
- Immediate police action—this usually is demanded by so called leaders of that particular area or community. They pressurize the police to register crime and make arrest of the doctor immediately. However, with the recent Supreme Court judgments, immediate arrest of doctors is not possible in case of death caused during the course of treatment. • Verbal or physical abuse and immediate retaliation with destruction of property.

Legal proof of documenting continuous monitoring:

Very often when treatment charts and case papers are reviewed by court, when a case of MN related to SUD is heard, it finds that so often medical professional s claim that they did continuous monitoring of patient related to heart rate / pulse, blood pressure, respiratory rate, temperature, oxygen saturation and electrocardiograph but clinical notes of case papers do not reflect any mention of these readings. Case papers do not showing any entry of vital parameters makes court to conclude that monitoring was not done Document clinical findings and obtain written consents & dissents

Very often when treatment charts and case papers are reviewed by court, when a case of MN related to SUD is heard it finds that so often medical professionals were busy treating acute cardiac failure, acute respiratory distress, acute renal failure, acute liver failure, acute severe brain edema and acute shock to maintain life. Court finds and points out that causative disease took a back seat while vigorous treatment for acute crisis was going on. So along with treatment of acute crisis clinical notes also write notes related to basic pathology being treated on day-to-day basis and it should be reflected in clinical notes and it should appear as if both acute crisis and basic pathology were being treated with same vigor. Similarly relatives need to be explained about need to monitor and maintain vitals and it does not constitute the treatment of basic pathology. Medical professional should make it abundantly clear to relatives that monitoring vitals is different from treating the original basic disease. High-risk consents, informed consents and dissents need to be recorded in writing on case papers and signed by relatives and a witness to allow court to evaluate whether relatives were informed time to time without confusion.

For documentation read chapter no. 10

Legal view regarding diagnosis:

We as doctors view diagnosis with great respect. Making a accurate diagnosis makes a doctor feel as if he has reached to zenith of his mental abilities. Courts are not very serious about accuracy of diagnosis but at the same time are not ready to condone palpably wrong diagnosis. Very often when case papers are reviewed by court, when a case of MN related to SUD is heard and so often medical professional s vehemently claim that they made accurate diagnosis so there is no MN on the part of doctor. Even if diagnosis is not accurate but usual conduct of care of patient by doctor is correct then courts take lenient

view regarding making errors in diagnosis of main disease. This is because so many diseases present to medical professional with common signs and symptom complexes and medical professional may make an error of judgement, if the disease presents with rare, atypical signs and symptoms. Medical professional has liberty to choose treatment after arriving at tentative diagnosis. This liberty is related to causative disease and not to acute emergency syndrome management.

Deficiency in attending the patient and conducting a procedure Very often when treatment charts and case papers are reviewed by court, when a case of MN related to SUD is being heard it finds that so often medical professional s did not attend the patient then courts take very strict view. Courts take very strict view if there is deficiency in procedure of putting intravenous lines, intra gastric tubes, urine catheters, and tracheal intubation. Courts take very strict view if there is deficiency in continuous monitoring system readings related to pulse/ heart rate, blood pressure, respiratory rate, temperature, oxygen saturation and electrocardiograph finding no entry in clinical notes on case papers. Deficiency in procedures or conducts of treatment of acute cardiac failure, acute respiratory distress, acute renal failure, acute liver failure, acute severe brain edema and acute shock or anaphylaxis to maintain life is also taken as gross negligence. If SUD occurs due to such breach of duty of medical professional in emergency room then he may have to defend himself from the charges of MN in courts. Since treatment costs are high and results of critically ill sometimes may result in SUD so compensations asked by patient litigant are astronomical. Asking postmortem

If there is doubt as to cause of death, postmortem examination be proposed. It takes a lot of efforts to convince the relatives about the necessity of postmortem but it is worth taking the efforts than to avoid legal trouble later on. If doctor feels postmortem is a must and relatives are not giving consent one can take help of the police and get it done.

Indemnity insurance

Ultimate answer lies in insuring oneself for professional work with insurance companies. This article is designed to reduce the trauma accompanied with alleged MN in SUD, where there is no MN of doctor. Some knowledge of ML aspects of SUD may sharpen your record keeping skills and also communication skills while dealing with relatives, police, and politician or for that matter a goon walking in your office. Medical indemnity insurance policy is the only way out to practice medicine and only safe

way to practice emergency and critical care medicine peacefully even when SUD occurs. Looking in to such an unequal & odious medico-legal scenario where risks of litigations shall continue to increase in days to come. Then where lies the answer? Highest level of record keeping and best communication skills may not be too effective to trade off the compensation benefits patient party gets by dragging you in court of law. This is not a corollary or licence for improper record keeping or arrogant communication with patient party. These risks could possibly be managed and if any claim arises it could be paid by buying indemnity cum hospital error and omission policy issued by private and public insurance companies in India.

