NEWSLETTER OF SRI LANKA SOCIETY OF GASTROENTEROLOGY

# SRI LANKA GASTROENTEROLOGY

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## SRI LANKA GASTROENTEROLOGY
## Editorial Board 2022




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# Abdominal pain in a young woman

Initial assessment of acute onset right sided abdominal pain

This graphic offers a step-by step approach to assessing the most urgent and common conditions that could be affecting a patient with this presentation. Several of these are time-critical, and timely referral to the right specialty will expedite the most appropriate care for the patient. Good communication is vital - transparency is sometimes more important than having all the answers.

**Young woman***

Presenting to emergency department with acute right sided abdominal pain

Not pregnant, or uncertain of pregnancy

* Applies to any patient with childbearing potential – those of reproductive age, with female reproductive organs

## Manage critical issues

The ABCDE framework is the starting point for any unwell patient

| A | B | C | D | E |
|---|---|---|---|---|
| Airway | Breathing | Circulation | Disability | Exposure |

+ urgent pregnancy test (urine βhCG) if patient is of reproductive age

**Clinically stable**

**Clinically unstable**

Initiate resuscitation

Positive βhCG and/or heavy vaginal bleeding — YES / NO

Ruptured ectopic pregnancy is an emergency, and must not be missed

**Refer urgently to gynaecology**

## Detailed history and examination

The challenge is to understand whether referral is needed to surgery, gynaecology, general medicine, or whether the patient can be managed conservatively or in primary care

### Testing

- Full blood count
- C reactive protein
- Renal and liver function
- Cross match bloods if imminent surgery is likely

### Imaging

- Ultrasound (transabdominal and/or transvaginal) to exclude ovarian pathology
- CT scan if suspected perforation or diagnostic uncertainty

**YES** High temperature (>38°C)? **NO**

Very late sign of ruptured appendix, if peritonism and sepsis are evident

## Consider appendicitis

Migratory pain | Localised peritonism | Rosvig's sign positive

NO / YES

Surgical referral for suspected appendicitis

## Consider sepsis

or other conditions requiring emergency treatment such as:

Pyelonephritis | Hollow viscus perforation | Pancreatitis | Tubo-ovarian abscess

## Gastrointestinal symptoms

An important diagnosis not to miss is a first presentation of Crohn's disease. Consider the symptomatic triad of:

- Chronic mucoid watery diarrhoea without blood
- Weight loss
- Chronic, intermittent, colicky pain

NO / YES

## Further investigations

- Family history
- Consider symptoms from mouth to anus
- Perform a digital rectal examination if appropriate

**General medical referral for suspected Crohn's disease**

## Consider other gynaecological causes

### Ovarian torsion

Patient may give history of:

- Known ovarian cysts
- Severe colicky pain
- Vomiting

### Pelvic inflammatory disease

Patient may give history of:

- Fevers
- Previous STI
- Abnormal vaginal discharge
- + Abscess or sepsis

### Ectopic pregnancy

### Miscarriage

+ Major haemorrhage, sepsis, or cervical shock

Chronic pelvic pain

Endometriosis | Dysmenorrhoea

Ovarian cyst rupture or haemorrhage

Mittelschmerz | Malignancy (rare)

## Safety net

Some intra-abdominal pathology (such as appendicitis) is an evolving diagnosis so providing good safety netting and empowering the patient by involving them in their management is key

