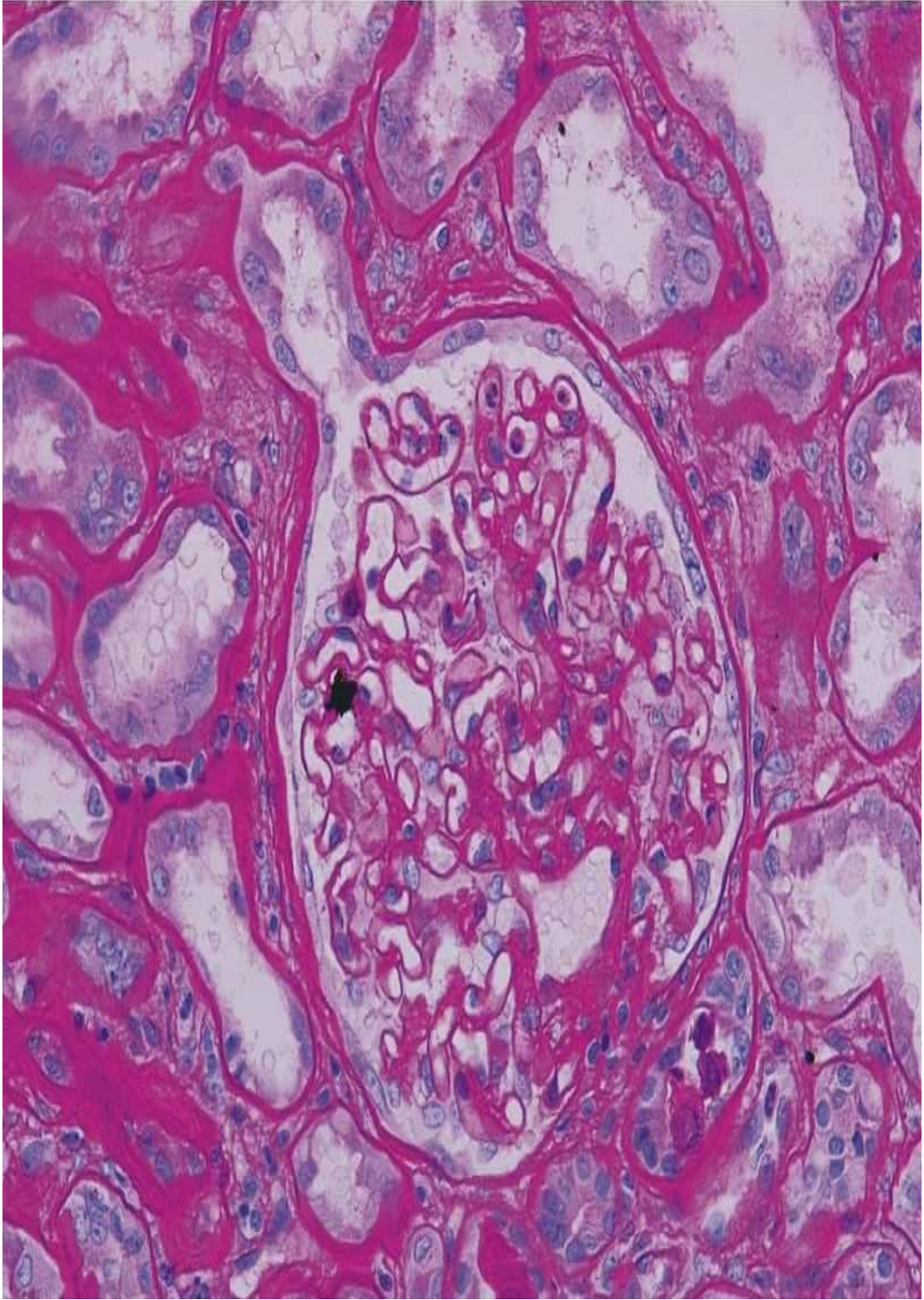


GUIDELINES FOR THE MANAGEMENT OF GASTROENTEROLOGICAL DISEASES

**Ministry of Health & Family Welfare
Govt. of India**

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APPROACH TO DYSPHAGIA

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I. When to suspect/recognize?

a. Introduction:

Dysphagia is an important symptom of esophageal or oropharyngeal disorder. It usually indicates the presence of an underlying disease process and, therefore, requires prompt evaluation.

b. Case definition:

Dysphagia is defined as difficulty or inability to transfer food from the oral cavity to the stomach.

II. Incidence of Dysphagia^{1,2}

Dysphagia has been shown to be present in 20% of patients seen at primary care level and 15% of elderly patients in the community. Many of the elderly patients do not seek medical advice. Incidence data from India are lacking.

III. Differential Diagnosis^{3,4}

Dysphagia is sub classified into two types depending on the location of the lesion. Inability or difficulty in transferring food from oral cavity to upper esophagus is referred to as **oropharyngeal dysphagia**. Inability or difficulty in transferring food from upper esophageal region to stomach is termed as **esophageal dysphagia**.

A careful history is vital in identifying the type and underlying cause of dysphagia. Difficulty in initiating a swallow suggests oropharyngeal dysphagia. This may be accompanied by sensation of food getting stuck above suprasternal notch, choking sensation, nasal regurgitation of food, aspiration, dysarthria and dysphonia. When swallow is initiated but a few seconds later the patient feels food is getting stuck in esophagus (below suprasternal notch), esophageal dysphagia is likely. Dysphagia may occur due to structural lesions in the pathway of food bolus transit (**Mechanical dysphagia**) or neuromuscular dysfunction (**Motor dysphagia**). Difficulty in swallowing liquids and solids from the onset of dysphagia suggests motor dysphagia while difficulty in swallowing solids alone at the onset of illness indicates mechanical dysphagia. A short duration of progressive symptoms with significant weight loss is suggestive of malignancy. Presence of pain during swallowing (odynophagia) may occur in infective lesions (Candida, CMV, etc.) or acute ulcerating lesions (pill esophagitis). History of gastroesophageal reflux symptoms, systemic illness (Stroke,

Parkinson’s disease, myasthenia gravis, muscular dystrophy, scleroderma, AIDS, etc.), drug/corrosive ingestion, exposure to radiation and surgeries in past may provide further clues to the diagnosis. Clinical examination for thyromegaly, cervical lymph nodes, oral cavity lesions and central nervous system function may be helpful. Hence, a careful history usually enables a physician to narrow down the list of differentials. Table 1 shows the differential diagnosis of dysphagia.

Table 1: Differential diagnosis in a patient with dysphagia

Oropharyngeal	Esophageal
<p>Mechanical</p> <ul style="list-style-type: none"> • Oropharyngeal malignancy • Upper esophageal web • Zenker’s diverticulum • Cervical osteophytes • Thyromegaly • Retropharyngeal abscess • Oropharyngeal infection 	<p>Mechanical</p> <ul style="list-style-type: none"> • Esophageal tumours • Corrosive stricture • Peptic stricture • Post radiation stricture • Anastomotic stricture • Food bolus impaction • Foreign body impaction • Esophageal webs and rings • Diverticula • Mediastinal mass lesion • Vascular compression
<p>Motor</p> <ul style="list-style-type: none"> • Cerebrovascular accident • Myasthenia Gravis • Parkinson’s disease • Intracranial Tumour • Polymyositis or Dermatomyositis • Muscular Dystrophy • Rabies • Tetanus • Cricopharyngeal achalasia 	<p>Motor</p> <ul style="list-style-type: none"> • Achalasia Cardia • Nutcracker esophagus • Diffuse esophageal spasm • Hypertensive lower esophageal sphincter • Scleroderma

IV. Prevention and Counselling

As dysphagia is a symptom resulting from various disease entities rather than a single disease, preventive measures are feasible in only certain situations. Diseases resulting from specific inciting agents may be prevented if awareness is improved among the general population.

1. Esophageal cancers have been associated with alcohol consumption, obesity and smoking and counselling about avoiding these risk factors might be useful.

2. Appropriate labelling of corrosive substances and keeping them away from the reach of children may prevent corrosive injuries of esophagus.
3. Swallowing pills in upright position with plenty of fluid may prevent pill esophagitis.
4. Prompt therapy of gastroesophageal reflux disease with proton pump inhibitors may heal esophageal ulcers and prevent development of peptic stricture.
5. Adequate chewing of food reduces the chance of food bolus impaction.
6. Counselling patients that dysphagia is an alarm symptom which requires prompt medical attention may help in early diagnosis and management of malignancy of esophagus.

V. Optimal diagnostic criteria, Investigations, Treatment and Referral Criteria⁵⁻⁸

Situation 1: At Secondary Hospital/Non-Metro situation

a. Clinical diagnosis: Dysphagia is a symptom and hence a history of difficulty/inability to initiate or complete a swallow is sufficient to confirm its presence. Further probing about the duration, nature (solids alone or for both solids and liquids), course (progressive or intermittent) of the symptom as well as associated symptoms (see section on differential diagnosis) helps to reduce the number of possible etiologies and plan appropriate focussed investigations.

b. Investigations:

- Prior to investigations for oropharyngeal/esophageal lesions, systemic illnesses causing dysphagia (stroke, Parkinson's disease, Myasthenia gravis, etc.) should be considered and appropriately evaluated.
- For structural oropharyngeal dysphagia, a nasopharyngeal endoscopy is appropriate. Specimen from lesions should be obtained for histopathology and/or microbiological evaluation. For motor oropharyngeal dysphagia, video-fluoroscopic swallowing study is the best modality.
- In patients with structural esophageal dysphagia, upper gastrointestinal endoscopy is appropriate as it enables better characterisation of lesion and collection of specimen for histopathology and/or microbiological evaluation. In suspected esophageal motility disorder, barium swallow study may be the appropriate initial test. For further characterisation of the motility disorder, patient may be referred to a higher center for esophageal manometry.

c. Treatment:

Oropharyngeal dysphagia:

1. Treatment of neuromuscular causes is difficult but in conditions like myasthenia gravis and Parkinson's disease medical therapy may be useful.
2. Adequate nutrition is crucial. Thick fluids or soft solids are better tolerated.
3. If risk of aspiration is high, feeding through nasogastric tube may be considered or surgical gastrostomy/jejunostomy may be performed for feeding.
4. For infective lesions, antibiotics may be used.
5. Malignant lesions require a multidisciplinary approach at a higher center.

Esophageal dysphagia:

1. Both structural and motor lesions require therapeutic endoscopic procedures or surgery and, hence, are better managed at a higher center.
2. Calcium channel blockers provide some relief in achalasia cardia or diffuse esophageal spasm.
3. Proton pump inhibitors may be given for peptic strictures.
4. Soft foods should be recommended in case of esophageal webs and rings.

Standard Operating Procedure

Out Patient: Patients without systemic illness and with adequate hydration and good sensorium can be managed as outpatients.

In Patient: Patients with severe systemic illness, dehydration and impaired level of consciousness should be managed as inpatients.

Day Care: Patients with dehydration can be managed on a day care basis and discharged after proper hydration and intervention if they can subsequently adequately nourish themselves at home.

Referral criteria:

1. **Oropharyngeal dysphagia:** Non-availability of video fluoroscopy or gastrointestinal endoscopy facilities or for management of malignant lesions
2. **Esophageal dysphagia:** For further evaluation and management after barium swallow study

3. Patients with systemic illnesses requiring specialised care

Situation 2: At super speciality facility in Metro Location where higher-end technology is available

a. Clinical diagnosis: Same as in situation 1.

b. Investigations:

1. Prior to investigations for oropharyngeal/esophageal lesions, systemic illnesses causing dysphagia (stroke, Parkinson's disease, Myasthenia gravis, etc.) should be considered and appropriately evaluated. Consultation with a neurologist and imaging of brain as well as electrophysiological study of nerve/muscles may be warranted.

2. Structural oropharyngeal dysphagia:

(i) Consultation with an ENT specialist for nasopharyngeal endoscopy is appropriate. Specimen from lesions should be obtained for histopathology and/or microbiological evaluation.

(ii) If no lesion is found, a computed tomography of the lower head and neck region would be required.

3. Motor oropharyngeal dysphagia:

(i) Video-fluoroscopic swallowing study is the best modality.

(ii) Manometric study may be undertaken to assess for upper esophageal sphincter dysfunction and cricopharyngeal achalasia.

4. Structural esophageal dysphagia:

(i) Upper gastrointestinal endoscopy is appropriate as it enables better characterisation of lesion and collection of specimen for histopathology and/or microbiological evaluation.

(ii) If a peptic stricture is suspected, a 24 hour esophageal pH study would confirm the presence of gastroesophageal reflux.

(iii) If the esophageal mucosa is normal during endoscopy, extrinsic compressive lesions should be suspected and computed tomography of mediastinum or endoscopic ultrasonography from esophagus is required to locate these lesions.

5. Esophageal motility disorder:

(i) Barium swallow study may be the appropriate initial test.

(ii) For further characterisation of the motility disorder, esophageal manometry should be performed.

Figure 1 shows the approach to a patient with dysphagia.

c. Treatment:

Oropharyngeal dysphagia:

1. Treatment of neuromuscular causes is difficult but in conditions like myasthenia gravis and Parkinson's disease medical therapy may be useful.
2. Adequate nutrition is crucial. Thick fluids or soft solids are better tolerated.
3. If risk of aspiration is high, feeding through nasogastric tube may be considered or percutaneous endoscopic gastrostomy may be performed. If percutaneous endoscopic gastrostomy is not feasible, surgical gastrostomy/jejunostomy may be performed for feeding.
4. For infective lesions, antibiotics may be used. For malignant lesions a multidisciplinary approach involving the surgeon, radiotherapist and oncologist is required.

Esophageal dysphagia: Treatment depends on the disease.

1. Achalasia Cardia:

- a) For surgically low risk patients, graded pneumatic dilatation should be performed.
- b) If two sessions of pneumatic dilatation does not provide adequate symptom relief, surgical (laparoscopic or open) myotomy of lower esophageal sphincter should be performed. Direct referral of patients for surgery is also an option.
- c) For high risk surgical patients, botulinum toxin injection in the LES region may provide short term symptom relief.

2. Strictures (Corrosive, Radiation, Peptic and Anastomotic):

- a) Endoscopic dilatation (bougie dilators) is the preferred initial treatment if feasible. Repeated dilatations may be required.

b) For long, tight strictures or those with frequent recurrence of symptoms after endoscopic dilatation, surgery may be considered. For short strictures dilatation using CRE (Controlled Radial Expansion) under endoscopic vision is effective. For Anastomotic strictures, dilatation using CRE balloons is beneficial.

c) Anti-reflux therapy with proton pump inhibitors reduces the need for further dilatation in peptic strictures.