LIKELY SITUATIONS OF ML IMPORTANCE IN SUD REQUIRING INFORMATION TO POLICE

SUD IN ML CASES:

ML situation where one may get patients who mysteriously develop SUD either after entry to emergency unit / room / ward or sometimes afterwards. Medical professional should continue to treat patient with meticulous history recording, examination, investigations needed and treatment as per reasonable norms of medical practice. As per ML norms it is mandatory to inform law enforcers and/or legal authorities (Usually it is local police station). Why? Because medical professional's duty is to receive and treat the ill / critical patient and duty of police is to find out whether any crime was committed on patient victim/patient for making him/her suffer from problems listed below:

SUD in Tetanus, gas gangrene, significant burns, head injuries, significant violence needing indoor admission, motor vehicular and other accidental fractures, accidental falls needing indoor admission, attempted suicides, attempted poisoning, attempted homicide, Human or animal or snakebite, rape, minor's pregnancy and MTP, battered baby. Insist for post mortem in SUD.

SUD in poisoning

SUD in case of attempted/ alleged/suicidal/homicidal or accidental poisoning medical professional is duty bound to preserve specimen stomach wash if it is done as per protocol (usually 100ml or more in a clean glass bottle), blood samples in EDTA and plain bulbs (usually 2ml each), as applicable and feasible and hand it over to police with proper labeling of name, sex, age, time of collection, brief or detailed history as per the direction of police and full treatment record be given. In case of SUD due to poisoning insist for post mortem. Law demands consent for post mortem to be obtained from relatives by

postmortem performing doctor and not by treating medical professional.

Following are other SUD's which needs to be informed to police:

1. SUD in indoor admitted patient falling from cot or in hospital bathroom
2. SUD on operation table or SUD in post operative patients
3. SUD resulting from anaphylaxis due to a drug
4. SUD due to Steven Johnson syndrome
5. SUD as a Post procedure event, for example after lumbar puncture, liver biopsy and other biopsies.
6. SUD due to internal or incessant external bleeding and disseminated intra vascular coagulation.
7. SUD during or Post anesthesia

How to transport sick and serious dying patient:

1. Medical professional and nurse team should accompany the dying patient.
2. Ambulance should have enough variety and stock of emergency medicines, injections, intravenous fluids and oxygen
3. Monitoring equipments like stethoscope, blood pressure instrument, cardiac monitor machine with preferably a defibrillator and a Ventilator
4. In case SUD occurs bring back the patient to your hospital and declare death after proper explanations satisfying the relatives.

Legal standards of reasonable medical care in emergency rooms in case of SUD:

Standard of medical care in emergency room are higher because emergency rooms care claims giving state of art services being given to patient admitted as below:

1. Duty of care in emergency room (which means actively avoiding all kinds of dangers i.e. health risks from all sources i.e. from disease, drugs and surgery, all the time) to your patients by continuous monitoring of all relevant vital parameters & investigations to avoid any further acceleration in disease process.
2. Law requires higher proportionate degree of medical care in emergency room. Higher the risks undertaken then higher are the standard of monitoring and medical procedural skill demanded by law in caring for critically ill.

3. Any lack or shortcoming or deficiency of medical care on the part of medical practitioner in monitoring or treatment of critically ill, medical professional's actions, which causes acceleration of disease process leading to SUD, is actionable under law. Under law for actionable negligence, such breach of duty should cause acceleration of SUD.
4. There should be close nexus between negligence leading to acceleration of SUD.

For documentation read chapter no. 17

ABOUT CONSENT, DISSENT, ASSENT, COUNSELING, FOREWARNING IN SUD

1. In emergency rooms standards for consent, informed are much lower than usual cold situations. In dire emergency courts waive of consent in favour of giving lifesaving treatment, even though nature of treatment may amount to adventure. In a case of a road side accident victim's (In case of accident victim court takes very strict view if no attempt is made to save life), vitals were stabilized by giving emergency treatment before shifting to higher center, where one limb had to be amputated because of delay in referral, court did not hold medical professional negligent in causing delay in referring because stabilization of vitals were crucial before transfer of patient otherwise patient would have suffered SUD during transit. Defendant would be liable for such SUD occurs if vitals were not stabilized before transferring. In another case of vehicular accident, a reasonable delay in preparing for operation and arranging for 19 bottles of blood was permitted by court even though patient postoperatively suddenly died
2. Some times in emergency omission to perform operation for want of consent may amount to negligence. Here emergency appendectomy not done, for want of consent nor dissent from patient taken in writing, doctor was held liable. In this case appendix later burst and SUD occurred. Remember written dissent is more important than consent for invasive procedure, surgery, investigation, transfer and referral in emergency situations

For documentation read chapter no. 12,13,14,15

ABOUT MONITORING AND RECORD KEEPING PRIOR TO SUD OCCURRENCE

1. Monitoring serious patients by keeping record and using available gadgets and investigations or refer by providing ambulance to transfer. Bottom line for monitoring is recording pulse, respiration temperature, blood pressure, and intake and output chart.
2. Remember proper record is valid defence in MN case as the law asks for show of care rather than cure

ABOUT CRITICALLY ILL PATIENTS WHERE "KNOWN COMPLICATION" WHICH CAN NOT BE PREVENTED PRIOR TO SUD OCCURRENCE

Son bitten by cat. ARV given. He developed neuro-paralytic reaction. Hospitalized SUD occurred in ICU. No negligence as standard textbooks and WHO report of 1984 mentions Neuro-paralytic reactions well known complication of ARV. Proper ICU treatment given with care.