**Discharge may be appropriate if low likelihood of clinically significant pathology**

# Acute Liver Failure

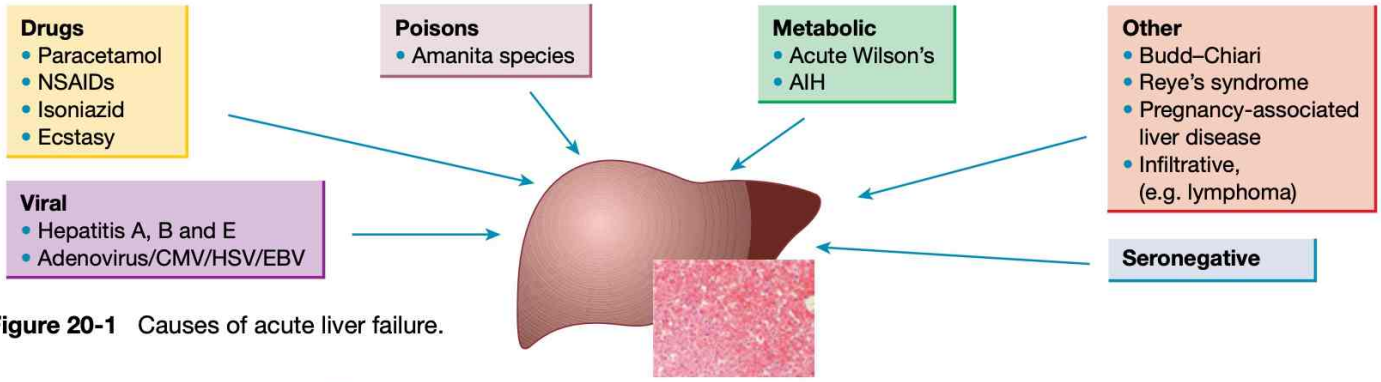



**Figure 20-1** Causes of acute liver failure.

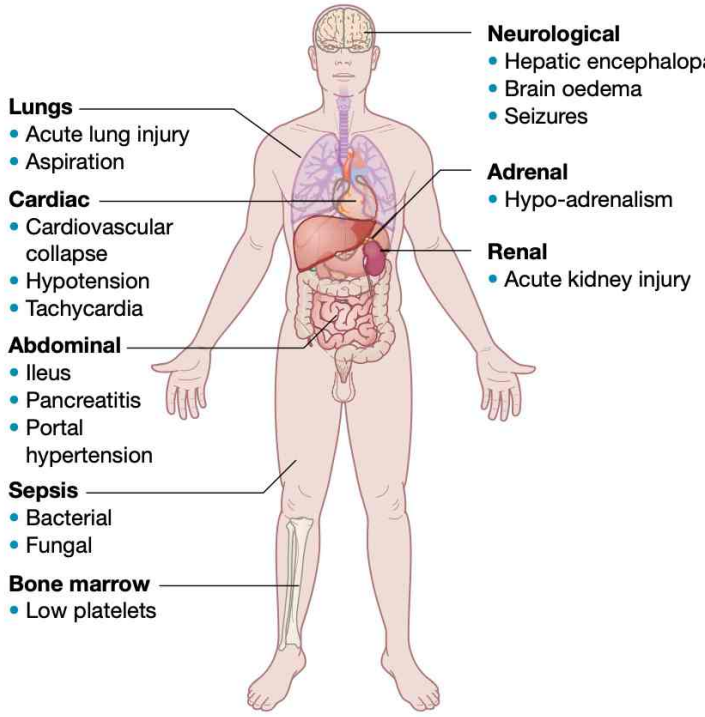



**Figure 20-2** Clinical manifestations.

**Table 20-1** Classification.

| | Hyperacute | Acute | Subacute |
|---|---|---|---|
| **Time from jaundice to HE** | 0–7 days | 1–4 weeks | 4–12 weeks |
| **Typical cause** | Paracetamol | Hepatitis A, B and E | Drug induced (non-paracetamol) |
| **Jaundice** | Mild | Moderate | Severe |
| **Coagulopathy** | Severe | Moderate | Mild |

**Table 20-2** King's College criteria for transplantation.

| Paracetamol-induced ALF | Non-paracetamol induced ALF |
|---|---|
| pH<7.3 (following volume resuscitation, irrespective of grade of HE)<br>*or*<br>Grade 3 or 4 HE<br>Creatinine > 300 µmol/L<br>INR >6.5<br>Arterial lactate >3.5 mmol/L at 4 h or >3 mmol/L at 12 h (following volume resuscitation) | INR > 6.5<br>*or*<br>Any three of the following:<br>• Etiology: seronegative hepatitis or drug induced<br>• Age <10 or >40 years<br>• Jaundice to encephalopathy >7 days<br>• Bilirubin >300 µmol/L<br>• INR >3.5 |

**Box 20-1 Investigations for acute liver failure**

**Haematology**
- Full blood count with blood film
- INR and clotting studies
- Haemolysis screen
- Pro-thrombotic screen*
- Bone marrow aspirate and trephine*

**Biochemistry**
- U and Es
- LFTs
- Arterial blood gases and arterial lactate
- Amylase
- Toxicology screen including paracetamol and salicylate levels
- Copper studies
- Serum urate*