3. Esophageal tumours:

a) Benign tumours causing dysphagia should be surgically resected.

b) If lesion is small and does not extend beyond submucosa (assessed using endoscopic ultrasonography), endoscopic mucosal resection/endoscopic submucosal dissection are less invasive options.

c) For operable malignant tumours, chemoradiation followed by surgery is the treatment of choice.

d) Most malignant lesions of esophagus are inoperable at diagnosis and hence palliation is the only option. Palliative options include – i) placement of a nasogastric feeding tube over a guide wire (placed during endoscopy), ii) esophageal stenting using self expandable metal stent (SEMS), iii) radiation therapy and iv) surgical gastrostomy/jejunostomy for feeding.

4. Esophageal webs and rings: Esophageal webs and rings should be managed with endoscopic dilatation.

5. Other conditions:

a) Nitrates or calcium channel blockers can be used for diffuse esophageal spasm.

b) Systemic illness like scleroderma requires appropriate medical therapy.

Standard Operating Procedure

Same as in situation 1.

Referral criteria:

While a super speciality center is expected to have all the diagnostic and therapeutic facilities, patients may be referred if required equipments/expertise are not available.

VI. Who does what and timelines?

a. Doctor:

1. Within one hour of patient's arrival at the hospital

Initial resuscitation if patient is sick, dehydrated, etc.,

History and clinical examination

Need for hospitalisation

After the patient is stabilised

Planning further diagnostic tests

Explaining the condition to patients and relatives and obtaining informed consent for procedures

Definitive treatment of the patient

Referral to other specialists in the same hospital or to other centers

b. Nurse:

1. **On patient's arrival at the hospital** - Measure blood pressure, pulse rate, breathing rate and level of consciousness and inquire about the chief complaint. The doctor should be accordingly informed.

2. Obtain intravenous access, collect blood samples for investigations and carry out treatment orders for patients requiring emergency care.

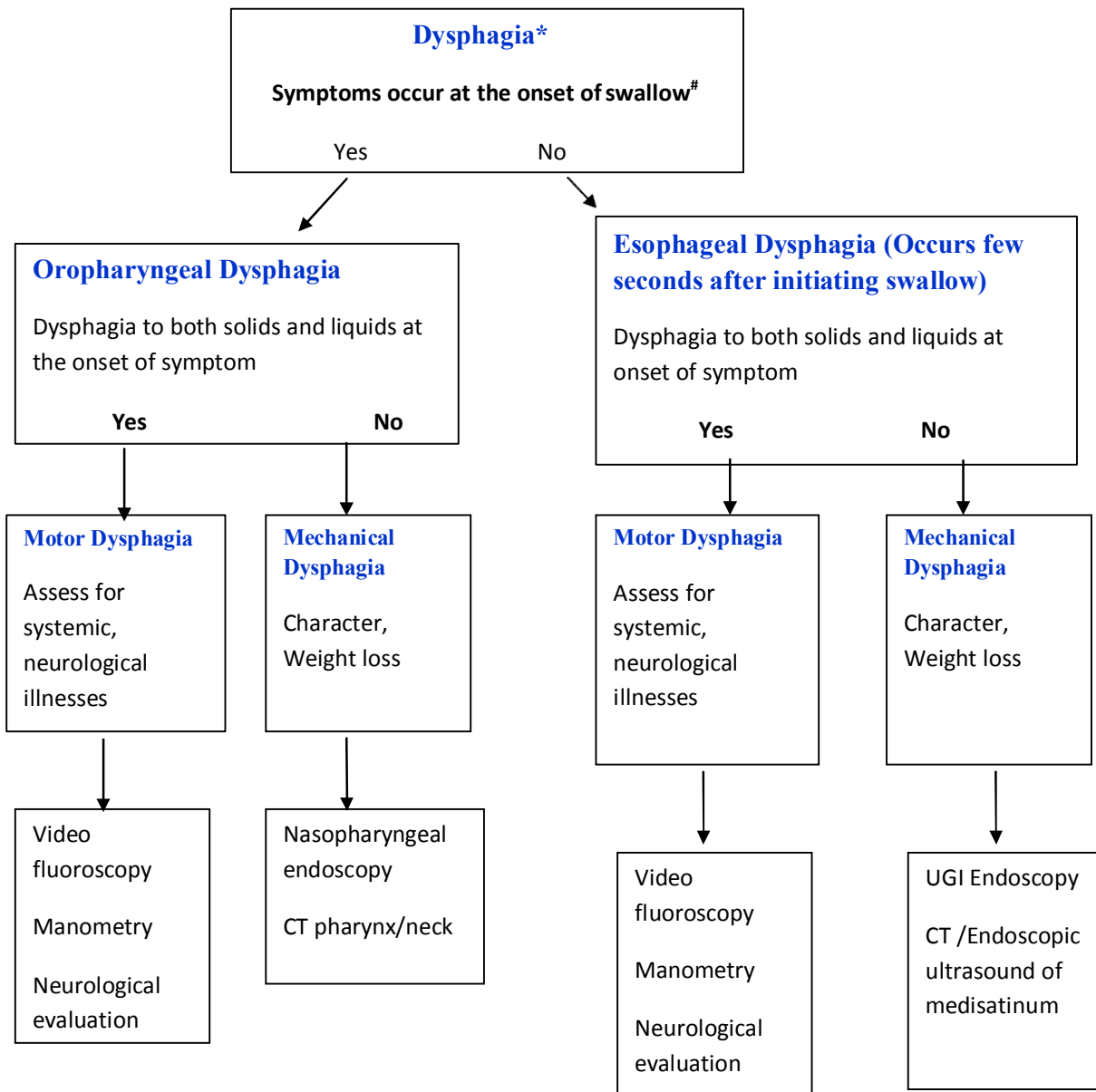
3. Assist the doctor in performing procedures, surgery, etc.

c. Technician

1. Set up and maintenance of equipments required for emergency or elective care

2. Assist the doctor during endoscopic or surgical procedures

3. Keep a ready stock of instruments/accessories required for emergency or elective procedures



*Patients with absolute dysphagia (unable to swallow at all) should be stabilised prior to evaluation of the cause

#Key points in history – character (solid/liquid food), duration, course (progressive/intermittent), weight loss, pain during swallowing, medications/corrosive ingestion, radiation, systemic illness

CT – Computed Tomography; UGI – Upper Gastrointestinal

Figure 1: Approach to a patient with dysphagia

VII. Further reading/References:

1. Kawashima K, Motohashi Y, Fujishima I. Prevalence of dysphagia among community-dwelling elderly individuals as estimated using a questionnaire for dysphagia screening. *Dysphagia*. 2004 Fall;19(4):266-71
2. Thad Wilkins, Ralph A. Gillies, Andria M. Thomas, et al. The Prevalence of Dysphagia in Primary Care Patients: A HamesNet Research Network Study *The Journal of the American Board of Family Medicine* 2007; 20 (2): 144-150
3. Sleisenger and Fordtran's *Gastrointestinal and Liver Disease*, 9th edition, Elsevier.
4. Trate, DM, Parkman, HP, et al. Dysphagia. Evaluation, diagnosis, and treatment. *Prim Care* 1996; 23:417
5. Varadarajulu S, Eloubeidi MA, Patel RS, et al. The yield and the predictors of esophageal pathology when upper endoscopy is used for the initial evaluation of dysphagia. *Gastrointest Endosc* 2005; 61:804
6. Kumar S. Swallowing and dysphagia in neurological disorders. *Rev Neurol Dis*. 2010;7(1):19-27.
7. Garcia JM, Chambers E 4th. *Am J Nurs*. Managing dysphagia through diet modifications. 2010 Nov;110(11):26-33
8. Sreedharan A, Harris K, Crellin A, et al. Interventions for dysphagia in oesophageal cancer *Cochrane Database Syst Rev*. 2009 Oct 7;(4):CD005048.

ACHALASIA CARDIA

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I. When to suspect/recognize?

a. Introduction¹: Esophageal dysphagia involves difficulty in transferring food from the upper esophageal region to the stomach. It is subdivided into mechanical dysphagia (caused by structural lesions in the path of food bolus transit) or motor dysphagia (caused by neuromuscular dysfunction). Mechanical dysphagia is generally progressive in nature with dysphagia to solids being noticed at first followed in time by dysphagia to liquids. On the other hand, in motor dysphagia, there is simultaneous dysphagia to both solids and liquids from the onset. Among the various causes of motor dysphagia, achalasia cardia remains the most recognised. Besides dysphagia, clinical features of achalasia include regurgitation, chest pain, hiccups, halitosis, weight loss, aspiration pneumonia and heartburn. The exact pathophysiology of this entity is unknown although increasing evidence suggests that loss of ganglion cells within the myenteric plexus supplying the smooth muscles of the esophagus (including the lower esophageal sphincter) to be responsible.

b. Case definition¹: Achalasia (a Greek term meaning "failure to relax") is a disease of unknown etiology characterised by loss of peristalsis in the distal esophagus and a failure of lower esophageal sphincter (LES) relaxation.

II. Incidence²⁻⁴: In the West, the incidence of achalasia cardia is reported to be approximately 1/100,000 population per year with prevalence being 7.1 to 13.4/100,000 population per year. There is hardly any data on the incidence and prevalence of the disease in India. A recent retrospective study from Lucknow conducted in a tertiary care centre reported that achalasia cardia constituted 77% of the patients who presented with motor dysphagia. This disease affects people of both genders equally and presents between 25 to 50 years age.

III. Differential Diagnosis¹:

Other motor disorders: Diffuse esophageal spasm

Nutcracker esophagus

Hypertensive LES

Diseases with distinct etiology but with functional consequences mimicking achalasia:

Pseudoachalasia (associated with malignancies and infiltrative diseases)

Chagas disease

Post surgical (following fundoplication and bariatric surgery)

IV. Prevention and Counselling⁵:

There are no particular preventive measures for achalasia cardia. Long standing dilatation of esophagus can result in development of squamous cell carcinoma of the esophagus although there are no recommendations for surveillance.

V. Optimal diagnostic criteria, Investigations, Treatment and Referral Criteria

Situation 1: At Secondary Hospital/Non-Metro situation

a. Clinical diagnosis^{1,6}: Presence of insidious onset, gradually progressing dysphagia to both solids and liquids with varying combinations of regurgitation, chest pain, hiccups, halitosis, weight loss, heart burn and aspiration pneumonia may suggest achalasia cardia. The patient may give history of adopting certain manoeuvres like lifting the head or throwing the shoulders back to facilitate esophageal emptying.

b. Investigations^{1,6}:

- Chest X ray: Mediastinal widening with presence of air fluid level and absence of gastric bubble maybe seen.
- Barium swallow:
 - i) Dilated esophagus with a characteristic “bird beak” deformity in the lower esophagus
 - ii) Massively dilated esophagus may produce a “sigmoid” esophagus
- Upper GI Endoscopy: Typical findings include
 - i) Dilated esophagus with food or fluid residue
 - ii) Esophageal mucosa that is generally normal may show inflammation caused by retained food, pills or Candidiasis
 - iii) Contracted LES that can be negotiated with a gentle push of the endoscope.

Endoscopy is essential to rule out neoplasms in the cardia and the fundus.

c. Treatment^{1,7}: Treatment of achalasia cardia maybe pharmacological, endoscopic or surgical.

- Surgical therapy is generally not performed in secondary hospitals.
- Endoscopic therapy can be attempted only if endoscopes, appropriate accessories, fluoroscopy and preferably, a surgical backup are available.

- Pharmacological therapy includes sublingual nitrates and calcium channel blockers given immediately before meals. These have the advantage of being non-invasive but they are not as effective as the other therapeutic options. Side effects and tachyphylaxis limit their use. Generally these are given to patients who cannot tolerate or are unwilling for invasive therapies.

Standard Operating Procedure

If adequate facilities of investigation and invasive therapy are available, patients in secondary hospitals are generally managed as outpatients. If endoscopic therapy is possible, this can be done as a day care procedure.

Referral criteria:

- Non-availability of endoscopy, endoscopic accessories, manometry, fluoroscopy and surgery
- Patients with systemic illness requiring specialised care

Situation 2: At super speciality facility in Metro Location where higher-end technology is available

a. Clinical diagnosis: Same as in situation 1.

b. Investigations^{1,6-8}: In addition to those discussed in situation 1, other investigations that can be performed include conventional manometry and high resolution manometry of the esophagus.

- Characteristic findings on conventional manometry include failure of the LES to relax, elevated basal LES pressure and aperistalsis of the esophageal body.
- High resolution manometry (HRM) of the esophagus is an advanced form of manometry. Based on HRM, achalasia is classified into 3 subtypes:

Type 1(Classic) achalasia, where swallowing results in no significant change in esophageal pressurization.

Type II achalasia, where swallowing results in simultaneous pressurization that spans the entire length of the esophagus.

Type III (spastic) achalasia, where swallowing results in abnormal, lumen-obliterating contractions (spasms).

Responses to all types of achalasia treatment (both endoscopic and surgical) were best in type II patients and worst in type III patients.

- It is preferable to quantify the basal LES pressure by manometry prior to endotherapy or surgery to assess the efficacy of the same on follow up especially in the event of recurrence of symptoms.

c. Treatment^{1,9,10}: Pharmacological treatment would be same as in situation 1.

The other forms of therapy are broadly divided into endoscopic or surgical.

1) Endoscopic therapy:

- a. Pneumatic dilatation: Forceful stretching of the LES is accomplished using a Rigiflex pneumatic balloon dilator in a graded fashion under fluoroscopy. Although this is quite effective and can be done as a day care procedure, there is a 3-5% risk for perforation which can warrant additional surgery. If two sessions of pneumatic dilatation are unsuccessful, surgery should be performed.
- b. Botulinum injection: Botulinum toxin A is injected into the LES so as to reduce its pressure. Its effect may not be as durable as that following pneumatic dilatation. Minor side effects of this procedure include rash and transient chest pain. It is ideal for high risk surgical patients in providing short term relief.