ABOUT CASES RELATED TO ANAPHYLAXIS CAUSING SUD

- Medical professional did not do penicillin test dose also did not keep emergency medicines ready for treating anaphylaxis of penicillin and failed to treat complication, patient developed SUD, medical professional held negligent for not treating complication.

EMERGENCY BLOOD TRANSFUSION PRIOR TO SUD OCCURRENCE:

Sometimes emergency blood transfusion may belong to wrong blood group causing mismatch transfusion reaction leading to SUD It is better to be safe than sorry by following proper blood checking norms.

MEDICO-LEGAL ASPECTS OF EUTHANASIA, DNR, VEGETATIVE LIFE AND ACTIVE AND PASSIVE WITHDRAWAL OF LIFE SUPPORT SYSTEMS

Advance directives / Do-not-resuscitate (DNR) instructions by patients [not valid in India after SC's Aruna Shanbhag's judgement]

- a. Take a high-risk consent from the patient party for continuing neonatal resuscitation in spite of failure to resuscitate after 10 minutes.
- b. Record specifically in the consent the patient's advance directive/s if parents want the neonatal resuscitation be discontinued at the end of 10 minutes.
- c. Take signature of two independent witnesses on

DNR document.

Removing active life support system and passively keeping alive patient by feeding tubes, etc.

- a. Make sure that the patient is brain dead as per guidelines given in HOTA-1994
- b. Take high court order after taking consent from the patient's closest relative and caretaker that in the given circumstance it is no use continuing active or passive life support.
- c. Take signature of two independent witnesses.

Information for parents and families about ventilator withdrawal

- a. The pediatrician's counseling of families is a critical aspect of care for the dying patient who is to be removed from a ventilator. Ideally near and dear in the family should be involved in the decision to withdraw the ventilator. Before withdrawal, the following issues should be discussed.
- b. Potential outcome of ventilator withdrawal. Assuming all other life-sustaining and supportive treatments are stopped, along with artificial hydration and supplemental nutrition, there are several potential outcomes: rapid death within minutes or death within hours to days and very rarely patient may survive also.
- c. The procedure of ventilator withdrawal: Never make assumptions about what the family understands; describe the procedure in clear, simple terms and answer each and every question. Near and dear family members should be informed in advance about the steps of withdrawal and whether or not it is planned/ desired to remove the endotracheal tube. In addition, they should be told in a simple way that the use of oxygen and medications are for symptomatic relief. Make sure that the family should be told by pediatricians that the primary concern is painless patient's comfort even if it is end of life situation. Tell them that breathlessness may occur, but that can be managed. Always make sure that you have medications available to manage all discomforts. Also brief them that the patient is most likely to need the medicines to be kept asleep for controlling symptoms.

If asked, explain that they can show love and support by touching, wiping of the patient's cheeks, forehead, holding hand or talking to child, if conscious.

- d. Support the decision: Even though a family is able to make a definite decision for ventilator withdrawal, such decision remains emotionally highly charged. Families shall keep breeding second-thoughts and guesses in their minds, especially if the death appears to linger on and on following ventilator withdrawal. Pediatrician support, guidance and leadership are crucial, as the family will be looking to the pediatrician to ensure them that they are "doing the right thing at right time". They need constant support following death during the usual bereavement period, which is usually is emotionally traumatic.

MEDICO-LEGAL ASPECTS OF DIFFICULT SITUATIONS, DECLARATION OF DEATH AND COMMUNICATION WITH PATIENT PARTY

Most legal problems in health care systems arise from poor communication hence good communication can play a significant part in avoiding complaints and malpractice claims.

One big Barrier to good communication Arrogance and paternalistic automatic antagonism towards patient party is deeply ingrained into doctors. Doctors presume that they know the best about their patient hence they always issue commands. Doctors expect patients and their parents to follow commands unquestioningly. Doctor should willingly change this situation by being more adaptive and communicating.

Delivering Bad News

It is critical that the doctor is well prepared with professional explanation and skills of communication to deliver bad news or declare death. Some of the important points are:

- (a). Perceiving self-reflection: Pediatricians will invariably have strong negative emotions when they have to give bad news. In near death situations, pediatrician should not spontaneously discuss their own emotional reaction with a preceptor; therefore they should be introducing this topic. "This is a really very difficult and hard medical case, and what is being done medically for patient is challenging, yet patient is not at all responding as expected"
- (b). Create an appropriate setting: Say the child is serious and every bit of best is being continuously done.
- (c). Make sure you know basic information about the patient's disease, current situation, prognosis, and treatment options before delivering medically bad news.