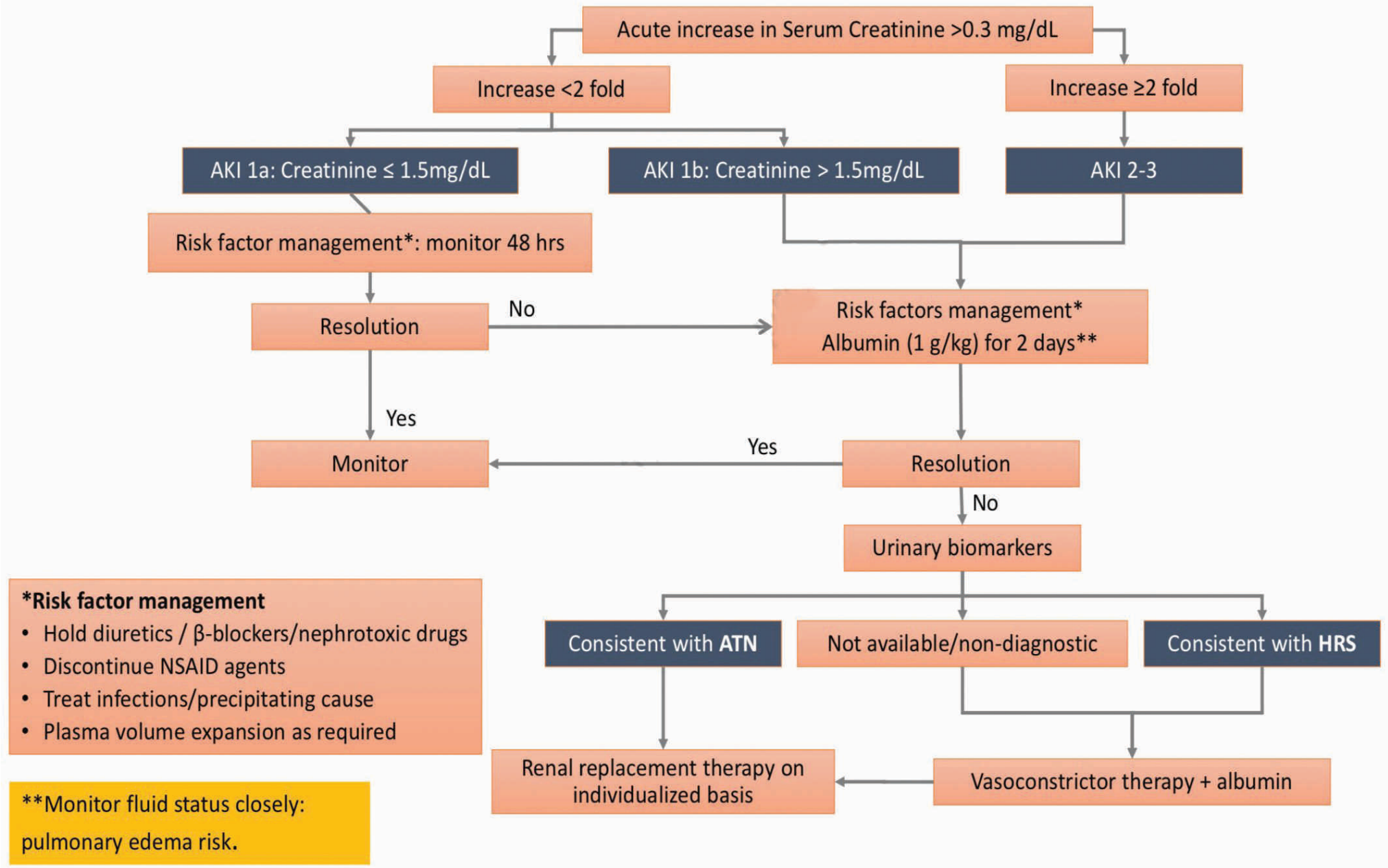
**Immunology**
- Auto-antibodies
- Immunoglobulin profile

**Virology/serology**
- Heptitis A IgM
- Hepatitis B (sAg, sAb, IgM core Ab, viral load)
- Hepatitis C Ab and viral load
- Hepatitis E IgM and viral load
- CMV IgM and viral load
- HSV IgM and viral load
- Leptospira IgM

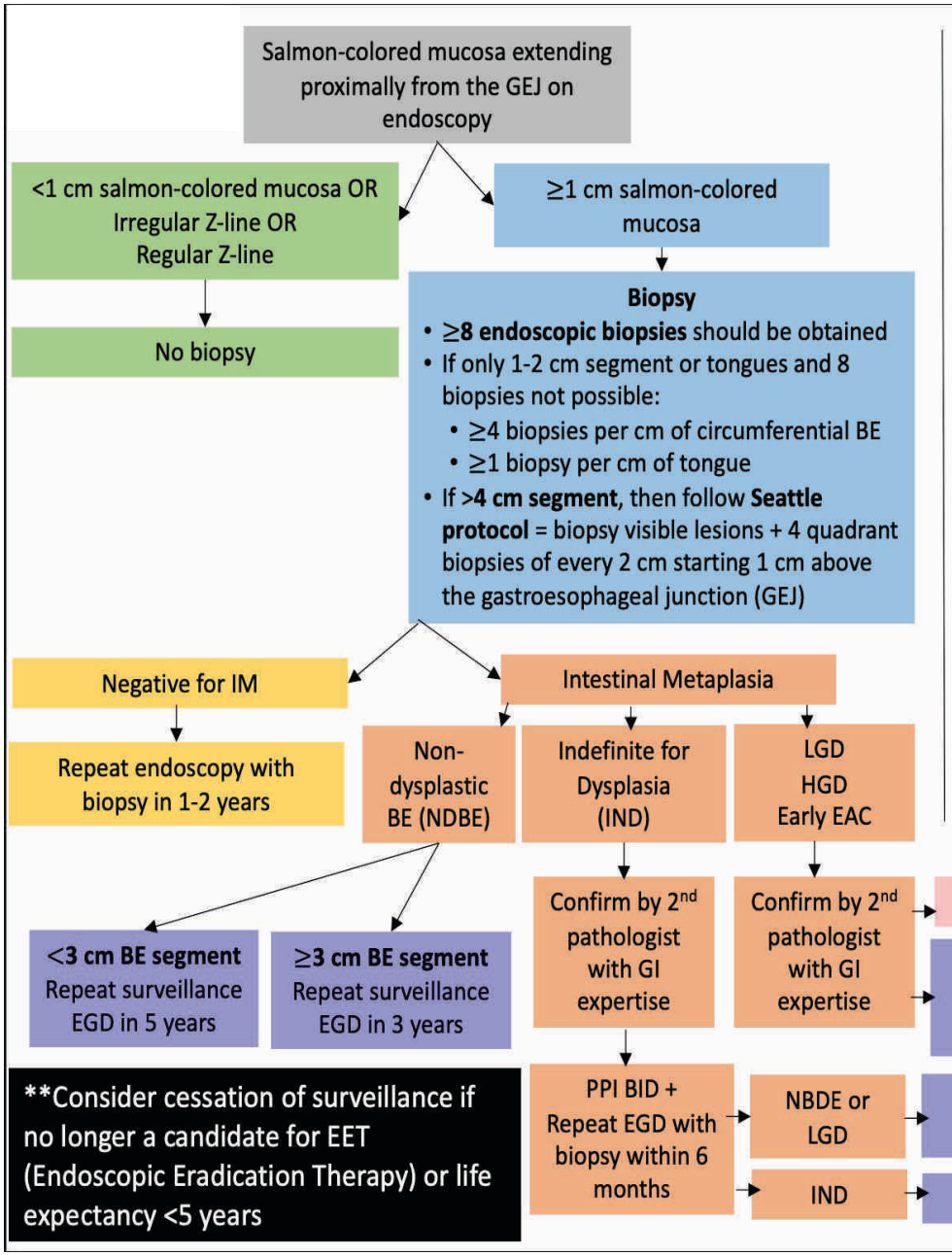
**Imaging**
- Doppler liver US
- CT with contrast