2) Surgery:

- a. Open Heller's myotomy (either transabdominal or transthoracic)
- b. Minimally invasive (either laparoscopic or thoracoscopic)

Surgical therapy is more effective and long lasting than endotherapy. Reflux esophagitis can be a problem after surgery but can be ameliorated by performing an antireflux procedure (fundoplication). Overall mortality from Heller's myotomy is less than 2%. Current evidence suggests that a laparoscopic approach is associated with similar efficacy, reduced morbidity, and shorter hospital stay when compared with surgical myotomy via other approaches. Direct referral for laparoscopic surgery can be considered for a low risk patient rather than pneumatic dilatation.

Standard Operating Procedure

Out Patient: Patients who do not have major comorbidities can be managed as outpatients. Post procedure patients can also be followed up.

In Patient: This would be required for those with major comorbidities that need stabilisation, for postoperative care and for monitoring those with anticipated complications following endotherapy. This may also be required for those who have copious food residue on initial endoscopy for clearing the contents (using a Ryle's tube) before the next endoscopy.

Day Care: This would be required for patients who are dehydrated for stabilisation following which they can nourish themselves at home. Endotherapy can also be performed as a day care procedure.

Referral criteria:

A superspecialty center is expected to have the required diagnostic and therapeutic facilities for management of achalasia. However, a patient may be referred if required equipment or expertise is unavailable.

VI. Who does what and timelines?**a. Doctor:****If patient is sick (within 1 hour of arrival to hospital)**

Initial resuscitation and assess if patient is dehydrated, has pneumonia, etc and requires admission.

If patient is otherwise well

History and clinical examination

Planning diagnostic tests

Discussion of condition and management options with patients and responsible well wishers and relatives and obtaining informed consent

Definitive treatment

Referral to other specialists in the same hospital or to a higher center

b. Nurse:

Measure vital signs and assess level of consciousness

Inquire chief complaints and inform the doctor accordingly

Obtain intravenous access for blood investigations

Carry out treatment orders as directed by the doctor

Assist the doctor in performing therapeutic procedures

c. Technician:

Maintain equipment required for emergency or elective care

Assist the doctor and the nurse during therapeutic procedures

VII. Further reading/References:

1. Sleisenger and Fortran's Gastroenterological and Liver Disease. 9th edition (Saunders Elsevier, 2010)
2. Howard, PJ, Maher, L, Pryde, A, et al. Five year prospective study of the incidence, clinical features, and diagnosis of achalasia in Edinburgh. *Gut* 1992; 33:1011.
3. Mayberry JF: Epidemiology and demographics of achalasia. *Gastrointest Endosc Clin North Am* 2001; 11:235-48.
4. Misra A, Chourasia D, Ghoshal UC. Manometric and symptomatic spectrum of motor dysphagia in a tertiary referral center in northern India. *Indian J Gastroenterol.* 2010 Jan;29(1):12-6
5. Sandler RS, Nyren O, Ekblom A, et al: The risk of esophageal cancer in patients with achalasia. A population-based study. *JAMA* 1995; 274:1359-62
6. Spechler SJ. Clinical manifestations and diagnosis of achalasia. Uptodate. [Internet] 2010 [cited 2011 April 19]. Available from: <http://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-achalasia>
7. Hirano, I, Tatum, RP, Shi, G, et al. Manometric heterogeneity in patients with idiopathic achalasia. *Gastroenterology* 2001; 120:789.
8. Pandolfino, JE, Kwiatek, MA, Nealis, T, et al. Achalasia: a new clinically relevant classification by high-resolution manometry. *Gastroenterology* 2008; 135:1526.
9. Spechler SJ. Overview of the treatment of achalasia. Uptodate. [Internet] 2010 [cited 2011 April 19]. Available from: <http://www.uptodate.com/contents/overview-of-the-treatment-of-achalasia>
10. Rosati R, Fumagalli U, Bona S, et al: Evaluating results of laparoscopic surgery for esophageal achalasia. *Surg Endosc* 1998; 12:270-3.

ACUTE LIVER FAILURE

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1. WHEN TO SUSPECT /RECOGNIZE?

a. Introduction:

Acute liver failure is a syndrome of varying aetiology resulting in rapid loss of hepatic metabolic and immunological functions, manifesting as altered mentation and coagulopathy and in many cases progressive multi-organ failure, in a previously normal individual. The condition is associated with high mortality and is a frequent indication for liver transplantation.

b. Case definition:

Various terminologies and definitions have been used.

Fulminant Hepatic Failure: Potentially reversible disorder that is the result of severe liver injury with onset of encephalopathy within eight weeks of symptoms and in the absence of pre-existing liver disease.

Acute Liver Failure: Evidence of coagulopathy (INR>1.5) and any degree of mental alteration (encephalopathy) occurring within 26 weeks of onset of illness in a patient without preexisting liver disease.

Note: Diagnosis of hepatic encephalopathy is clinical; West Haven grading of encephalopathy is followed:-

Grade I: Changes in behaviour with minimal change in level of consciousness

Grade II: Disorientation, drowsiness, asterixis, inappropriate behaviour

Grade III: Marked confusion, incoherent speech, sleeping but arousable

Grade IV: Comatose, unresponsive, decorticate or decerebrate posturing

2. INCIDENCE OF THE CONDITION IN OUR COUNTRY

No systemic studies till date to define the incidence of acute liver failure in our country.

3. DIFFERENTIAL DIAGNOSIS of causes of ALF:

Country	HAV	HBV	HEV	PCM	Non-PCM	Unknown
India	2%	15%	44%	0%	1%	31%
USA	4%	7%	-	39%	13%	18%
UK	2%	5%	1%	57%	11%	17%

Legend to above table: HAV= Hepatitis A virus ; HBV = Hepatitis B virus; HEV = Hepatitis E virus; PCM= Paracetamol

4. PREVENTION AND COUNSELING

The prevention of ALF due to viral hepatitis entails prevention of hepatitis and avoiding use of hepato-toxic drugs after onset of hepatitis, including paracetamol. Immunization against hepatitis B is recommended for all. In India over 95% adults are exposed to HAV. Vaccine for HAV is available and may be used in children. Use of potable water is recommended for prevention of HAV and HEV.

5. OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS, TREATMENT & REFERRAL CRITERIA

a. Situation 1: At secondary hospital / non-metro situation

i. Clinical Diagnosis: Patients presenting with acute hepatitis should be monitored for altered sensorium or asterixis. Liver span (normal 12-15 cm) should be examined daily. Spleen tip may be palpable in 20% patients with acute hepatitis due to reticulo-endothelial cell hyperplasia. Evidence of chronic liver disease in the form of spider naevi, gynecomastia, palmar erythema, splenomegaly or ascites goes against the diagnosis of ALF.

ii. Investigations: For severity and aetiology of liver failure and to exclude acute on chronic liver failure and chronic liver disease.

CBC, LFT, INR, Creatinine, Electrolytes, Sugar, USG upper abdomen, HbsAg.

iii. General Measures:

1. Patients with altered mentation should be admitted to ICU.
2. Avoid stimulation, avoid sedation.
3. Nurse with head end elevation to 30°
4. Consider intubation if grade III/IV encephalopathy.
5. Fluid and electrolyte maintenance. Any fluid may be used.
6. Enteral nutrition preferred till grade 1/2 encephalopathy. Protein intake 1G/Kg.
7. Inj PPI (proton pump inhibitor) IV once daily to prevent stress induced erosive gastritis / ulcers.

iv. Specific Measures

1. Inj Vitamin K 10 mg IV single dose.
2. Inj Mannitol bolus 0.5-1g/Kg if signs of cerebral edema: systolic hypertension, bradycardia, irregular respiration or unequal pupils or posturing. Use mannitol only if plasma Osmolality is < 320 mosmol/L.
3. Antibiotics. Surveillance blood culture followed by prophylactic antimicrobials. Suggested protocol : Piperacillin + Tazobactam, Teicoplanin, Metronidazole and Fluconazole. Modify as per prevalence of local flora / sensitivity pattern.
4. Inj N-acetyl cysteine 150mg/Kg over 1 hour, 12.5mg/Kg/hour over next 4 hours and then 6.25mg/Kg/hour over 67 hours. Start early in

course of illness i.e., for patients with grade 1 or 2 hepatic encephalopathy.

5. Treatment directed at aetiology:
 - a. Acetaminophen: N-Acetyl Cystine
 - b. HSV: Acyclovir: Skin lesions in 50% only
 - c. Amanita phalloides: Penicillin G 1 million units/kg/day
 - d. HELLP/AFLP: Terminate pregnancy
 - e. HBV: Tenofovir. Concerns of lactic acidosis with Entecavir

v. Avoid

1. Fresh frozen plasma (FFP) as it interferes with assessment for liver transplantation. Use only if invasive procedure planned.
2. Platelet transfusion with platelets >10,000/cmm unless invasive procedure planned
3. Protein restriction to <1G/Kg
4. Branched chain amino acids
5. Lactulose
6. Inj L-Ornithine L-Aspartate
7. Prophylactic anticonvulsants
8. Hypothermia

vi. Referral Criteria

1. Diagnosis not clear: ALF Vs ACLF / CLD
2. Aetiology not clear
3. Worsening clinical condition
4. Transfer to tertiary care facility may be considered early in the course of illness (Grade 2 encephalopathy).

b. Situation 2: At super-speciality facility in metro location

i. Clinical Diagnosis: same as level 1

ii. Investigations: Same as level 1. In addition:-

IgM-anti HBc if HbsAg positive, IgM-antiHEV. If viral markers negative then ANA, SMA, serum ceruloplasmin, examination for KF rings. In ICU setting: ABG, arterial ammonia.

iii. Treatment: Same as at level 1. In addition:-

3% saline infusion @ 30 ml/hour till serum Na 145 mEq/L, then maintain 10-15 ml/hour (aim serum Na 145-155 mEq/L). Monitor serum sodium 12 hourly.

iv. Referral criteria for liver transplantation

The approach to liver transplantation for ALF has to be clear and mathematical. The following questions to be answered:-

Step 1. Is liver transplantation indicated?

Apply modified King's College Hospital (KCH) criteria:-

Prothrombin time >100s or INR >6.5
Or, any 3 of the following:-
Age < 10 years or > 40 years
Jaundice-Hepatic Encephalopathy interval > 7 days
S bilirubin > 17.5 mg /dl
Prothrombin time >50s or INR > 3.5
NonA-NonE Hepatitis

If answer to step 1 is yes, proceed to step 2. If answer to step 1 is no, keep looking for indication for liver transplantation as the negative predictive value of KCH criteria is low. Give due consideration to MELD score (>30) and arterial ammonia (>124).

Step 2: If answer to step 1 is yes, is there a contraindication to transplantation? Contraindications include sepsis and posturing due to severe cerebral edema. Get in touch with the nearest liver transplant centre for advice before deciding against transplantation.

Step 3: If answer to step 2 is no, can the family afford liver transplantation?

Step 4: If answer to step 3 is yes, is there a suitable donor?
Test blood group of patient and willing near relatives aged 18 to 50 years. If ABO compatible donor is available, counsel on success and risk of liver transplantation and donor surgery.

Step 5: Transfer patient and ABO compatible willing near relative to transplant centre. Consult the nearest liver transplant centre for precautions during the process of transfer.

6. FURTHER READING / REFERENCES

- i. Polson J, Lee WM. AASLD Position paper: The management of Acute Liver Failure. *Hepatology* 2005;41:1179-97.
- ii. Bernal A, Auzinger G, Dhawan A, Wendon J. Acute liver failure. *Lancet* 2010;376:190-201

LIVER CANCER OR HEPATOCELLULAR CARCINOMA (HCC)

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Medanta Medicity

I. WHEN TO SUSPECT/ RECOGNIZE?

a) Introduction:

Liver cancer or primary liver cancer or hepatocellular carcinoma (HCC) is, as the name suggests, a primary malignant tumor arising from the liver cells or hepatocytes. It usually arises on a background of cirrhosis, but it may even arise in non-cirrhotic livers. The etiology of cirrhosis may be hepatitis B, hepatitis C, alcohol or even non-alcoholic steatohepatitis. Liver cancer has been reported as the 5th most common cancer and the third most common cause of cancer related mortality in the world literature.

b) Case definition:

For both situations of care (*mentioned below**)

Secondary care centers: Any space occupying lesion (SOL) or a nodule in the liver detected either on ultrasonography, CT scan or MRI, in a patient who has underlying cirrhosis, should be considered as an HCC unless proved otherwise. The presence of high AFP >400ng/ml confirms the diagnosis of the SOL/nodule being HCC. However, normal AFP levels do not exclude the presence of HCC. Appearance of a new SOL in liver even in patients, who do not have underlying cirrhosis, should be investigated with HCC being the first differential diagnosis.

Tertiary care centers:

1. In a patient who has cirrhosis any SOL, more than 2 cm, if it shows enhancement in the arterial phase and washout in the delayed phase, on at least one dynamic imaging (either a triple phase contrast CT scan of abdomen or dynamic contrast enhanced MRI of the abdomen), should be labeled as HCC unless proved otherwise, irrespective of alpha feto protein (AFP) levels.
2. In a patient who has cirrhosis, and the SOL is between 1-2 cm, then the typical contrast enhancement and washout pattern needs to be demonstrated on at least two imaging modalities to make a diagnosis of HCC
3. Patients who have SOL in the liver but do not have cirrhosis, or those who have atypical enhancement characteristics on dynamic imaging, the diagnosis of HCC can be confirmed by characteristic microscopic features on a biopsy of the lesion or fine needle aspiration cytology of the lesion.

Where ever possible efforts should be made to make a non-invasive diagnosis of HCC, that is, without putting any needle in to the tumor

II. INCIDENCE OF THE CONDITION IN OUR COUNTRY

In our country, the reported incidence of HCC is 1.2 /100,000 females per year and 2.7/100,000 males per year and a prevalence of 1.9% of all cancers, based on population cancer registry data.

In a prospective study in patient with cirrhosis, the reported incidence is 1.6 per 100 person years of follow up.

III. DIFFERENTIAL DIAGNOSIS

Differential diagnosis include all other causes of mass /SOL in the liver. These can be either benign or malignant

Benign liver masses	Malignant liver masses
Hemangioma	Metastases from other malignant tumors (secondaries)
Focal nodular hyperplasia	Cholangiocarcinoma
Hepatocellular adenoma	
Complex liver cysts	

These lesions can be differentiated from HCC by means of characteristic imaging findings of these lesions as well as the clinical setting of presentation.