Situations Requiring Extra Caution

Medical professional should keep in mind certain high-risk situations, which are common causes for medical negligence actions; situations that require extra cautions are anesthesia resulting in loss of sight, hearing, paralysis, vegetative life and death

RECOVERY OF BILLS IN SUD IN SMALL NURSING HOMES

This is again a very difficult situation and to tackle this situation you need more of social, managerial and manipulative skills rather than medical knowledge. Taking regular advance equivalent to the expected bill on that day is probably the best policy. One should not stop or discontinue treatment for nonpayment of bills so also keeping the patient in the door of OT before operation starts for the sake of advance also is not a good practice. But, when it comes to sudden death or unexpected complications which drain the resources of patient and relatives, the situation arises. Doctors currently are at the receiving end of the society for many reasons. People never understand the use of intellectual property and its encashment and always feel that we are taking money unnecessarily. This misconception needs to be removed and it will take time. Things work until they are straightforward but once something goes wrong, they bounce back upon you. Ultimately, all these difficult situations are going to stay and we have to learn to live with them, tackle them as nicely as possible and reduce our stress. In the light of basic legal knowledge, let us dispel these unfounded legal fears and do right things in right direction. Let us not give up and practice defensive medicine for fear of legal wrangles. So far as the legal aspect of the recovery and nonpayment of bills is considered, we really have very few options which are as follows:

1. Forgo the bill and forget it once for all.
2. Take an advance cheque for the remaining amount so that some legal option remains with us.
3. Reduce the bill, give concession and such things.
4. Use mediators or request acquaintances from both parties to mediate.
5. File a civil suit for recovery of the bill which requires an advocate, court fees and long waiting period not really advisable.
6. If there is previous nonpayment of bill, next time you can refuse to see or treat the patient as of right, exercise the right to refusal to see the patient.
7. One cannot hold the dead body till the bill is paid. Bombay high court denounced the "inhuman"

practice of hospitals detaining live patients till their medical bills were paid. A division bench of Justices VM Kanade and P D Kode heard a petition filed by Sanjay Prajapati urging it to direct MIDC Police, Andheri (East), to act against doctors and staff of Seven Hills Hospital, for wrongfully confining his brother over a disputed bill.

Summary: This write up is intended to provide ML information as to what a medical professional should do in the event of SUD. How a medical professional and CC & ET provider should deal with relative, police, press, politicians, social workers, lawyers and what kind of mindset the law courts pursue. The knowledge of mindset of non medical people should allow you to refocus on your clinical notes documentation. This ML knowledge and wisdom to bridge the gap of ignorance of relevant laws as applicable to practicing medical professional and CC & ET provider to prevent solve and understand the ML problems related to SUD. All of us know and have experienced in our life that ignorance breeds and feeds uncertainty. Uncertainty breeds and feeds unfounded fears. We also know unfounded fears usually never become true or actually happen in one's life but makes life stressful and unlivable. In the light of basis legal knowledge, let us dispel these unfounded legal fears and do right things in right direction. Let us not give up and practice defensive medicine for fear of legal wrangles.

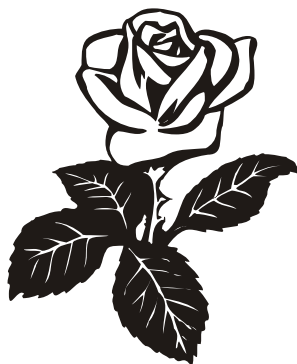
Does and don'ts

1. The difficult situations one can face after death are:
 - A. Sudden death and mob violence
 - B. Brought dead
 - C. Settlement of bills or recovery of dues.
2. Medical professional are expected to chart the course of the health of their critically ill patients with minimal health hazards
3. Keep crisis trolley along with designated team member to communicate to relatives.
4. Keeping all resuscitative measures ready all 24 hours. They include emergency drug tray with drugs within expiry date. Resuscitation equipments, oxygen cylinder if there is no central oxygen, tubes, etc.
5. One person specially trained in communication skills be designated team member to talk to relatives during crisis in separate room.
6. Moment sudden unexpected cardiac arrest or respiratory difficulty arises then immediately tell

- politely all the relatives and bystanders to leave the ward or resuscitation room or else wheel the patient to resuscitation room.
7. Keep one of designated team member during "sudden death crisis" for informing the seriousness of the health and assurance that all measures are being taken to revive the patient.
 8. Designated team member should also take additional high-risk consents, informed consents and dissents which should be in writing and need to record in writing on case papers and signed by relatives and a witness.
 9. If there is doubt as to cause of death, postmortem examination
 10. Ultimate answer lies in insuring oneself for professional work with insurance companies.
2. Enumerate few causes of sudden and unexpected death in clinical practice
 - a) Anaphylactic shock
 - b) shock due to Embolism
 - c) Endotoxic shock
 - d) Hemorrhagic shock
 - e) All of above Ans. e)
 3. Deaths which needs to be informed to police:
 - a) Falls in hospital
 - b) operation table death
 - c) death due to drug anaphylaxis
 - d) death due to accident of any type
 - e) all of the above ans.

MCQ

1. What to do before declaring death in SUD
 - a) treatment continue in SUD till relatives are completely satisfied
 - b) documentation is complete.
 - c) Preserve all empty vials of injection and injection syringes.
 - d) Collect 10 ml of blood in plain bulb, EDTA bulb and Sugar bulb and label them.
 - e) All of the above Ans. e)



APPROACH TO THE CANCER PATIENT

Dr. Anish Maru

DM (Medical Oncology)

Sarvodya Multispeciality & Cancer Hospital, Hissar

Cancer is the second most common cause of death in developed countries.

In India approximately 10,00,000 new cases of cancer are reported yearly and 800,000 die due to cancer.

In males Head & Neck cancer is the most common cancer followed by Lung Cancer. In females breast cancer is the most common cancer followed by cervical cancer.