* When clinically indicated

# Hepatorenal Syndrome



# Screening & Surveillance of Barrett's Oesophagus



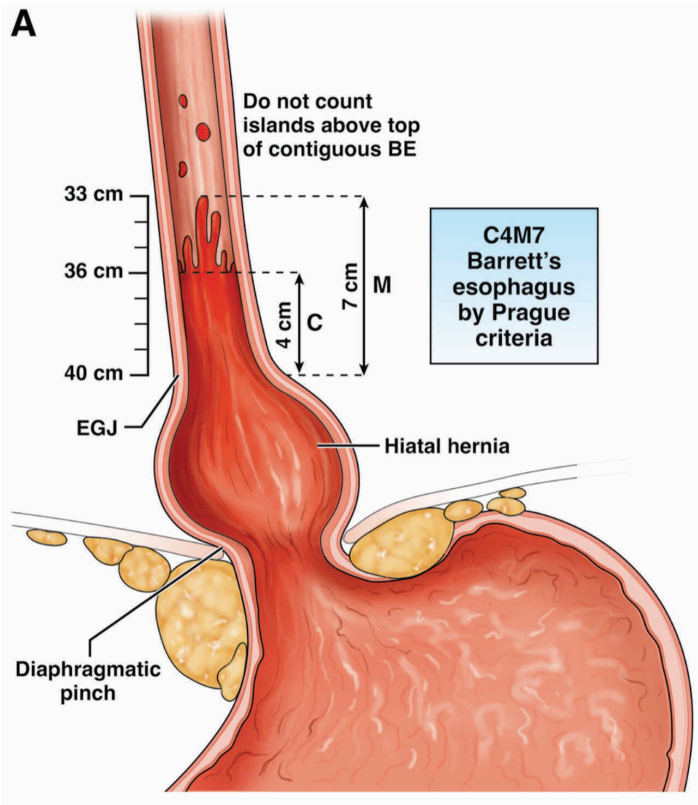

## 10 step approach to endoscopic exam of BE

| Approach | Rationale |
|---|---|
| Identify esophageal landmarks: diaphragmatic hiatus, GEJ, squamocolumnar junction | Critical for further examinations |
| Consider distal attachment cap (especially if prior dysplasia) | Facilitate visualization |
| Clean mucosa well (water jet and careful suction) | Remove mucus or debris and limit mucosal trauma |
| Use insufflation & deflation | Fine adjustments can help detect subtle changes |
| Spend adequate time inspecting the Barrett's segment & gastric cardia on retroflexion | Increases dysplasia detection |
| Examine with chromoendoscopy | Enhances mucosa pattern and surface vasculature |
| Use Prague classification- circumferential and maximal length | Standardized reporting system |
| Use Paris classification- describe superficial neoplasia | Standardized reporting system |
| Use Seattle protocol with partially deflated esophagus to sample Barrett's segment | Increases dysplasia detection |

**Quality indicators for screening and surveillance**

1. Documentation of landmarks + extent of BE
2. Not obtaining biopsies in the setting of normal-appearing junction
3. Sampling using Seattle protocol
4. Performing surveillance endoscopy in patients with NDBE no sooner than 3-5 years

# Hiatus Hernia and Hill Classification



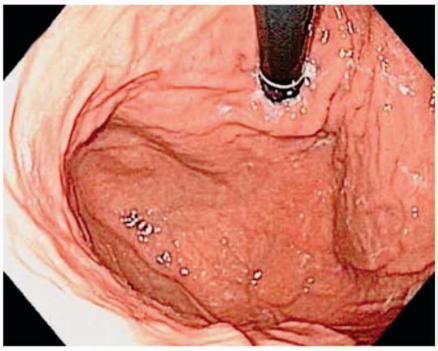



**Fig. 1** Hill Grade I: a prominent fold of tissue along the lesser curvature next to the endoscope.

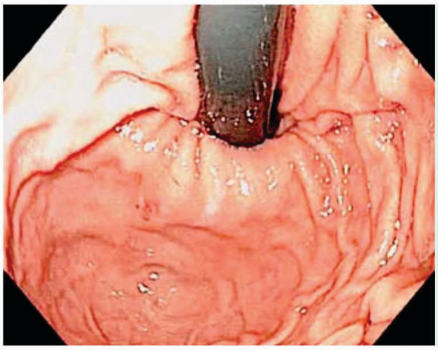


**Fig. 2** Hill Grade II: the fold is less prominent and there are periods of opening and rapid closing around the endoscope.