IV. PREVENTION AND COUNSELING

The most important risk factors for the development of liver cancer are hepatitis B infection, hepatitis C infection, alcoholic cirrhosis, non-alcoholic steatohepatitis related cirrhosis, and other causes of cirrhosis. Therefore the most important preventive measures would be to prevent development of these diseases. The following measures can me considered to be preventive strategies for HCC:

1. Vaccination against hepatitis B
2. Universal screening of donated blood for hepatitis B and C and discarding of infected units
3. Preventive strategies for diabetes and obesity
4. Strategies to prevent alcoholism

At present there is no recommended chemopreventive strategy, once a patient becomes cirrhotic.

V. OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS, TREATMENT & REFERRAL CRITERIA

*Situation 1: At Secondary Hospital/ Non-Metro situation: Optimal Standards of Treatment in Situations where technology and resources are limited

a. Clinical Diagnosis:

All patients with cirrhosis of liver who develop recent worsening of their clinical status should be suspected of having developed liver cancer and should be appropriately investigated. It should be suspected when:

1. A patient who has stable cirrhosis develops new ascites
2. A cirrhotic patient develops a difficult to control ascites.
3. A stable cirrhotic develops severe constitutional symptoms, such as weight loss or anorexia.
4. Any patient, especially one who has cirrhosis, develops prolonged fever of unknown origin (FUO)
5. A patient of cirrhosis develops pain in the upper abdomen.

b. Investigations:

The initial investigations should include:

1. Abdominal ultrasonography which can pick up features of cirrhosis and presence of any SOL in the liver.
2. Alpha feto protein (AFP) levels: Levels of more than 10 ng /ml should raise the suspicion of HCC and should be investigated further.

c. Treatment:

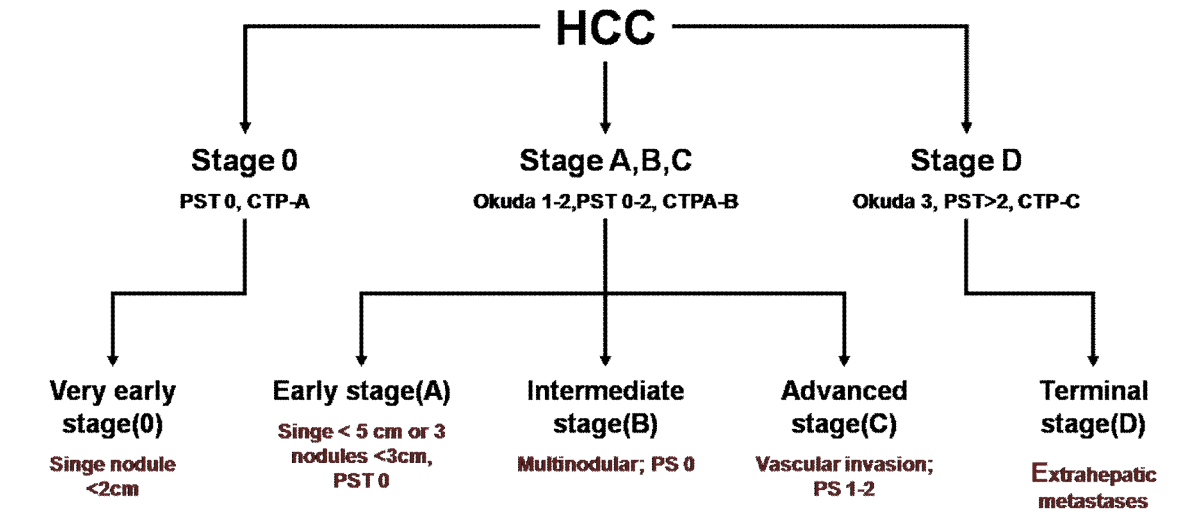
Standard Operating procedure

Comprehensive treatment of Liver cancer involves:

1. Treatment of the tumor
2. Treatment of cirrhosis
3. Treatment of complications of cirrhosis
4. Treatment of the virus if the cause is hepatitis B or C.

Treatment of the tumor is based on the stage of the tumor. The most commonly used staging and treatment plan followed is based on the BCLC (Barcelona clinic liver cancer) staging system. This system incorporates, not only the size and number of tumors, but also,

the residual liver function, the functional status of the liver and presence or absence of extrahepatic spread. The BCLC staging system is given below:



Scheme of BCLC staging for liver cancer (PST: Performance status score; CTP: Child Turcott Pugh class)

d. In Patient

In the secondary care set up, the patient should be managed for complications of cirrhosis, such as GI bleeding, ascites, hepatic encephalopathy.

e. Out Patient

Only in cases requiring symptomatic treatment

Referral criteria:

Patients requiring curative or palliative therapies for HCC [such as liver transplantation, liver resection, radiofrequency ablation, percutaneous ethanol injection, trans-arterial chemoembolization, trans arterial radioembolization, and biological therapy (tyrosine kinase inhibitors or monoclonal antibodies)], should all be referred to a hepatologist or gastroenterologists with interest in managing HCC at a tertiary care center

Even patients requiring advanced treatment for complications of cirrhosis, should be referred to tertiary care centers.

*Situation 2: At Super Specialty Facility in Metro location where higher-end technology is available

a) Clinical Diagnosis:

All patients with cirrhosis of liver who develop recent worsening of their clinical status should be suspected of having developed liver cancer and should be appropriately investigated. It should be suspected when:

1. A patient who has stable cirrhosis develops new ascites
2. A cirrhotic patient develops a difficult to control ascites.
3. A stable cirrhotic develops severe constitutional symptoms, such as weight loss or anorexia.
4. Any patient, especially one who has cirrhosis, develops prolonged fever of unknown origin (FUO)
5. A patient of cirrhosis develops pain in the upper abdomen.

b) Investigations:

The initial investigations should include:

1. Abdominal ultrasonography which can pick up features of cirrhosis and presence of any SOL in the liver.
2. Alpha feto protein (AFP) levels: Levels of more than 10 ng /ml should raise the suspicion of HCC and should be investigated further. The further investigations required are:
 3. Triple phase contrast enhanced CT Scan of abdomen
 4. Dynamic contrast enhanced MRI of the abdomen
 5. Contrast enhanced ultrasonography
 6. PET CT scan of the abdomen
 7. Bone scan
 8. Biopsy or FNAC of the tumor with immunohistochemical staining
 9. Other tumor markers such as PIVKA-II and AFP-L3

The investigations from number 3-9 may all be required or may be required selectively for confirming the diagnosis, determining the stage of the disease, determining extrahepatic spread and suitability for treatment.

c) Treatment:

As has been mentioned earlier, treatment depends on the BCLC stage of the disease

Standard Operating procedure

a. In-Patient

Curative forms of therapy for liver cancer include:

1. Liver resection (surgical removal of a part of the liver involved by the tumor)
2. Percutaneous ablative therapies [burning the tumor with either radiofrequency current (radiofrequency ablation) or with injection of 100% alcohol or with injection of 50% acetic acid] or
3. Liver transplantation.

Palliative therapies include:

1. Transarterial chemoembolization,
2. Transarterial radio-embolization
3. Systemic chemotherapy.
4. Continuous intra-arterial chemotherapy through implantable port.

As mentioned earlier, BCLC staging system guides therapy as follows:

Patients with very early HCC (stage 0) and early HCC (stage A): Can be offered all forms of curative therapy:

1. Liver transplantation
2. Liver resection
3. Percutaneous ablative therapies (radio frequency ablation, percutaneous ethanol injection or percutaneous acetic acid injection therapy)

Patients with intermediate HCC (stage B):

Can be offered therapy with:

1. Transarterial chemoembolization (TACE).
2. Oral chemotherapy with biological such as sorafenib
3. Liver transplantation can be offered to patients with single large tumor or even multiple tumors, but only at experienced centres. (these would be patients outside Milan's criteria [single tumor \leq 5 cm or up to 3 tumors with diameter \leq 3 cm])

Patients with (stage C):

Can be offered

1. Systemic chemotherapy with Sorafenib
2. Radioembolization with yttrium 99 theraspheres

Patients with end-stage disease (stage D):

Can be offered

1. Oral chemotherapy with Sorafenib
2. Symptomatic therapy

b. Out Patient

Patients being given symptomatic therapy or oral chemotherapy can be managed in the out patient setting. Follow up after curative therapies would also be done in the out patient setting.

Following percutaneous ablative therapies, patients need to undergo regular FU with a dynamic contrast imaging (either a CT or MRI scan), liver function profiles and tumor markers at regular intervals or as indicated.

d) Referral criteria:

Patients fit for undergoing liver transplantation should be referred to a hepatologist at a liver transplant center

VI. WHO DOES WHAT? and TIMELINES

a. Doctor

1. Diagnosis and treatment (medical, radiological and surgical) of cases

b. Nurse

1. Nursing care to patients undergoing treatment at in-patients.
2. Endoscopy nurse

c. Technician

1. Endoscopy technician
2. Interventional radiology technician

3. Liver surgery technician
4. Lab technician, biochemistry/hematology lab
5. Blood bank technician

VII. FURTHER READING / REFERENCES

1. J, Sherman M. Management of hepatocellular carcinoma. *Hepatology* 2005; 42: 1208-1236.
2. Paul SB, Sreenivas B, Gulati MS, Madaqn K, Gupta AK, Mukhopadhaya S, Panda SK, Acharya SK. Incidence of hepatocellular carcinoma among Indian patients with cirrhosis of liver. An experience from a tertiary care center in northern India. *Indian J Gastroenterol* 2007; 26: 274-8.
3. Paul SB, Chalamalasetty SB, Vishnubhatla S, Madan K, Gamnagatti SR, batra Y, Gupta SD, Panda SK, Acharya SK. Clinical profile, etiology and therapeutic outcomes in 324 hepatocellular carcinoma patients in a tertiary care center in India. *Oncology* 2009; 77: 162-71.

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE

(PATIENT WEIGHT 60 KGS)

**(Units to be specified for human resources, investigations, drugs and consumables and equipment.
Quantity to also be specified)**

Situation	HUMAN RESOURCES	INVESTIGATIONS	DRUGS & CONSUMABLES	EQUIPMENT
1	Specialist MD or DM doctor, Nurse, Technician	CBC, LFT, RFT, INR, AFP, Blood cultures, urine cultures, ultrasound	IV set, IV cannulas, Syringes, Diuretics (aldactone, furosemide, torsemide), lactulose, pantoprazole oral and iv, somatostatin, terlipressin, tenofovir, entecavir, telbivudine, b complex, calcium, oral iron, UDCA, vitamin E, insulin, oral hypoglycemic drugs	Ryles tube, Sengstaken blackmore tube, Ultrasound machine, ECG machine
2	DM gastroenterologist or hepatologist, Interventional radiologist, GI/liver transplant surgeon, biochemist, hematologist, microbiologist, Resident doctors in all specialities Nurses, technicians	Endoscopy, Multislice CT scanner, MRI, Ultrasound, PET scanner, bone scan.....in addition to what is required in situation 1	All drugs and consumables required in liver surgeries and liver transplant surgeries, interventional radiology set up, sorafenib,in addition to what is required in situation 1	Endoscopy equipments, Multislice CT scanner, MRI scanner, PET scanner, bone scanner, Contrast ultrasound machine and the contrast (sonovue/sonazoid) Wards, ICUs, OTs, Interventional radiology set up and all equipment required in such set ups.....in addition to what is required in situation 1

INFLAMMATORY BOWEL DISEASE

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I. WHEN TO SUSPECT/ RECOGNIZE?

a) Introduction:

Inflammatory bowel disease (IBD) represents a group of idiopathic chronic inflammatory intestinal conditions. The two main disease categories included are Crohn's disease (CD) and ulcerative colitis (UC), with both overlapping and distinct clinical and pathological features. In addition the spectrum also comprises two categories ; IBD unclassified and indeterminate colitis. The pathogenesis of IBD is still under investigation. The most simplified view is that intestinal injury results due to aberrant immune response to commensal bacteria in a background of genetic predisposition.

b) Case definition:

- Ulcerative colitis:
Ulcerative colitis is a chronic disorder of unknown etiology in which a part or the whole of the mucosa of the colon becomes diffusely inflamed and ulcerated. Rectum is involved in a vast majority of the cases.
- Crohn's Disease:
A chronic granulomatous disease which can affect any part of GI tract in a discontinuous, asymmetric manner. Unlike Ulcerative colitis which is a mucosal disease, Crohn's disease is a transmural disease.
- IBD unclassified:
Categorization is not possible after clinical, radiological, endoscopic and histological features
- Indeterminate colitis:
Categorization is not possible even after evaluation of resected specimen.

II. INCIDENCE OF THE CONDITION IN OUR COUNTRY

The epidemiological studies from this region are being made available it is clear that the incidence and prevalence rates of IBD in Asia–Pacific region are low compared with Europe and North America. They are however, increasing rapidly.

There are substantial variations in the incidence and prevalence rates of IBD in various ethnic groups in Asia. The highest incidence rates are recorded from India, Japan and the Middle East and there exists a genetic predisposition of South Asians (Indians, Pakistanis and Bangladeshis) to ulcerative colitis (UC). In a study done in 2001 from Ludhiana in Punjab, the crude prevalence rate was $44.3/10^5$. The incidence was calculated after a second visit to the same area one year later. The crude incidence rate was $6.02/10^5$. The incidence rate of UC in India is higher than in Asian countries like Japan ($1.95/10^5$) and Korea ($1.23/10^5$). There is no population-based study on Crohn's disease (CD) in India, however, the accepted perception is that the incidence of CD is rapidly rising in India. The peak age of incidence of CD is the third decade of life, with a decreasing incidence rate with age. The incidence rate in UC is quite stable between the third and seventh decades.

III. DIFFERENTIAL DIAGNOSIS

The main differential diagnosis of Ulcerative colitis and Crohn's disease are :

Ulcerative colitis	Crohn's disease
Acute self limiting colitis	Intestinal Tuberculosis
Amoebic colitis	Acute self limiting colitis
Crohn's Disease	Amoebic colitis
HIV enteropathy	Ulcerative colitis
	Behcet's disease
	NSAID enteropathy
	HIV enteropathy

IV. PREVENTION AND COUNSELING

IBD is an autoimmune disease, and environmental factors which can trigger an episode have not been identified, hence primary prevention strategy is not possible. There is a family history in 5% of IBD patients.

V. OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS, TREATMENT & REFERRAL CRITERIA

*Situation 1: At Secondary Hospital/ Non-Metro situation: Optimal Standards of Treatment in Situations where technology and resources are limited

There is no gold standard test available to make a diagnosis of IBD. A diagnosis of ulcerative colitis or Crohn's disease is made on the basis of compatible clinical history, examination, imaging, endoscopy and histology findings.

a)

Clinical

Diagnosis:

History

The most important features are the chronic duration of symptoms and the frequent remissions and relapses which characterize IBD and help in distinguishing from other infectious diseases affecting the large and small bowel.

Ulcerative colitis

1. Diarrhea : Large bowel diarrhea which is daytime or nocturnal
2. LGI bleed : Blood may be mixed with stool and at times separate from the stools
3. Rectal symptoms: tenesmus, urgency , frequency of stools
4. Abdominal pain and fever in case of severe disease
5. Symptoms are limited to large bowel

Crohn's disease(CD)

Symptoms depend upon the site of involvement. In CD , any part of bowel from esophagus to anal canal may be involved .

Small Intestinal involvement / Ileocolonic involvement :

1. Abdominal Pain
2. Symptoms suggestive of recurrent partial intestinal obstruction may be present
3. Chronic diarrhea
4. Fever, anorexia, weight loss

Large bowel involvement :

1. Chronic diarrhea
2. Hematochezia
3. Peri anal disease
4. Fever, weight loss
5. Abdominal pain

Upper GI involvement

1. Dysphagia, odynophagia
2. Epigastric pain
3. Symptoms suggestive of gastric outlet obstruction

Extraintestinal manifestations (EIM) :Arthritis is the most common EIM and is observed in 15-20% of cases. Other extraintestinal complications include ankylosing spondylitis, pyodermagangrenosum, erythema nodosum, iritis, uveitis, episcleritis, and primary sclerosing cholangitis.

Complications include :

1. Hemorrhage: profuse bleeding from ulcers in UC. Bleeding is less common in CD. Massive bleeding in CD is more often seen from ileal ulceration than colitis.
2. Strictures, bowel perforation and Intra-abdominal abscesses in CD.
3. Fistulas and perianal disease in CD
4. Colorectal cancer: Significantly increased risk of colon cancer in UC after 8 years of diagnosis; the risk is lower in CD as compared to UC.

b) **Investigations:**

Primary and secondary care settings :

- Stool for ova and cysts
- Haemogram and Serum albumin
- Sigmoidoscopy (if available)
- Barium enema for ulcerative colitis
- In India, at present, a suspected case of Crohn's disease should always be referred to a tertiary care hospital before initiating therapy. This is so as to rule out intestinal tuberculosis which closely mimics Crohn's disease.

Referral Hospitals:

- Stool examination:
 - i) Parasites
 - ii) *Clostridium difficile* (should be considered even in absence of antecedent antibiotics).
- Blood tests :
 - i) Haemoglobin , ESR , serum albumin , Human immunodeficiency virus (HIV)
 - ii) Perinuclear antineutrophil cytoplasmic antibody (p-ANCA) and anti-*Saccharomyces cerevisiae* antibodies (ASCA): need not be done
 - iii) Celiac serology
- Montoux skin test
- Chest X ray
- Sigmoidoscopy / Colonoscopy

An endoscopic examination is necessary a) to establish the diagnosis b) to assess severity of disease c) to take targeted mucosal biopsies . In cases with severe activity , a full length colonoscopy is not indicated as there is a high risk of perforation.

Ulcerative colitis: An ileocolonoscopy is indicated to establish the extent of disease

Crohn's Disease: Endoscopic procedures required include : 1) ileocolonoscopy 2) UGI endoscopy (especially in pediatric cases) 3)Capsule endoscopy/ double balloon enteroscopy if small bowel involvement suspected.

- Histology

Multiple mucosal biopsies should be taken from inflamed areas. Features suggestive of ulcerative colitis include crypt architectural destruction, crypt abscesses, goblet cell depletion, paneth cell metaplasia, basal plasmocytosis. In Crohn's disease non caseating granulomas may be seen in 30% of cases

- Imaging

1. Abdominal X ray: In severe UC or CD where perforation or toxic mega colon is suspected. In CD, if intestinal obstruction is suspected.
2. Contrast enhanced computed tomography (Enteroclysis/Enterography) : In cases with CD i) to evaluate small intestinal involvement ii) to differentiate from intestinal tuberculosis
3. Barium meal follow through: If facilities for CT (enteroclysis) are not present.

- These investigations not only help in diagnosing the disease but also help in determining the extent and severity of the disease . The tables below show the classification for extent and severity of UC and CD.

Table 1: Disease extent in Ulcerative colitis:

Extent	Disease	Description
E1	Ulcerative proctitis	Involvement limited to rectum
E2	Left sided UC (distal colitis)	Involvement distal to the splenic flexure
E3	Extensive UC (Pancolitis)	Involvement extends proximal to the splenic flexure

Table 2: Disease activity in ulcerative colitis (adapted from World Gastroenterology Organisation Global Guidelines June 2009)

	Mild	Moderate	Severe
Bloody stools/day	< 4	4 or more <i>if</i>	≥ 6 <i>and</i>
Pulse	< 90 bpm	≤ 90 bpm	> 90 bpm <i>or</i>
Temperature	< 37.5 °C	≤ 37.8 °C	> 37.8 °C <i>or</i>
Hemoglobin	> 11.5 g/dL	≥ 10.5 g/dL	< 10.5 g/dL <i>or</i>
ESR	< 20 mm/h	≤ 30 mm/h	> 30 mm/h <i>or</i>
<i>or</i> CRP	Normal	≤ 30 mg/L	> 30 mg/L

Table 3 : Disease activity in Crohn’s Disease (ACG classification)

Mild (corresponds to CDAI < 150)	Moderate (corresponds to CDAI 150-220)	Severe(corresponds to CDAI >450)
Ambulatory, eating < 10% weight loss No features of dehydration Obstruction, fever, Abdominal mass or tenderness CRP > upper limit	Intermittent vomiting, abdominal pain, > 10% weight loss No features of dehydration Obstruction, fever, Abdominal mass or tenderness CRP > upper limit	BMI < 18, cachexia, dehydration Evidence of Obstruction or abscess Persistent symptoms despite intensive Rx

Table 4 : Montreal Classification of Crohn's disease phenotype

Age	A1 : < 16 years A2 : 17-40 years A3 : > 40 years
Location	L1 : Ileal L2 : Colonic L3 : Ileocolonic L4 : Isolated upper GI disease*
Behaviour	B1 : Non stricturing non penetrating disease B2 : Stricturing disease B3 : Penetrating disease p : perianal disease modifier #

*L4 is a modifier that can be added to L1–L3 when concomitant upper gastrointestinal disease is present.
"p" is added to B1–B3 when concomitant perianal disease is present

In addition , these investigations also help in

- 1) differentiating Ulcerative colitis from Crohn's disease
- 2) Intestinal tuberculosis from Crohn's disease as shown in the tables below

Table 5 : Features for differentiating between ulcerative colitis (UC) and Crohn’s disease (CD)
 (adapted from World Gastroenterology Organisation Global Guidelines June 2009)

	Typical UC features	Typical CD features
Clinical	<p>Frequent small-volume diarrhea with urgency</p> <p>Predominantly bloody diarrhea</p>	<p>Diarrhea accompanied by abdominal pain and malnutrition</p> <p>Stomatitis</p> <p>Abdominal mass</p> <p>Perianal lesions</p>
Endoscopic and radiological	<p>Diffuse superficial colonic inflammation</p> <p>Involvement of rectum, but this can be patchy</p> <p>Shallow erosions and ulcers</p> <p>Spontaneous bleeding</p>	<p>Discontinuous transmural asymmetric lesions</p> <p>Mainly involving ileum and right-sided colon</p> <p>Cobblestone appearance</p> <p>Longitudinal ulcer</p> <p>Deep fissures</p>
Histopathological	<p>Diffuse inflammation in mucosa or submucosa</p> <p>Crypt architecture distortion</p>	<p>Granulomatous inflammation</p> <p>Fissures or aphthous ulcers can be seen; often transmural inflammation</p>

Table 6 : Distinguishing between tuberculosis and Crohn's disease (adapted from World Gastroenterology Organisation global Guidelines June 2009)

Features	TB	CD
Clinical	<p>History of, or current TB</p> <p>Positive TB contact</p> <p>Less frequent fistulas, abdominal abscesses, or perianal involvement</p> <p>Abnormal CXR (not universal)</p>	<p>Fistulas</p> <p>Bowel wall abscess</p> <p>Anal perirectal disorders</p> <p>Bloody stools</p> <p>Bowel perforation</p> <p>Recurrence after intestinal resection</p>
Endoscopic	<p>Superficial, irregular transverse ulcers without predominant segmental distribution</p> <p>Pseudopolyps</p> <p>Cecum > ileum</p> <p>ICV involved (gaping)</p>	<p>May appear similar to changes in TB</p> <p>TB features less common in intestinal TB (favoring CD):</p> <ul style="list-style-type: none"> — Longitudinal ulceration — Cobblestoning — Aphthous ulceration — Ileum > cecum — ICV may be stenosed or ulcerated
Histopathological	<p>Large, dense, confluent granulomas</p> <p>Submucosal granulomas</p> <p>Caseous necrosis and submucosal stenosis</p> <p>Caseating changes in intestinal wall and mesenteric lymph nodes</p> <p>Positive for acid-fast bacilli</p> <p>Disproportionate submucosal inflammation</p> <p>Bands of epithelioid histiocytes lining ulcers</p>	<p>Noncaseous granulomas/necrosis may be found in up to 50%</p>
Specific tests	<p>TB DNA analysis with TB-specific primer</p> <p>TB bacteria culture</p> <p>Tuberculin PPD skin test</p> <p>Serum PPD antibody test</p> <p>IFN-γ assays</p> <p>Chest radiography for pulmonary TB</p> <p>ASCA and p-ANCA are of no value in differentiating the two conditions</p>	
Cross-sectional	Cecum > ileum	Ileum > cecum

IBD management should be based on:

- ☐ UC vs. CD (although this is less important for early aspects of treatment)
- ☐ Disease location and phenotype
- ☐ Severity

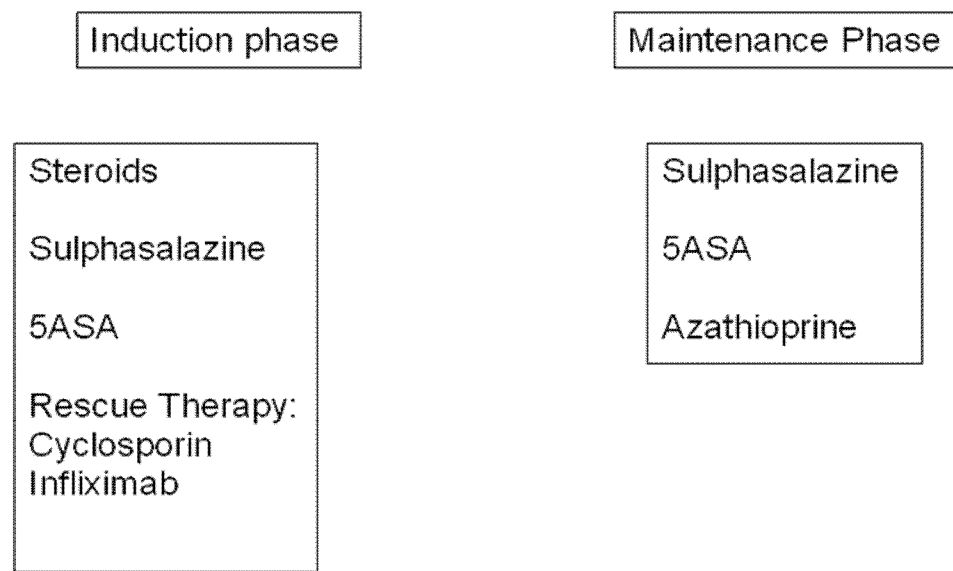
The goals of treatment are to:

- Maintain steroid-free remissions (decreasing the frequency and severity of recurrences and reliance on steroids)
- Prevent complications hospitalization and surgery

Treatment include two phases

- É Induction of remission
- É Maintenance of remission

The figure below shows the drugs which are useful in induction and maintenance phase



Ulcerative colitis :

Disease extent: Proctitis and Proctosigmoiditis

- Inducing Remission

Proctitis : 5ASA suppositories ; Optimal dose : 1gm/day

Proctosigmoiditis : 5ASA enemas : 2-4 gm/

If no response within 4 week

Use of topical glucocorticoids

If still no response :

Use of hydrocortisone rectal drip: 200mg/200ml/1-2 hrs

- Maintaining remission

Oral 5 ASA in a dose of 1600-2400 mg daily may be used with or without topical therapy

If disease extent is left sided colitis/pancolitis, then the treatment may be planned as in the table below:

A. For Induction of remission depending upon the severity of disease

Mild to Moderate:

SZA :4-6 gm/day

5ASA: 4 gm/day

No clinical response:

Prednisolone :

40-60 mg/day

Steroid tapering :

Very gradual

Over3-4 months

Severe :

Oral steroids

No response :

Hospitalization

IV steroids

IntensiveTherapy

Fulminant :

Hospitalization

IV steroids

Intensive Therapy

Surgery

B. For maintenance of remission : Use of 5 ASA alone or 5ASA and azathioprine in patients who require frequent steroids

Management guidelines in Crohn's Disease (as in the table below)

Inflammatory CD (B1 phenotype)	Mild to moderate	Induction regime SSZ : left colon Budesonide : ileocolon	Remission : Azathioprine Methotrexate
	Moderate with systemic	Induction regime	Remission :

	Symptoms Severe Small bowel extensive disease	Prednisolone IV Hydrocortisone	Azathioprine Methotrexate
	Introduction of Infliximab	Corticosteroid dependent Corticosteroid refractory 2 courses of steroids/year Risk factors for Top Down rx group	Remission : IFX + Azathioprine Not Methotrexate
Fistulizing Disease (B3 phenotype)	Perianal : Simple vs complex	Antibiotics, azathioprine Infliximab , Surgery	
	Non perianal	Surgery	
Stricturing Disease (B2 phenotype)		Steroids/balloon dilatation/surgery/? Infliximab	

ii) Referral criteria:

Criteria for surgery referral :

- Toxic megacolon
- Perforation peritonitis
- Severe bleed
- Refractory to medical therapy
- Stricturing Crohn's disease

Standard Operating procedure

- a. **In Patient** : Patients with severe disease not responding to oral steroids
- b. **Out Patient** : Patients with mild to moderate disease or patients in remission on follow up
- c. **Day Care** : Any patient requiring infliximab maintenance infusion

VI. WHO DOES WHAT? and TIMELINES

- a. **Doctor:** Diagnose and strategise therapy
- b. **Nurse :** Assist in administering drugs
- c. **Technician :** Assist in endoscopy and imaging investigations

VII. FURTHER READING / REFERENCES

1. Inflammatory bowel disease: a global perspective. World Gastroenterology Organisation Global Guidelines. June 2009
2. Hanauer SB. Medical management of Crohn's disease: treatment algorithms 2009. Dig Dis. 2009;27(4):536-41.
3. Van Assche G, Vermeire S, Rutgeerts P. Management of acute severe ulcerative colitis. Gut. 2011 Jan;60(1):130-3.
4. Mowat C, Cole A, Windsor A, et al ; on behalf of the IBD
5. Section of the British Society of Gastroenterology. Guidelines for the management of inflammatory bowel disease in adults. Gut. 2011 May;60(5):571-607.