- 1) Signs & Symptoms of cancer : (warning signals)
- A) Painless Lump in breast, neck, axilla or groin.
 - B) Non healing in mouth & tongue.
 - C) Abnormal bleeding-vagina, cough, vomiting, urine, rectal bleeding, gum bleeding
 - D) Hoarseness of Voice - Cancer of Larynx & Lung.
 - E) Difficulty in swallowing.
 - F) Sudden increase in size of male
 - G) Abdominal distension.
 - H) Unexplained fever & weight loss.

If any of above symptoms are presented by a patient then he/she should be investigated to rule out malignant process.

Once suspected to have cancer, various investigations can be performed depending upon presenting symptoms of the patient.

Routinely following investigations may be helpful

- A) CBC, KFI, LFT, May give a..... lymphoma & multiple myeloma
- B) CXR-PA
- C) USG - Whole abdomen
- D) Mammography of Breast
- E) CFCT NECK, CHEST & ABDOMEN (As Required)
- F) MRI of Neck, Brain
- G) PET-CT Scan whole body
- H) DL Scopy/upper GI endoscopy/colonoscopy cystoscopy etc.
- I) FNAC of any lump.
- J) Biopsy of or growth or lump.

In addition to above investigations further specialised investigations may be required as per the disease to decide treatment.

- A) Breast Cancer - ER, PgR, Her-2-, Ki-67%
- B) Lung Cancer - (Non Squamous) - EGFR mutation, ALK, ROS, MET, PD-I, PDL-I
- C) Leukemia - Flow cytometry, cytogenetic.
- D) Multiple myeloma - S. Protein Electrophoresis, Ig G, Ig M, Ig A, Free light chain assay, S.B2..... Bone Marrow aspiration biopsy.
- E) Immunohistochemistry.
- F) Tumour Markers : PSA, S.CA-125, S.CEA, AFP, B-HCG, S.CA-19.9, LDH, S. CA 15.3.

Once diagnosis is established then stage is confirmed by doing various CT scan or PET-CT scan.

Treatment of cancer is always a multimodality approach involving chemotherapy, radiotherapy & surgery in different combinations and sequence depending upon stage of patient.

Early stage (1&2) the primary treatment is surgery followed by adjuvant chemotherapy +/- radiotherapy.

Locally advanced (stage 3) is treated with neoadjuvant chemotherapy radiotherapy followed by surgery.

Metastatic disease (stage 4) is treated for palliation of symptoms by surgery or radiotherapy or palliative chemotherapy.

Screening of Cancer:

Cancer is curable if detected early so screening for cancer can be done in asymptomatic healthy people to diagnosed cancer in premalignant conditions or early stage of cancer.

The most common diseases for screening are cervical cancer & breast cancer.

Cervical cancer screening is done by PAP SMEAR examination & COLPOSCOPY immediate after starting sexual intercourse.

Breast cancer screening is done by mammography with USG once a year from 45-55 yr age, group

EMERGENCY MANAGEMENT OF ACUTE UPPER GI BLEEDING

Dr. Gursewak Singh

MBBS, MD, DM (gastroenterology)
Care Hospital, Bhatti Road, Bathinda

Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis (vomiting of blood or coffee-ground-like material) and/or melena (black, tarry stools). The initial evaluation of patients with acute upper GI bleeding involves an assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow, with the goal of both diagnosis, and when possible, treatment of the specific disorder.

Initial Evaluation —

The initial evaluation of a patient with a suspected clinically significant acute upper GI bleed includes a history, physical examination, laboratory tests, and in some cases, nasogastric lavage. The goal of the evaluation is to assess the severity of the bleed, identify potential sources of the bleed, and determine if there are conditions present that may affect subsequent management. The information gathered as part of the initial evaluation is used to guide decisions regarding triage, resuscitation, empiric medical therapy, and diagnostic testing.

Factors that are predictive of a bleed coming from an upper GI source include history of melena, melenic stool on examination, blood or coffee grounds detected during nasogastric lavage, and a ratio of blood urea nitrogen to serum creatinine greater than 30. On the other hand, the presence of blood clots in the stool made an upper GI source less likely. Factors associated with severe bleeding included red blood detected during nasogastric lavage, tachycardia, or a hemoglobin level of less than 8 g/dL.

Bleeding manifestations —

Hematemesis (either red blood or coffee-ground emesis) suggests bleeding proximal to the ligament of Treitz. The presence of frankly bloody emesis suggests moderate to severe bleeding that may be ongoing, whereas coffee-ground emesis suggests more limited bleeding. The majority of melena (black, tarry stool) originates proximal to the ligament of Treitz (90 percent), though it may also originate from the oropharynx or nasopharynx, small bowel, or right colon. Melena may be seen with variable degrees of blood loss, being seen with as little as 50 mL of blood. Hematochezia (red or maroon blood in the stool) is usually due to lower GI bleeding. However, it can occur with massive upper GI bleeding, which is typically associated with orthostatic hypotension.

Past medical history:

Potential bleeding sources suggested by a patient's past medical history include:

- Varices in a patient with a history of liver disease or alcohol abuse
- Peptic ulcer disease in a patient with a history of NSAIDs use or smoking.
- Angiodysplasia in a patient with renal disease.
- Ulcers at an anastomotic site in a patient with a gastroenteric anastomosis.