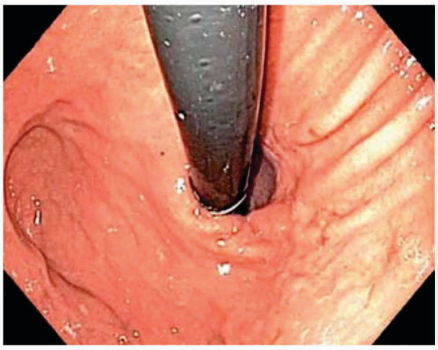


**Fig. 3** Hill Grade III: the fold is not prominent and the endoscope is not tightly gripped by the tissue.

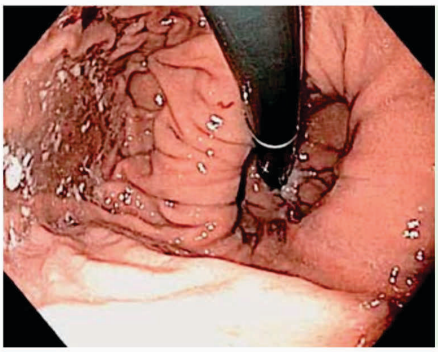
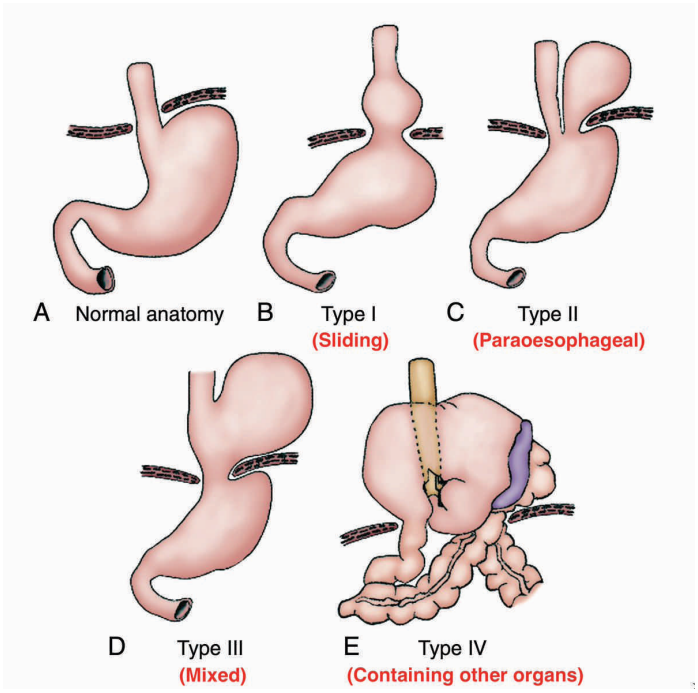
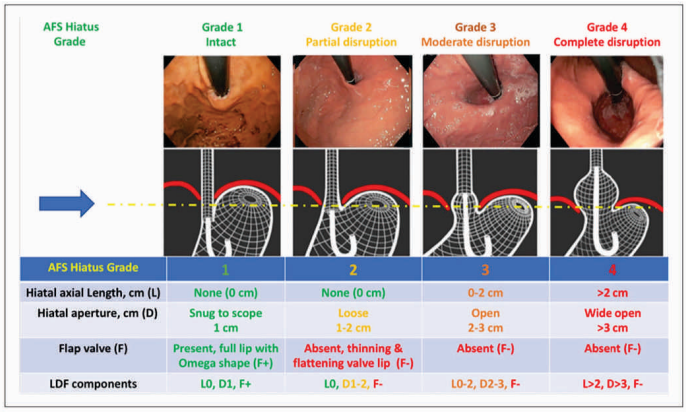