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE

(PATIENT WEIGHT 60 KGS)

(Units to be specified for human resources, investigations, drugs and consumables and equipment. Quantity to also be specified)

Situation	HUMAN RESOURCES	INVESTIGATIONS	DRUGS & CONSUMABLES	EQUIPMENT
1				
2				

LOWER GASTROINTESTINAL BLEED

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I. WHEN TO SUSPECT/ RECOGNIZE?

c) Introduction:

Lower GI bleeding is a common GI problem. It may present as acute lower GI bleed or may present as occult GI bleed leading to chronic GI blood loss. It may also present as a challenging problem in the form of obscure GI bleed.

d) Case definition:

Any bleeding beyond ligament of Treitz is called as lower GI bleed. Mid GI bleed is any bleed between ampulla of Vater and ileocaecal valve. The source of bleed is colonic in 80-90% of the cases and small intestinal in 1-10% of the cases

II. INCIDENCE OF THE CONDITION IN OUR COUNTRY

It is less common than Upper GI bleed. Men are affected more commonly than women. An increased incidence is seen with increase in age. The incidence in Western countries is 20-30/10,000. Incidence data are not available for India.

III. DIFFERENTIAL DIAGNOSIS

LGI bleed may present with a constellation of clinical symptoms like anaemia, haematochezia, maelena, maroon coloured blood in the stools with or without constitutional features. The common causes of LGI bleed in the Indian context include anorectal causes (haemorrhoids, anal fissures), acute self limiting colitis (Shigella, E. coli), amoebiasis , enteric fever, colonic tuberculosis , inflammatory bowel disease (ulcerative colitis and Crohn's disease), solitary rectal ulcer syndrome , colon cancer, polyposis coli, radiation colitis, diverticular disease, NSAID induced bleed, Meckel's diverticulum and ischaemic colitis .

IV. PREVENTION AND COUNSELING

Acute self limiting colitis and parasitic diseases are often food or water borne diseases and maintaining high levels of hygiene may prevent these diseases.

V. OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS, TREATMENT & REFERRAL CRITERIA

*Situation 1: At Secondary Hospital/ Non-Metro situation: Optimal Standards of Treatment in Situations where technology and resources are limited

Clinical Diagnosis:

History should include following points, which may aid in clinical diagnosis:

- a) Age: Elderly are more predisposed to cancer colon, diverticular disease and ischaemic colitis. Pediatric age group is predisposed to bleed from rectal polyp or Meckel's diverticulum.
- b) History of fever : suggests infectious disease as a cause
- c) H/o of altered bowel habits for a long duration suggests inflammatory bowel disease or intestinal tuberculosis
- d) H/o constipation with bleed suggests haemorrhoidal bleed or solitary rectal ulcer syndrome
- e) Constitutional symptoms – anorexia, pain abdomen, weight loss
- f) History suggestive of partial intestinal or colonic obstruction
- g) H/o of drugs – NSAIDS, anticoagulants
- h) H/o of radiation, mainly to pelvic region in past
- i) Past H/o of similar or less threatening LGI Bleed
- j) Past H/o of endoscopic intervention e.g. polypectomy

Assess for

a) Type of blood per rectum - red, maroon or malaenic which depends upon source and rapidity of blood loss

b) Whether blood is mixed with stools, separate from stool or only blood is seen. Blood if separate from the stools suggests haemorrhoidal bleed or solitary rectal ulcer syndrome.

Investigations:

- É Assess for signs of blood volume loss-Pulse rate, BP
- É Assess for aetiology of bleed
 - a) Skin telangiectesia, Lymph nodes, abdominal tenderness, abdominal lump
 - b) Digital rectal examination – to look for anorectal causes and stool colour
 - c) Proctoscopy to rule out haemorrhoids and evaluate rectal mucosa
- É Assessment of other organ systems mainly cardiovascular and pulmonary

Treatment:

Standard Operating procedure

- É Resuscitation and assessment go hand in hand
- É Two large bore i.v.cannula or a central venous line if acute LGI bleed
- É Start crystalloids and plasma expanders, arrange packed RBC
- É Send samples for CBC, electrolytes, urea, coagulation profile and sample for grouping and cross match
- É Ryle's tube (RT) placement and check for RT aspirate
 - a. InPatient: Patients should be admitted if presenting with LGI bleed
 - b. OutPatient: If a patient presents with occult GI bleed and anaemia the management may be outpatient based if the anaemia is not very severe
 - c. DayCare: In occult GI bleed, the patient may be given blood transfusions to treat for symptoms resulting from anaemia and thereafter referred to a tertiary centre for further investigations

k) Referral criteria:

- É Continuous active bleed
- É Hemodynamic compromise either hypotension or orthostatic hypotension
- É Transfusion requirement of >2 packed RBCs to maintain haemodynamic stability
- É Presence with comorbid illness who require monitoring
- É Obscure GI bleed where the cause of bleed needs to be ascertained

***Situation 2: At Super Specialty Facility in Metro location where higher-end technology is available**

- a) **Clinical Diagnosis:** Same as mentioned before

b) **Investigations:**

Active LGI bleed:

- UGI endoscopy
- Colonoscopy
- Angiography
- Radio nuclide scan
- Exploratory Laparotomy with intra-operative endoscopy

Occult/Obscure GI bleed:

- Stool for occult blood
- Stool for parasites
- UGI endoscopy
- Colonoscopy
- Capsule endoscopy/Double balloon enteroscopy

- Computed tomography (Enteroclysis)
- Radionuclide scan
- Exploratory laparotomy with intra-operative endoscopy

c) Treatment:

- Endoscopic therapy :10% to 15% of patients undergoing urgent colonoscopy receive endoscopic therapy
 1. Injection therapy (epinephrine or saline)
 2. Heater probe therapy
 3. Argon plasma coagulation
 4. Haemoclips
- Radiology techniques
 1. Vasopressin infusion
 2. Selective embolisation:

Definitive means of controlling hemorrhage

- ó stops bleeding 67-100%
- ó Re-bleed 15%-40%
- ó High incidence of bowel ischemia

3. Super selective embolisation

Target artery - the vasa recta; also within the marginal arteries and the

distal intestinal arcades

A number of primary embolic agents

- a. Microcoils
- b. Gelatin sponge particles
- c. Poly vinyl sponge particles

- Surgery
 1. Bleeding refractory to other therapies
 2. Requirement of more than 8 units of blood over 24 hours
 3. Hemodynamic instability
 4. Re-bleeding after non-operative treatment, esp if localized
 5. Segmentectomy if site of bleed is known
 6. Right hemicolectomy if bleed from unknown

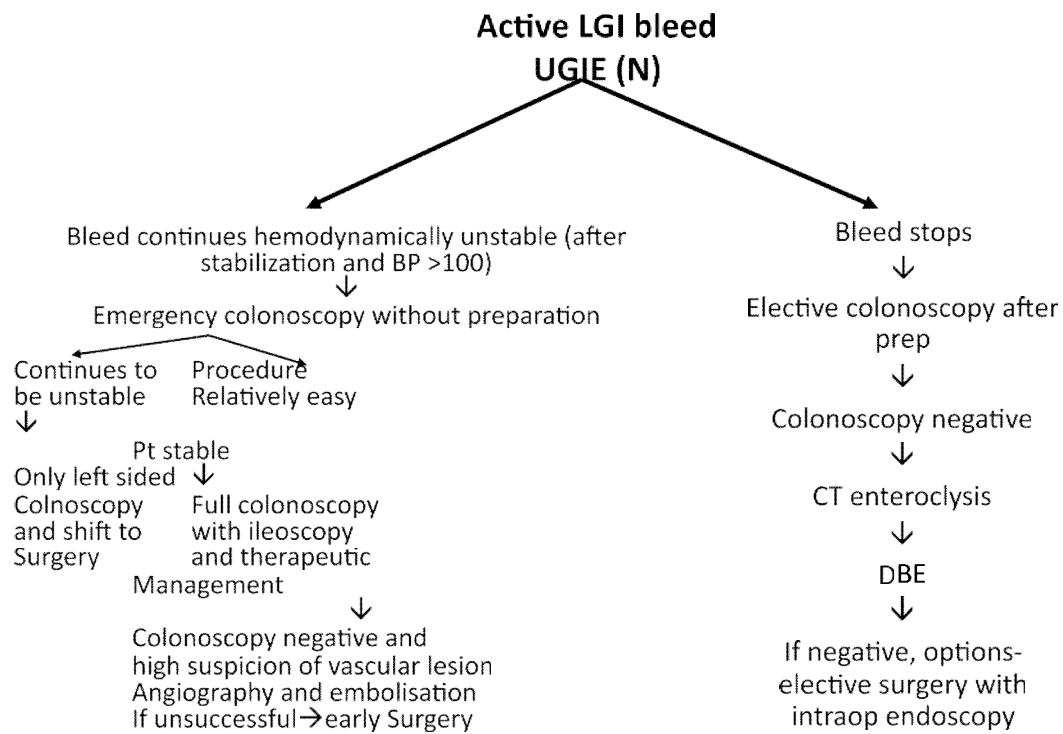
d) Referral centre:

Outcomes at a tertiary referral centre

- Mortality at 3 yr follow up 19%
- Most patients stop bleeding spontaneously
- Rebleed occurs in 10% to 40% of patients
- Between 5% and 50% of patients with persistent bleeding require surgical hemostasis

The figure below shows the algorithm of management of acute LGI bleed

(Abbreviations in the figure : UGIE : upper gastrointestinal endoscopy; CT : computed tomography; DBE : double balloon enteroscopy)



VI. WHO DOES WHAT? and TIMELINES

- Doctor*
- Nurse*
- Technician*

VII. FURTHER READING / REFERENCES

1: Lee J, Costantini TW, Coimbra R. Acute lower GI bleeding for the acute care surgeon: current diagnosis and management. *Scand J Surg.* 2009;98(3):135-42. Review. PubMed PMID: 19919917.

2: Singh V, Alexander JA. The evaluation and management of obscure and occult gastrointestinal bleeding. *Abdom Imaging.* 2009 May-Jun;34(3):311-9. Review.

PubMed PMID: 18581161.

3: Strate LL, Naumann CR. The role of colonoscopy and radiological procedures in the management of acute lower intestinal bleeding. *ClinGastroenterolHepatol.*

2010 Apr; 8(4):333-43.Review. PubMed PMID: 20036757

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE (PATIENT WEIGHT 60 KGS)

(Units to be specified for human resources, investigations, drugs and consumables and equipment. Quantity to also be specified)

Situation	HUMAN RESOURCES	INVESTIGATIONS	DRUGS & CONSUMABLES	EQUIPMENT
1				
2				

INTESTINAL OBSTRUCTION

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WHEN TO SUSPECT/ RECOGNIZE?

Introduction

Intestinal obstruction is an important gastrointestinal emergency and a common cause of hospitalization. Most of the patients with intestinal obstruction report to surgeons or surgical care units. Some of them however report to physicians or medical emergency units. . Intestinal obstruction carries a high risk of mortality and morbidity if surgical therapy is inappropriately delayed because of misdiagnosis.

Case definition

Acute intestinal obstruction occurs when there is an interruption in the forward flow of intestinal contents. This interruption can occur at any point along the length of the gastrointestinal tract. The clinical presentation varies depending on the severity, duration, site and type of intestinal obstruction..The classical clinical tetrad of presentation is colicky abdominal pain, nausea and vomiting, abdominal distention, and progressive obstipation. There are two types of intestinal obstruction-mechanical and adynamic.

Mechanical intestinal obstruction

Site of obstruction: The obstructive pathology may lie in the proximal small intestinal or distal intestine including the colon.

Grade and severity of the obstructing lesion: The intestinal obstruction could be either complete or incomplete (partial). While in the partial intestinal obstruction, the gas or liquid stool can pass through the point of narrowing, in complete intestinal obstruction, the obstruction is complete and there is no passage of gas or the liquid stool beyond the point of the obstruction. Partial obstruction is further classified as high grade or low grade according to the severity of the narrowing. In general, surgical intervention is often required for complete intestinal obstruction; partial intestinal obstruction can be treated using conservative measures. The mechanical intestinal obstruction could remain uncomplicated or may get complicated by vascular compromise, intestinal perforation and septicemia.

Adynamic intestinal obstruction

There are two types of adynamic intestinal obstruction - paralytic ileus and acute intestinal pseudo-obstruction.

It is important to classify intestinal obstruction into above mentioned categories as the treatment of intestinal obstruction will vary according to the site, severity and cause of the intestinal obstruction. Therefore, following questions should be addressed while dealing with a patient with intestinal obstruction:

1. Differentiation between complete mechanical obstruction, partial mechanical obstruction or adynamic obstruction
2. The site of obstruction: Small bowel obstruction (proximal or distal) vs. large bowel obstruction (colon/rectal)

Patho-physiology

In simple intestinal obstruction, the intestine is occluded at one point. Fluid and chyme accumulate proximal to the site of intestinal obstruction. This leads to impairment in intestinal water and electrolyte absorption and enhanced intestinal secretion resulting in volume depletion. Stasis of intestinal contents predisposes to bacterial overgrowth (predominantly anaerobes) in the segment proximal to obstruction. The bacterial overgrowth leads to bacterial fermentation and increased production of gas. Accumulation of liquid and gas in the intestine leads to intestinal dilatation which induces local inflammation and neuro-endocrine reflexes that initially increase enteric propulsive activity to attempt to overcome the obstruction. Intestinal motility gradually decreases as intestinal muscle becomes fatigued. Intestinal dilatation gradually compromises vascular perfusion because of increasing intraluminal pressure and intramural tension. Forward arterial flow is severely

compromised when the intraluminal pressure reaches the diastolic pressure, and ceases when the intraluminal pressure reaches the systolic pressure. Venous obstruction causes increased intraluminal fluid transudation and intramural edema. Almost 80% of blood flow to the intestine goes to the mucosa, therefore intestinal mucosa is highly sensitive to ischemia as it is perfused by end arteries and has a high rate of metabolic activity.