Comorbid illnesses -May influence patient management e.g.:

It may make patients more susceptible to adverse effects of anemia as in CAD.

It may predispose patients to volume overload in the setting of vigorous fluid resuscitation or blood transfusions as in renal disease or heart failure).

It may result in bleeding that is more difficult to control as in coagulopathies, thrombocytopenia, significant hepatic dysfunction.

It may predispose to aspiration as in hepatic encephalopathy. Symptom assessment —Severe bleeding is suggested by orthostatic dizziness, confusion, angina, severe palpitations, and cold/clammy extremities. Specific causes of upper GI bleeding may be suggested by the patient's symptoms:

Peptic ulcer: Upper abdominal pain

Esophageal ulcer: Odynophagia, gastroesophageal reflux, dysphagia

Mallory-Weiss tear: Emesis or retching prior to hematemesis

Variceal bleed: Jaundice, abdominal distention (ascites)

Malignancy: Dysphagia, early satiety, involuntary weight loss, cachexia

Physical examination — Signs of hypovolemia include:

Mild to moderate hypovolemia (less than 15 percent of blood volume lost): Tachycardia.

Blood volume loss of at least 15 percent: Orthostatic hypotension

Blood volume loss of at least 40 percent: Supine hypotension.

Examination of the stool color may provide a clue to the location of the bleeding, but it is not a reliable indicator. Nasogastric lavage may be carried out if

there is doubt as to whether a bleed originates from the upper GI tract. Laboratory data — Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies. The initial hemoglobin level in patients with acute upper GI bleeding will often be at the patient's baseline because the patient is losing whole blood. With time the hemoglobin level will decline as the blood is diluted by the influx of extravascular fluid into the vascular space and by fluid administered during resuscitation. Patients with acute bleeding should have normocytic red blood cells. Microcytic red blood cells or iron deficiency anemia suggest chronic bleeding. Patients with acute upper GI bleeding typically have an elevated urea-to-creatinine ratio.

Nasogastric lavage — Nasogastric tube lavage was associated with a shorter time to endoscopy. More often, it is used when it is unclear if a patient has ongoing bleeding. Gastric lavage may not be positive if bleeding has ceased or arises beyond a closed pylorus. It is suggested that patients only undergo NGT lavage if particulate matter, fresh blood, or clots need to be removed from the stomach to facilitate endoscopy.

GENERAL MANAGEMENT

Triage — All patients with hemodynamic instability (shock, orthostatic hypotension) or active bleeding (manifested by hematemesis, bright red blood per nasogastric tube, or hematochezia) should be admitted to an intensive care unit for resuscitation and close observation with automated blood pressure monitoring, electrocardiographic monitoring, and pulse oximetry

General support — Patients should receive supplemental oxygen by nasal cannula and should receive nothing per mouth. Two large caliber (18 gauge or larger) peripheral intravenous catheters or a central venous line should be inserted. Elective endotracheal intubation in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration.

Fluid resuscitation — Adequate resuscitation and hemodynamic stabilization is essential prior to endoscopy to minimize treatment-associated complications. Patients with active bleeding should receive intravenous fluids (eg, 500 mL of normal saline or lactated Ringer's solution over 30 minutes) while being typed and cross-matched for blood transfusion. Patients at risk of fluid overload may require intensive monitoring.

Blood product transfusions —The decision to

initiate blood transfusions must be individualized. Our approach is to initiate blood transfusions if the hemoglobin is <7 g/dL (70 g/L) for most patients. However, our goal is to maintain the hemoglobin at a level of ≥ 9 g/dL (90 g/L) for patients at increased risk of suffering adverse events such as those with unstable coronary artery disease or in those with evidence of ongoing active bleeding. Transfusing patients with suspected variceal bleeding to a hemoglobin >10 g/dL (100 g/L) should be avoided as it can precipitate worsening of the bleeding. Patients with active bleeding and a low platelet count ($<50,000/\text{microL}$) should be transfused with platelets. Patients with INR >1.5 should generally be transfused with fresh frozen plasma.

MEDICATIONS

Acid suppression — We suggest that patients with acute upper GI bleeding be started empirically on an intravenous (IV) PPI (eg, esomeprazole 40 mg IV twice daily after an initial bolus of 80 mg IV). The PPI can be started at presentation and continued until confirmation of the cause of bleeding. Once the source of the bleeding has been identified and treated (if possible), the need for ongoing acid suppression can be determined. In the setting of active upper GI bleeding from an ulcer, acid suppressive therapy with H₂ receptor antagonists has not been shown to significantly lower the rate of ulcer rebleeding. By contrast, high dose I.V. infusion of a PPI significantly reduces the rate of rebleeding compared with standard treatment in patients with bleeding ulcers.. In addition, PPIs given as intermittent boluses appear to be at least as effective as PPIs given as a continuous infusion. Oral and intravenous PPI therapy also decrease the length of hospital stay, rebleeding rate, and need for blood transfusion in patients with high-risk ulcers treated with endoscopic therapy. PPIs may also promote hemostasis in patients with lesions other than ulcers, likely because neutralization of gastric acid leads to the stabilization of blood clots .