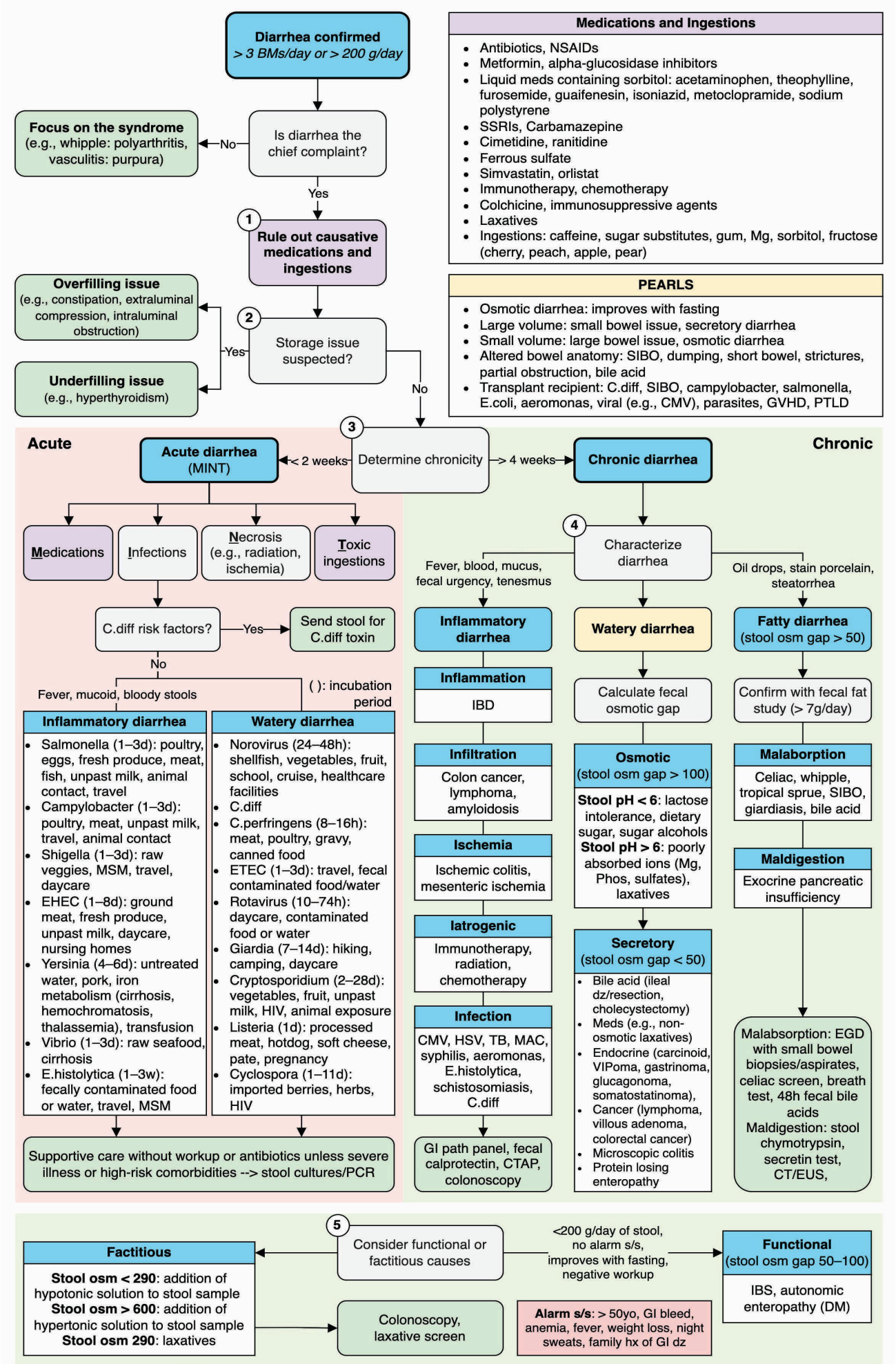
**Fig. 4** Hill Grade IV: there is no fold, and the lumen of the esophagus is open, often allowing the squamous epithelium to be viewed from below. A hiatal hernia is always present.