In *closed-loop* obstruction, an intestinal segment is occluded at two points. Common forms of closed loops include an incarcerated hernia, in which a loop of the intestine is compressed at both ends within a hernial sac, and volvulus, in which a loop of intestine is mechanically compressed at both ends because of a twist in the supporting mesentery. If the ileocecal valve is competent, an obstructive lesion in the colon causes a closed loop due to a pathologic mechanical obstruction at the distal end and a physiologic occlusion at the ileocecal valve proximally. If the ileocecal valve is incompetent, the obstructive lesion in the colon produces a simple obstruction as the colon is decompressed by way of the small intestine. A closed loop of intestine rapidly dilates because of the lack of a proximal and a distal outlet for the accumulated gas and liquid. The mucosa consequently develops ischemia rapidly. Extrinsic compression that strangulates mesenteric vessels (e.g. at a point of volvulus or at the neck of a hernial sac) exacerbates the ischemia and rapidly produces intestinal gangrene or necrosis and perforation.

INCIDENCE OF THE CONDITION IN OUR COUNTRY

The most common (75%) cause of small intestinal obstruction is post-operative adhesions. The rate of adhesive small intestinal obstruction development is highest in the first few postoperative years following the index surgery, particularly after colorectal surgery, but the risk remains life-long. The risk of recurrent intestinal obstruction from adhesions after 10 years of the index surgery varies widely from 15 to 50%. Although, post-operative intestinal obstruction recovers mostly with conservative management, 25 to 66% of them may require exploratory laparotomy and adhesiolysis. Although hernia was the second most important cause of intestinal obstruction, there has been a marked decline of intestinal obstruction due to hernia from 40% in the 1960s to 15%, presumably due to an increased rate of elective hernia repair. Other causes of intestinal obstruction include intestinal tuberculosis, bands, and volvulus.

OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS,

Clinical features of intestinal obstruction:

The clinical presentation varies depending on the severity, duration, and type of intestinal obstruction. The classical clinical tetrad is colicky abdominal pain, nausea and vomiting, abdominal distension and progressive obstipation.

Differentiation between proximal vs distal intestinal obstruction

Proximal intestinal obstruction typically produces epigastric pain that occurs every 3 to 4 minutes, with frequent bilious vomiting. Distal intestinal obstruction typically produces peri-umbilical pain that occurs every 15 to 20 minutes, with infrequent feculent emesis. Abdominal distension is more marked in the distal obstruction as there is a large length of the intestine proximal to the obstructive lesion, which can accumulate volumes of fluid and gas. Closed-loop obstruction often presents with pain out of proportion to the abdominal signs because of concurrent mesenteric ischemia. Inspection occasionally reveals visible peristalsis. Air-filled intestinal loops produce abdominal tympany. Palpation reveals mild generalized abdominal tenderness. Peritoneal signs, such as rebound tenderness and guarding, are typically absent, unless intestinal necrosis or perforation supervenes. Intestinal sounds are initially high-pitched (tinkling) and hyperactive, with audible rushes or borborygmi corresponding to paroxysms of abdominal pain, but become progressively hypoactive and softer, and then disappear because of intestinal fatigue. Patients with obstructive lesion in the rectum may give a history of spurious diarrhoea, altered bowel habit and progressive constipation before presenting with large bowel obstruction.

Features that suggest complications

The development of rigors, high fever, or systemic toxicity suggests that the obstruction may be complicated by intestinal necrosis or perforation. Intussusception typically presents in infants with episodic abdominal pain, currant jelly stools, and bilious vomiting. Physical examination reveals an acutely ill, restless, and febrile patient. Signs of intravascular volume depletion include tachycardia, orthostatic hypotension, dry mucous membranes, and poor skin turgor. Jaundice suggests possible gallstone ileus or malignant obstruction.

Features that suggest aetiology of intestinal obstruction

A good systemic and abdominal examination can provide important clues to the aetiology of the intestinal obstruction. A history of previous abdominal surgery especially intestinal surgery can point towards the most common cause of intestinal obstruction i.e. postoperative adhesive intestinal obstruction. Other causes of intestinal obstruction such as abdominal scars, clinically obvious external incarcerated hernia (femoral, inguinal, umbilical, or incisional), per-rectal examination (fecal impaction, or a rectal mass), and digital examination of the stoma to rule out stomal obstruction in patients with colostomy or ileostomy. Presence of hepatomegaly, splenomegaly, a palpable abdominal mass, or periumbilical (Sister Mary Joseph nodule), inguinal, and right

supraclavicular (Virchow node) lymphadenopathy may suggest presence of a malignant obstructive lesion in the intestine with systemic dissemination.

Evaluation at the time of acute obstruction

Plain upright abdominal radiograph

Plain upright abdominal X-ray film should be done for initial confirmation of intestinal obstruction in patients presenting with clinical signs and symptoms of intestinal obstruction. It can confirm intestinal obstruction in approximately 60% and its positive predictive value approaches 80% in patients with high-grade intestinal obstruction. Presence of multiple air fluid levels (> 3) in the erect film is an important sign pointing towards the diagnosis of intestinal obstruction. At times when the patient is too sick a decubitus film also may help in detecting the presence of air fluid levels. Large air fluid levels occurring more or less at the same level on the radiograph are suggestive of paralytic ileus whereas air fluid levels at different levels are more likely in a dynamic obstruction. Additionally, the abdominal film can show dilation of intestinal loops (dilation >3 cm), and a transition zone between dilated and non dilated intestinal segment. The pattern of dilation can suggest the site of intestinal obstruction. While the supine abdominal film in patients with small intestinal obstruction shows dilatation of multiple loops of small intestine, with a paucity of air in the large intestine, the large intestinal obstruction shows dilatation of the colon, with decompressed small intestine in the setting of a competent ileocecal valve. It is important to assess the diameter of the distended caecum in such cases as dilatation beyond 10 cm indicates impending caecal rupture. Air-filled loops of small intestine are distinguished from those of large intestine by their more central abdominal location, by their narrower calibre even when dilated, and by the presence of valvulae conniventes that extend across the entire luminal diameter as opposed to the colonic haustral folds that extend only partially across the luminal diameter. Presence of free air under the diaphragms immediately confirms intestinal perforation. Although this finding diminishes in significance if it is found on an X-ray done in the early postoperative period following an abdominal surgery.

Limitation of abdominal radiograph: Plain abdominal film can appear normal in early obstruction and in high gut obstruction (jejunal or duodenal obstruction) and thus may be misleading.

Next step:

An abdominal CT scan, non contrast and contrast enhanced (both oral and intravenous contrast; water-soluble contrast preferred) is appropriate for further evaluation of patients with suspected intestinal obstruction in whom clinical examination and radiography do not yield a definitive diagnosis. CT is very useful in a patient suspected to have intestinal obstruction and it can show all that necessary information about the intestinal obstruction. The presence of a discrete transition point helps guide operative planning.

CT findings in patients with intestinal obstruction include dilated loops of intestine proximal to the site of obstruction, with distally decompressed intestine. In addition, CT can identify emergent causes of intestinal obstruction, such as volvulus or intestinal strangulation. Although CT is highly sensitive and specific for high-grade obstruction, its value diminishes in patients with partial obstruc-

tion. In these patients, oral contrast material may be seen traversing the length of the intestine to the rectum, with no discrete area of transition.

A C-loop of distended intestinal with radial mesenteric vessels with medial conversion is highly suspicious for intestinal volvulus. Thickened intestinal walls and poor flow of contrast material into a section of intestinal suggests ischemia, whereas pneumatosisintestinalis, free intra-peritoneal air, and mesenteric fat stranding suggest necrosis and perforation. The CT can also identify intussusceptions easily by picking up the intussusceptum in the intussuscepiens, as the mesenteric fat and vasculature can be identified within the lumen of the intestine or a 'double target sign' can be seen on cross-sectional imaging.

The American College of Radiology recommends non-contrast CT as the initial imaging modality of choice. However, because most causes of small intestinal obstruction will have systemic manifestations or fail to resolve necessitating operative intervention the additional diagnostic value of CT compared with radiography is limited. Radiation exposure is also significant. Therefore, in most patients, CT should be ordered when the diagnosis is in doubt, when there is no surgical history or hernias to explain the etiology, or when there is a high index of suspicion for complete or high-grade obstruction. The CT signs of strangulation include: a thickened intestinal wall because of intestinal wall edema, inflammation, or intramural hemorrhage; mural thumb-printing from intramural hemorrhage or edema; pneumatosisintestinalis from intramural gas produced by bacteria; absence of enhancement with intravenous contrast because of vascular hypoperfusion; hazy or streaky mesentery or "dirty" fat from inflammatory infiltration, portal venous gas, target sign, and ascites.

Evaluation after resolution of an episode of intestinal obstruction with conservative management

After the acute intestinal obstruction has resolved, the cause of intestinal obstruction should be investigated and treated appropriately (medical or surgical) depending upon the diagnosis. The basic principal of investigation is to find out site, cause, extent and etiology of the intestinal obstructive disease.

- Small intestine evaluation: Barium meal follow through (BMFT), Enteroclysis or CT enteroclysis
- Large intestinal evaluation: Preferably colonoscopy and if not available barium enema
- Histological examination of the biopsies for pathological diagnosis
- Microbiological tests such as PCR and culture for tuberculosis in appropriate clinical setting

Evaluation after control of an episode of intestinal obstruction with surgery

Often, in patients with refractory adhesive intestinal obstruction, all that is required at laparotomy is adhesiolysis to separate the adhesions and relieve the obstruction. In such cases there is no need for any excision of intestine unless there is a strangulated gangrenous segment of intestine. Whenever

resection is warranted, the resected specimen should be evaluated both histologically and microbiologically to determine the etiology. In some patients, inflammatory lesions such as tuberculosis or Crohn's disease, malignant lesions such as adenocarcinomas/gastrointestinal stromal tumors or lymphomas, a specific treatment will be required for the management of the disease postresection.

TREATMENT & REFERRAL CRITERIA

General measures

The principles in management of acute intestinal obstruction include correction of abnormalities in the fluid and electrolyte imbalances, gastric decompression, and bowel rest, prevention of infection, analgesics, and specific treatment for cause of obstruction.

- *Monitoring of clinical and hemodynamic status*
 - Pulse, Blood pressure

 - Respiratory rate

 - Abdominal girth

 - Intestinal sound

 - Abdominal tenderness

- *Fluid and electrolyte balance*
 - Intravenous fluid: Isotonic crystalloids

 - Arterial blood gas: Correction of acid base abnormalities

 - Correction of electrolyte imbalance, especially hypokalemia

 - Measurement of urine output

- *Symptomatic treatment*
- *Oral administration of water-soluble contrast agent (Urografin)(see below)**
Analgesics such as intravenous anti-cholinergics or NSAIDS

- *Anti-emetics:* Metoclopramide, ondansetron
- *Antibiotics:*
 - Antibiotics are used to treat intestinal overgrowth of bacteria and translocation across the intestinal wall. The presence of fever and leukocytosis should prompt inclusion of antibiotics in the initial treatment regimen. Antibiotics should have coverage against gram-negative organisms and anaerobes, and the choice of a specific agent should be determined by local susceptibility and availability.

- *Nutritional support:*
 - If intestinal obstruction is prolonged (for example, in patients with post operative state) and the patients cannot be fed enterally, they should be given total parenteral nutrition. Generally speaking, a previously healthy adult can tolerate 5-7 days of fasting or bowel rest without any significant clinical debility. Beyond this period appropriate parenteral nutritional support is advisable.

Outcome following conservative management

Conservative management is successful in 40 to 70 percent of clinically stable patients, with a higher success rate in those with partial intestinal obstruction. Although conservative management is associated with shorter initial hospitalization, there is also a higher rate of eventual recurrence. With conservative management, resolution generally occurs within 24 to 48 hours. Beyond this time frame, the risk of complications, including vascular compromise, increases. If intestinal obstruction is not resolved with conservative management, surgical evaluation is required.

Patients who develop *adhesive intestinal obstruction* of the small intestine in the postoperative period (both early and late) constitute a special group of patients in whom a new form of conservative intervention is indicated as outlined below. After the baseline abdominal radiographs have documented intestinal obstruction, the next step is to administer 100 ml of urograffin (water-soluble contrast agent) diluted in 400 ml of saline or drinking water orally or through the nasogastric tube which has already been inserted as part of the conservative management of intestinal obstruction. Thereafter, abdominal radiographs are obtained at 8-12 h intervals till 24 hours of ingestion of urograffin. The appearance of contrast in the colon within 8-24 h after its administration predicts resolution of small intestinal obstruction with a sensitivity of 96% and specificity of 98%. Water soluble contrast agent administration is effective in reducing the need for re-operation and shortening the hospital stay compared with conventional 'drip-and-suck' regimen. This simple therapeutic maneuver also has a diagnostic value and can delineate the level of obstruction in those patients in whom the contrast fails to reach the large intestine in the stipulated time frame. In those patients in whom water soluble contrast agent does not reach colon, the probability of need for surgery is higher. While administration of water soluble contrast agent can occasionally be repeated, one should keep a close vigil on abdominal signs and clinical status of the patient. This modality can also be used in other patients with intestinal tuberculosis or Crohn's disease who are likely to have a partial small intestinal obstruction.