Prokinetics —

Both erythromycin and metoclopramide have been studied in patients with acute upper GI bleeding. The goal of using a prokinetic agent is to improve gastric visualization at the time of endoscopy by clearing the stomach of blood, clots, and food residue. Erythromycin promotes gastric emptying based upon its ability to be an agonist of motilin receptors. It is suggested that a single dose of intravenous erythromycin given 20 to 120 minutes before endoscopy can significantly improve visibility, shorten endoscopy time, and reduce the need for second-look endoscopy. Treatment appears to be safe. Patients who received erythromycin were significantly more

likely to have an empty stomach at the time of endoscopy. Erythromycin has also been shown to be comparable with nasogastric lavage in a randomized trial with 253 patients.

Vasoactive medications —

Somatostatin, its analog octreotide, and terlipressin are used in the treatment of variceal bleeding. Terlipressin is preferred because of its ease of administration and better safety profile.

Octreotide is not recommended for routine use in patients with acute nonvariceal upper GI bleeding, but it can be used as adjunctive therapy in some cases.

Antibiotics for patients with cirrhosis —

Multiple trials suggest clear value of prophylactic antibiotics in cirrhotic patients hospitalized for GI bleeding. Patients with cirrhosis who present with acute upper GI bleeding (from varices or other causes) should be given prophylactic antibiotics, preferably before endoscopy.

Tranexamic acid —

Tranexamic acid is an antifibrinolytic agent that has been studied in patients with upper GI bleeding. A meta-analysis that included eight randomized trials of tranexamic acid in patients with upper GI bleeding found a benefit with regard to mortality but when only studies that used antiulcer drugs and/or endoscopic therapy were included, there was no beneficial effect. This suggests that there is no role for tranexamic acid in the treatment of upper GI bleeding, since the current standard of care is to treat patients with proton pump inhibitors and endoscopic therapy (if indicated).
Anticoagulants and antiplatelet agents — When possible, anticoagulants and antiplatelet agents should be held in patients with upper GI bleeding. When to resume these medications once hemostasis has been achieved will also depend on the patient's risks for thrombosis and recurrent bleeding.

Consultations —

Gastroenterological consultation should be obtained in all patients with suspected clinically significant acute upper GI bleeding. The decision to obtain surgical and interventional radiology consultations prior to endoscopy should be based upon the likelihood of persistent or recurrent bleeding, or risks/complications stemming from endoscopic therapy (perforation, precipitation of massive bleeding).

REFERENCES

1. Barkun A, Bardou M, Marshall JK, Nonvariceal Upper GI Bleeding Consensus Conference Group. Consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding. *Ann Intern Med* 2003; 139:843.
2. Barkun AN, Bardou M, Kuipers EJ, et al. International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding.

Ann Intern Med 2010; 152:101.

3. Hwang JH, Fisher DA, Ben-Menachem T, et al. The role of endoscopy in the management of acute non-variceal upper GI bleeding. *Gastrointest Endosc* 2012; 75:1132.
4. Laine L, Jensen DM. Management of patients with ulcer bleeding. *Am J Gastroenterol* 2012; 107:345.
5. Gralnek IM, Dumonceau JM, Kuipers EJ, et al. Diagnosis and management of nonvariceal upper gastrointestinal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. *Endoscopy* 2015; 47:a1.
6. Srygley FD, Gerardo CJ, Tran T, Fisher DA. Does this patient have a severe upper gastrointestinal bleed? *JAMA* 2012; 307:1072.
7. Cappell MS, Friedel D. Initial management of acute upper gastrointestinal bleeding: from initial evaluation up to gastrointestinal endoscopy. *Med Clin North Am* 2008; 92:491.
8. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. *Gastroenterology* 1988; 95:1569.
9. Krige JE, Kotze UK, Distiller G, et al. Predictive factors for rebleeding and death in alcoholic cirrhotic patients with acute variceal bleeding: a multivariate analysis. *World J Surg* 2009; 33:2127.
10. Blair SD, Janvrin SB, McCollum CN, Greenhalgh RM. Effect of early blood transfusion on gastrointestinal haemorrhage. *Br J Surg* 1986; 73:783.
11. Lee JM, Chun HJ, Lee JS. Target level hemoglobin correction in patients with acute non-variceal upper gastrointestinal bleeding. *Gastroenterology* 2014; 146:S321.
12. Odutayo A, Desborough MJ, Trivella M, et al. Restrictive versus liberal blood transfusion for gastrointestinal bleeding: a systematic review and meta-analysis of randomised controlled trials. *Lancet Gastroenterol Hepatol* 2017; 2:354.
13. Zakko L, Rustagi T, Douglas M, Laine L. No Benefit From Platelet Transfusion for Gastrointestinal Bleeding in Patients Taking Antiplatelet Agents. *Clin Gastroenterol Hepatol* 2017; 15:46.
14. Dorward S, Sreedharan A, Leontiadis GI, et al. Proton pump inhibitor treatment initiated prior to endoscopic diagnosis in upper gastrointestinal bleeding. *Cochrane Database Syst Rev* 2006; :CD005415.
15. Lau JY, Sung JJ, Lee KK, et al. Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. *N Engl J Med* 2000; 343:310.
16. Chan WH, Khin LW, Chung YF, et al. Randomized controlled trial of standard versus high-dose intravenous omeprazole after endoscopic therapy in high-risk patients with acute peptic ulcer bleeding. *Br J Surg* 2011; 98:640.
17. Bennett C, Klingenberg SL, Langholz E, Gluud LL. Tranexamic acid for upper gastrointestinal bleeding. *Cochrane Database Syst Rev* 2014; :CD006640.
18. Balderas V, Bhole R, Lara LF, et al. The hematocrit level in upper gastrointestinal hemorrhage: safety of endoscopy and outcomes. *Am J Med* 2011; 124:970.
19. Longstreth GF, Feitelberg SP. Successful outpatient management of acute upper gastrointestinal hemorrhage: use of practice guidelines in a large patient series. *Gastrointest Endosc* 1998; 47:219.