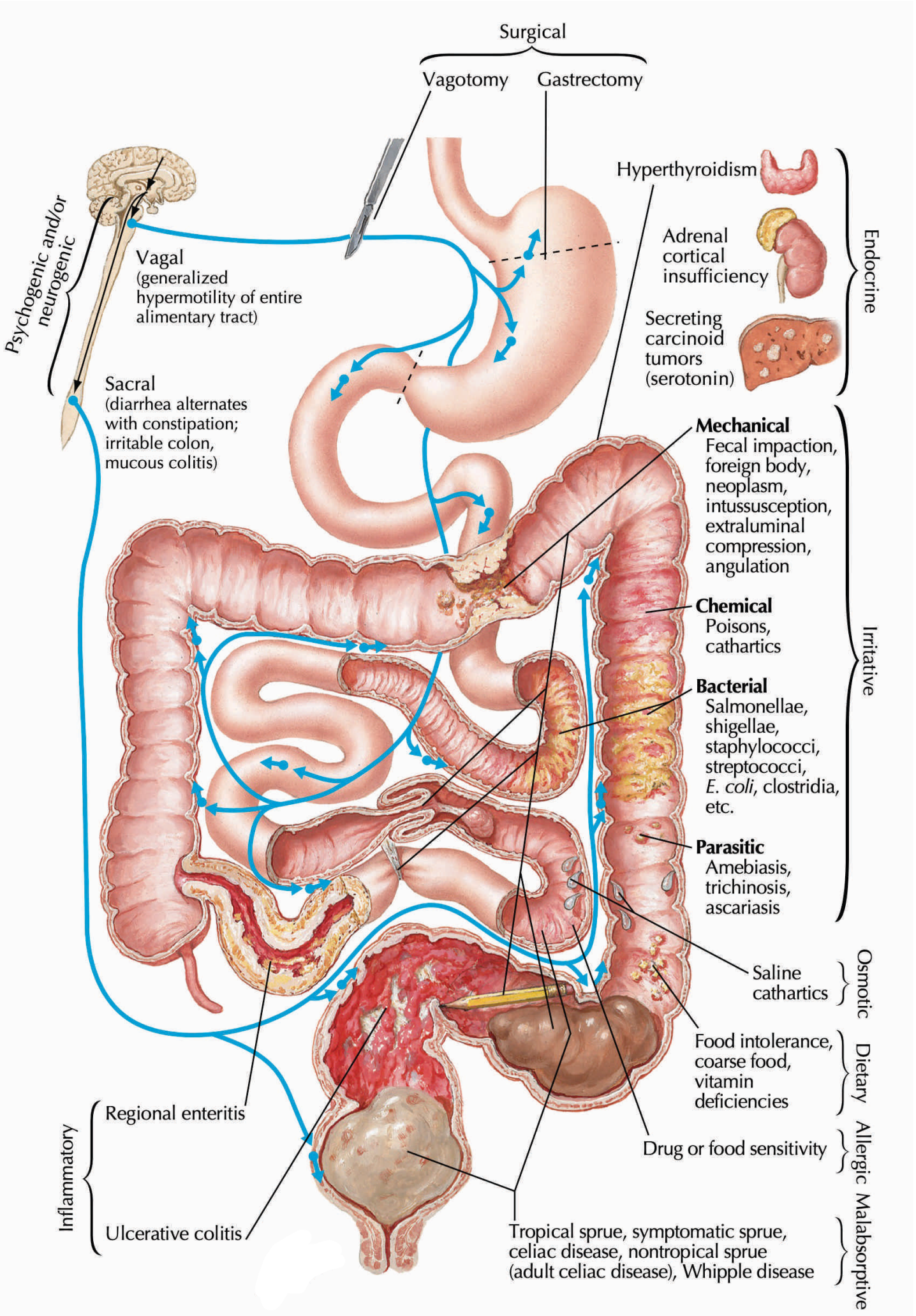


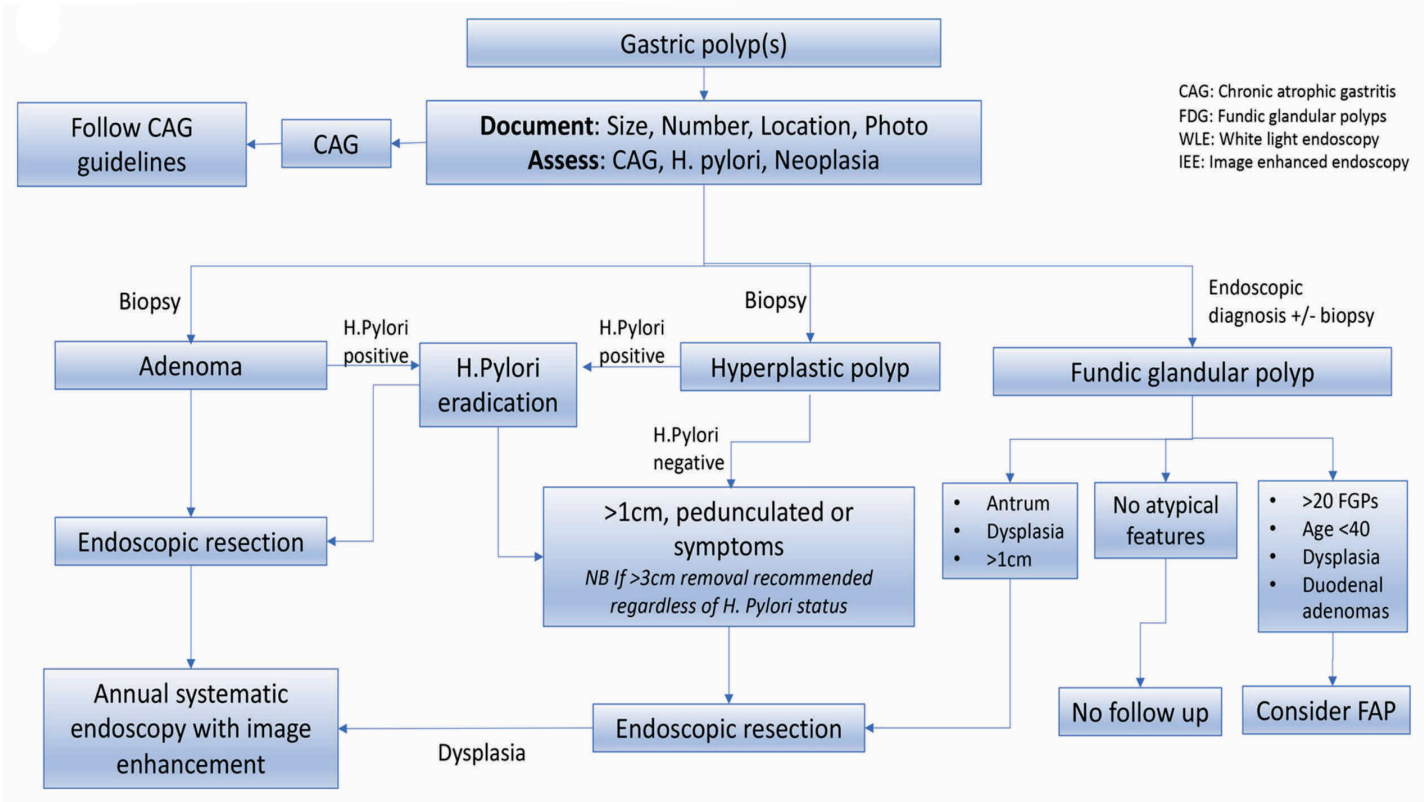

The AFS Endoscopic Classification of the Esophagogastric Junction Integrity as depicted by the LDF components (Length/Diameter/Flap valve). The arrow represents the relative level of the diaphragmatic hiatus starting with normal anatomy with a good segment of intraabdominal esophagus and an intact hiatus and progressing to increasing degree of hiatal disruption (hiatal axial length and widening of the crural defect) and loss of the flap valve.
*Note.* LDF components: hiatal axial length; hiatal aperture measured in centimeters; and the present or absent of a functioning flap valve.