Indications for surgery

Although there are clinical features which allow a clinician to decide about the time of surgery and also predict or suggest resolution of intestinal obstruction with conservative management in the index patient, such a prediction is challenging at occasions. As such, presence of fever, tachycardia, leukocytosis, or anion-gap acidosis, in the setting of abdominal imaging consistent with obstruction constitutes a low threshold for laparotomy. Peritonitis remains an absolute indication for exploration today as it did decades ago. There are clinical scenarios that are considered somewhat exempt from an "early" operative approach in the absence of peritonitis. These include small bowel

obstruction in the early postoperative period (unless following laparoscopic surgery), tuberculosis, Crohn's disease, or carcinomatosis. Postoperative small bowel obstruction occurs within 4–6 weeks of abdominal surgery with an estimated incidence of 1–4%. Caused by filmy adhesions in most open cases, up to 90% of these obstructions resolve (dissolve or reform) with non-operative management within 7–14 days of the index operation. The exception to this watchful waiting approach is postoperative obstruction following laparoscopic surgery which warrants an early surgical approach more often than not. The cause of obstruction will depend on the underlying operation, but it is likely that (amongst other causes) intestine is incarcerated within a peritoneal defect caused by trocar placement. Clinical situations such as, volvulus, obstructed hernia, large bowel obstruction with gross cecal dilatation and instances where a closed loop obstruction is suspected should have a low threshold for surgical intervention.

Patients with an intestinal obstruction that resolves after reduction of a hernia should be scheduled for elective hernia repair, whereas immediate surgery is required in patients with an irreducible or strangulated hernia. Stable patients with a history of abdominal malignancy or high suspicion for malignancy should be thoroughly evaluated for optimal surgical planning. Abdominal malignancy can be treated with primary resection and reconstruction or palliative diversion, or placement of venting and feeding tubes. There is emerging evidence to suggest that patients with colonic/rectal obstruction due to malignancies are best treated by emergency surgical diversion/or resection (wherever feasible) rather than colonoscopic stenting.

Patients with post operative adhesive obstruction

Treatment of stable patients with intestinal obstruction and a history of abdominal surgery present a challenge. Conservative management of a high-grade obstruction should be attempted initially, using nasogastric suction and decompression, aggressive intravenous rehydration, and antibiotics. Most show improvement in 48 hours. If not, then a therapeutic trial with water soluble contrast agent (as described above) is warranted in the absence of peritoneal signs.

Monitoring
Hemodynamic status
Vitals
Abdominal girth

Bad signs
Tachycardia
Tachypnea
Increase in abdominal girth
Persistent obstruction
GI bleed
Worsening of vitals
Persistence of absence of bowel sound
Appearance of rebound tenderness
Fever

Specific measures

The cause of the intestinal obstruction should be treated appropriately.

*Situation 1: At Secondary Hospital/ Non-Metro situation: Optimal Standards of Treatment in Situations where technology and resources are limited

- a) Clinical Diagnosis: As above
- b) Investigations: Haemogram, electrolytes, sugar, urea levels, chest and abdominal X rays
- c) Treatment:
 - Standard Operating procedure
 - a. Inpatient: All patients suspected or confirmed to have intestinal obstruction should be admitted and treated appropriately as described above.
 - b. Outpatient: Not applicable
 - c. Day Care: not applicable
- d) Referral criteria:

Non availability of a surgeon, basic facilities as outlined above

A sick patient

Failed conservative management

Complete obstruction

Complicated obstruction

Perforation

Obstruction secondary to suspected malignancy

Obstruction in the setting of IBD

*Situation 2: At Super Specialty Facility in Metro location where higher-end technology is available

- a) Clinical Diagnosis: As above
- b) Investigations: Hemogram, electrolytes, RFT, LFT, Blood gases, Serum lactate levels, X-ray: chest/abdomen, CT scan, endoscopic facilities

- c) Treatment:
Standard Operating procedure
- a. Inpatient: as outlined above
 - b. Outpatient Not recommended; provide follow up and counseling after discharge
 - c. Day Care: Not recommended in the acute setting;

d) Referral criteria:None

WHO DOES WHAT? and TIMELINES

- a. *Doctor*
- b. *Nurse*
- c. *Technician*

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE (PATIENT WEIGHT 60 KGS)

(Units to be specified for human resources, investigations, drugs and consumables and equipment.
Quantity to also be specified)

Situation	HUMAN RESOURCES	INVESTIGATIONS	DRUGS & CONSUMABLES	EQUIPMENT
1	2	2	-	2
2	3	1	-	3

PANCREATIC CARCINOMA

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I. WHEN TO SUSPECT/ RECOGNIZE?

a) Introduction:

The most common histological type of pancreatic cancer is ductal adenocarcinoma, which accounts for over 90% of all tumors. Most pancreatic cancers are advanced at the time of diagnosis. The overall 5-year survival rate is 4%. Because the 5-year survival of this condition is so poor, incidence and mortality rates are virtually identical. At the time of diagnosis, approximately 80% of all pancreatic cancers are advanced. The 5-year survival of operated patients approaches 40% when performed at specialized medical institutions. Pancreatic cancer is rare before the age of 45 years and 80% of cases occur in the 60–80 year age group.

b) Case definition:

For both situations of care (*mentioned below**)

The three main symptoms of pancreatic cancer are pain, loss of weight, and jaundice. However, signs and symptoms are non-specific.

Cases of pancreatic cancer are defined by image guided biopsy, surgical resection or intra-operative biopsy, or suggestive imaging features with a progressive and compatible clinical course on follow up.

II. INCIDENCE OF THE CONDITION IN OUR COUNTRY

The incidence of pancreatic cancer in India has traditionally been thought to be low (0.5-2.4 per 100,000 men and 0.2-1.8 per 100,000 women). However, this is likely to be a reflection of under-recognition and under-reporting. The incidence of pancreatic cancer is higher in the urban male populations of western and northern parts of India. Time trends show that the incidence is increasing for both carcinoma of the pancreas and also peri-ampullary tumors.

The annual pancreatic cancer load of India in 2001 was approximately 14,230 patients: the estimated current figure is approximately 17,000 and is likely to increase in the near future.

III. DIFFERENTIAL DIAGNOSIS

- 1) Chronic pancreatitis, including autoimmune pancreatitis.
- 2) Acute pancreatitis, including focal pancreatitis / pseudo-tumoral pancreatitis / groove pancreatitis.
- 3) Metastasis to the pancreas from renal cell carcinoma, melanoma, breast cancer, colon cancer e.t.c

IV. PREVENTION AND COUNSELING

No simple screening test is available for the general population.

The strongest etiological association of pancreatic cancer is with cigarette smoking. Hence health education to reduce tobacco consumption should lower the risk of developing pancreatic carcinoma.

Other high-risk groups include patients with chronic pancreatitis, adult onset diabetes of less than two years' duration, hereditary pancreatitis, familial pancreatic cancers, and certain familial cancer syndromes. However, there are no screening or preventive guidelines for these higher risk groups.

V. OPTIMAL DIAGNOSTIC CRITERIA, INVESTIGATIONS, TREATMENT & REFERRAL CRITERIA

INVESTIGATIONS,

The goals of investigations are:

- (a) Establishing the diagnosis, and
- (b) Assessing the tumor resectability

The following investigations can be used as indicated:

- 1) Abdominal ultrasound.
- 2) Computerized tomography (CT) scan.

- 3) Magnetic resonance (MR), including magnetic resonance cholangiopancreatography (MRCP).
- 4) Positron emission tomography (PET) - CT scan
- 5) Endosonography (EUS).
- 6) Laparoscopy with laparoscopic ultrasonography

Endoscopic retrograde cholangiopancreatography (ERCP) is now seldom used for the primary diagnosis. However, it can be used to obtain intra-ductal tissue for diagnosis, when biliary drainage is planned.

TREATMENT

(a) Curative treatment

Pancreaticoduodenectomy (with or without pylorus preservation) is the most appropriate resectional procedure for tumors of the pancreatic head. Left sided resection (with splenectomy) is appropriate for localized carcinomas of the body and tail of the pancreas. Involvement of the splenic vein or artery is not in itself a contraindication to such resection.

(b) Neoadjuvant treatment

Neoadjuvant treatment is indicated for tumors considered borderline resectable. This category includes the following:

- (1) No distant metastases.
- (2) Venous involvement of the SMV/portal vein demonstrating tumor abutment with or without impingement and narrowing of the lumen, encasement of the SMV/portal vein but without encasement of the nearby arteries, or short segment venous occlusion resulting from either tumor thrombus or encasement but with suitable vessel proximal and distal to the area of vessel involvement, allowing for safe resection and reconstruction.
- (3) Gastroduodenal artery encasement up to the hepatic artery with either short segment encasement or direct abutment of the hepatic artery, without extension to the celiac axis.
- (4) Tumor abutment of the SMA not to exceed greater than 180 degrees of the circumference of the vessel wall.

Gemcitabine or gemcitabine-based combination therapy may be considered as initial therapy prior to 5-FU-based chemoradiation.

(c) Palliative treatment

Palliative treatment is indicated for unresectable tumors, which are defined by the following criteria, according to the tumor location:

Pancreatic head tumors:

- 1) Distant metastases
- 2) Greater than 180 degrees SMA encasement, any celiac abutment
- 3) Unreconstructible SMV/portal occlusion
- 4) Aortic invasion or encasement

Pancreatic body tumors:

- 1) Distant metastases
- 2) SMA or celiac encasement greater than 180 degrees
- 3) Unreconstructible SMV/portal occlusion
- 4) Aortic invasion

Pancreatic tail tumors:

- 1) Distant metastases
- 2) SMA or celiac encasement greater than 180 degrees

All tumors with metastases to lymph nodes beyond the field of resection should be considered unresectable.

1. Surgical palliation

Surgery for palliative purpose is now rarely indicated. It may have some role in resource-limited setting. It may also be indicated if the patient is found to be unresectable during laparotomy. Duodenal and biliary bypass should be used during palliative surgery. The biliary bypass should be constructed with the bile duct in preference to the gall bladder.

2. Medical palliation

Patients should have access to palliative care specialists. Pain relief should be achieved using a progressive analgesic ladder.

Patients with pancreatic head tumors, who require relief of jaundice will need biliary stenting. Endoscopic stent placement is preferable to trans-hepatic stenting. Both plastic and self-expanding metal stents (SEMS) are effective in achieving biliary drainage but SEMS are preferable for patients with longer life expectancy (more than 6 months). Currently, the choice between these stents depends on clinical factors, local availability, and local expertise.

Plastic stents have been traditionally placed to relieve jaundice before surgery. However, covered SEMS can also be placed without compromising the surgery and afford a faster relief of jaundice.

Gastric outlet obstruction can be palliated with enteral SEMS.

Treatment of associated depression.

Pancreatic enzyme replacement for pancreatic insufficiency.

Low molecular weight heparin for prophylaxis of thromboembolic disease.

Gemcitabine is considered the standard front line chemotherapy for patients with metastatic disease. Gemcitabine combinations have shown a favorable impact on time to progression or survival for patients with good performance status.

***Situation 1: At Secondary Hospital/ Non-Metro situation: Optimal Standards of Treatment in Situations where technology and resources are limited**

a) **Clinical Diagnosis:**

b) **Investigations:**

- 1) Liver function tests.
- 2) CA 19.9 levels (CA 19-9 may be elevated in cases of benign biliary obstruction and does not represent an appropriate baseline until the patient is decompressed. In addition, CA19-9 may be undetectable in Lewis-a negative individuals).
- 3) Chest imaging.
- 4) Abdominal ultrasound.
- 5) CT or MRI scanning.

c) Treatment:

- 1) Nutritional build up and early referral for potentially resectable patients.
- 2) Symptom palliation for unresectable patients.

Standard Operating procedure

- a. In Patient
- b. Out Patient
- c. Day Care

d) Referral criteria:

- 1) Suspected carcinoma pancreas, where the diagnosis cannot be established with the existing resources.
- 2) Potentially resectable patients for further staging.
- 3) Potentially resectable patients for pancreaticoduodenectomy surgery to a high volume center.
- 4) Unresectable patients, with specialized palliation needs, like celiac plexus neurolysis, patient controlled analgesia, stenting for relief of biliary obstruction e.t.c.

***Situation 2: At Super Specialty Facility in Metro location where higher-end technology is available**

a) Clinical Diagnosis:

b) Investigations:

In addition to above:

- 1) PET-CT scan
- 2) Endoscopic ultrasound.
- 3) Laparoscopy and laparoscopic ultrasound.
- 4) Guided biopsy techniques for patients considered not suitable for surgical resection. If biopsy is required in a resectable lesion, then EUS-directed FNA biopsy is preferable to a CT-guided FNA because of lower risk of peritoneal seeding with the former. Biopsy proof of malignancy is not required before surgical resection and a non-diagnostic biopsy should not delay surgical resection when the clinical suspicion for pancreatic cancer is high.

c) Treatment:

In addition to above, super-specialty facilities should also have:

- 1) Facilities for assessment of resectability.
- 2) Increased therapeutic resources, including oncologic treatment.
- 3) Adequate surgical expertise for pancreatic resections.
- 4) Specialized services in histopathology, intensive care, and palliative care.
- 5) Facilities for the organization and conduct of local, national, and international trials.

Standard Operating procedure

- a. In Patient**
- b. Out Patient**
- c. Day Care**

VI. WHO DOES WHAT? and TIMELINES

a. **Doctor**

Clinical evaluation, decides investigations, interprets the investigations, and generates a treatment plan.

b. **Nurse**

Ensures adequacy of palliative care, and carries out the treatment plan.

c. **Technician**

Carries out the investigations planned.

VII. FURTHER READING / REFERENCES

1. NCCN guidelines:
http://www.nccn.org/professionals/physician_gls/pdf/pancreatic.pdf(accessed on 20.03.11).
2. Jemal A, Siegel R, Xu J, Ward E. Cancer statistics, 2010. CA Cancer J Clin 2010;60:277-300. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/20610543>.
3. Cascinu S, Falconi M, Valentini V, Jelic S. Pancreatic cancer: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up. Ann Oncol 2010; 21 Suppl 5 ():v55-8.
4. Tempero M, Arnoletti JP, Ben-Josef E, Bhargava P, Casper ES, Kim P, Malafa MP, Nakakura EK, Shibata S, Talamonti M, Wang H, Willett C. Pancreatic adenocarcinoma. Clinical Practice Guidelines in Oncology. J Natl Compr Canc Netw 2007; 5 (10):998-1033.
5. Guidelines for the management of patients with pancreatic cancer periampullary and ampullary carcinomas. Gut 2005; 54 Suppl 5 (): v1-16.

RESOURCES REQUIRED FOR ONE PATIENT / PROCEDURE (PATIENT WEIGHT 60 KGS)

(Units to be specified for human resources, investigations, drugs and consumables and equipment.
Quantity to also be specified)

Situation	HUMAN RESOURCES	INVESTIGATIONS	DRUGS CONSUMABLES	&EQUIPMENT
1				
2				