POLY-TRAUMA

Dr. Gurinder Singh Mann

M.S. (Orthopaedics)

Apex Hospital Rampura Phul

POLYTRAUMA means a syndrome of multiple injuries with systemic traumatic reactions which may lead to dysfunction or failure of remote organs and vital systems. In INDIA - > 146,377 Deaths last year 55000 crore (550 billion Dollars). The main principle behind the trauma management is an organized team approach. With increase in urbanization and industrialization more and injuries of varied type are increasing day by day. Industrialization and increase vehicles giving rise to more trauma rather poly-trauma patients, who require not only urgent treatment, but also different types of attitude, approach, dedication, planning, preparedness and the well coordinated as well as timely team - work to have an effective outcome of a "Golden hour"[1].

The first hours after trauma are decisive. Therefore the treatment chosen demands very strict planning according to concepts of modern quality management. This begins with the fastest possible and most efficient delivery of injured patients to the applicable clinic. Such institutions are permanently

ready and have at their service all the necessary diagnostic techniques and surgical and intensive care methods. Effective shock treatment entails standardized procedures accompanied by up-to-date diagnostic and therapeutic measures. After admittance and therapy of life-threatening injuries (immediate measures, damage control surgery), early-stage surgery will follow (soft tissue injuries and fractures). Strategy of damage control orthopedics is growing in acceptance because of the potential danger to life functions due to pro- and anti-inflammatory response induced additional trauma caused by following surgery. Fractures initially stabilized by external fixation can consecutively be treated safely by secondary conversion osteosynthesis. A considerable improvement in quality can be attained through therapeutic procedures approved by all concomitant disciplines and standardized systems with internal and external control methods.

TRIPLICATE URETER WITH CONTRALATERAL DUPLICATION ALONG WITH MULLERIAN DUCT ABNORMALITIES IN A CONGENITALLY INCONTINENT GIRL

Dr. Saurabh Gupta
Dr. Gurpreet Singh Gill

Adesh Institute of Medical Sciences and Research, Bathinda

Introduction and Objective

Triplicate ureter inserting ectopically on vagina with contralateral duplication is a rare mesonephric duct abnormality. Occasionally it may be complicated with uterine didelphys which itself is piecemeal of mullerian duct abnormalities (8% of all mullerian anomalies).

Methods

A 12 year old girl with normal voiding plus continuous urinary incontinence was evaluated with CT urography and found to have right triplicate ureter (Smith type III) inserting ectopically in right vagina with contralateral duplication and uterine didelphys. On cystoscopy and vaginoscopy she was found to have two vaginas with ectopic insertion of triplicated right moiety in right vagina. She underwent modified Lich Gregoir non refluxing right moiety reimplant with JJ stent.

Results

Complete resolution of symptoms occurred after reimplant of right triplicate moiety. Patient became dry immediately after the surgery.

Conclusion

Triplication of ipsilateral ureter with ectopic insertion on right vagina in uterine didelphys leading to urinary incontinence with contralateral duplication is a rare diagnosis. It presents a blend of both mesonephric and paramesonephric duct abnormalities and requires thorough evaluation.



CHALLENGES IN CARDIO-THORACIC SURGERY

Dr. Parshant Sevtia

M.Ch. (Cardio-Thoracic Surgery)

Adesh Institute of Medical Sciences and Research (AIMSR)

Bathinda, Punjab, 151001

45 year old male presented with rapidly worsening heart failure. ECHO suggested huge left ventricular aneurysm. EF was less than 20%. Severe mitral regurgitation. Plethoric lung field. An attempt was made to optimize with the help of inotropes, IABP and IV antibiotics.

Intraoperative finding- dense pericardial adhesions, contained rupture of left ventricle, thick layer of soft clot lining the whole pericardial cavity, whole lateral wall of left ventricle thinned out to about 1 mm thickness, Surgery- after removal of all the soft clots, excision of all aneurismal part of ventricle was done. Rent was from apex of heart to the level of mitral annulus. Valve apparatus repaired, ventricle reconstructed with modified DOR's technique.

Patient has started living his normal routine social life.

Another 50 year old male was operated for AVR through 5 cm incision in second ICS, 55 year operated for MVR through 5cm submammary incision. No intraoperative blood transfusion required. Patient started roaming around on first postoperative day, discharged on third POD and started normal routine life in about 15-20 days. Almost all single valves are being operated through MICS. All of our patients were discharged on fourth postoperative day. Patient acceptance for this technique is excellent because comparatively its less painful, wound complications are very less and off course cosmetically very much satisfying.