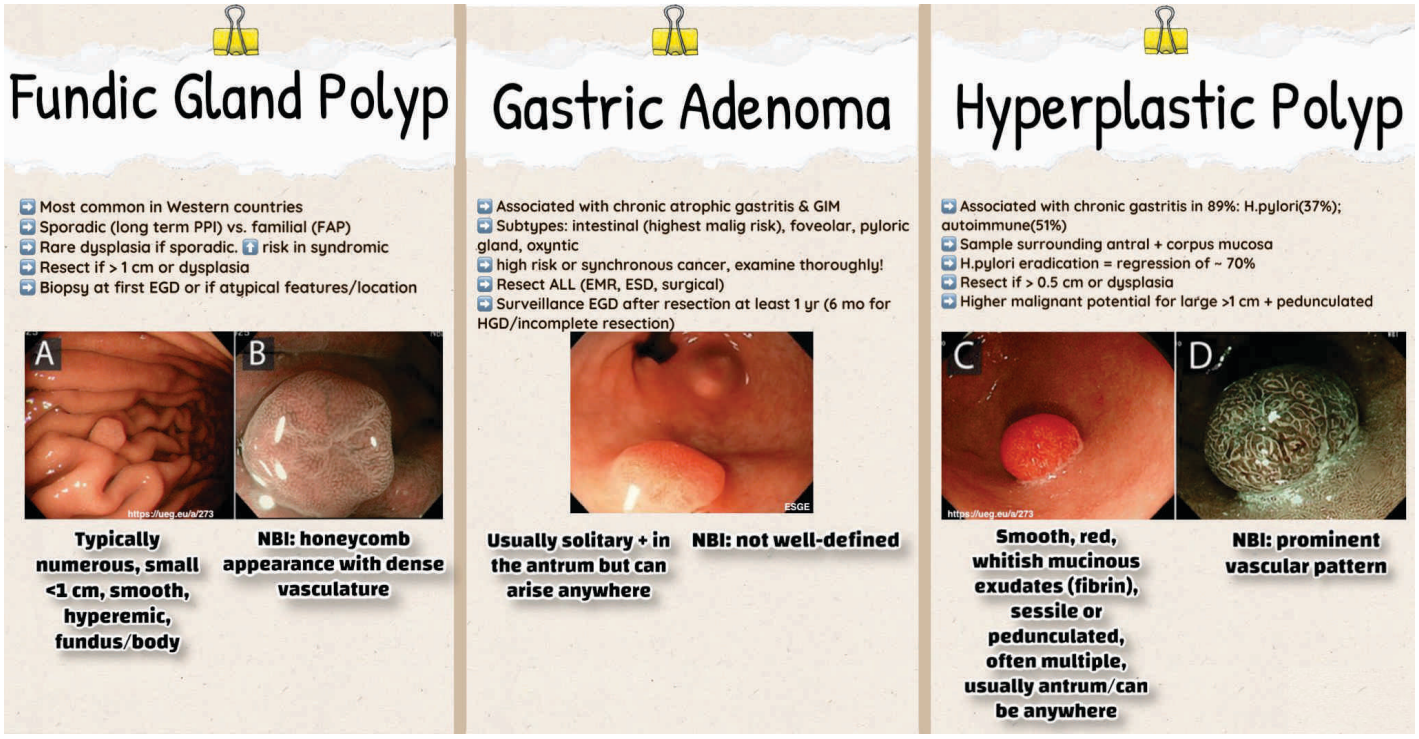
# Diarrhea Workup




Surgical
Vagotomy
Gastrectomy
Psychogenic and/or neurogenic
Vagal
(generalized hypermotility of entire alimentary tract)
Sacral
(diarrhea alternates with constipation; irritable colon, mucous colitis)
Hyperthyroidism
Adrenal cortical insufficiency
Secreting carcinoid tumors (serotonin)
Endocrine
Mechanical
Fecal impaction, foreign body, neoplasm, intussusception, extraluminal compression, angulation
Chemical
Poisons, cathartics
Bacterial
Salmonellae, shigellae, staphylococci, streptococci, E. coli, clostridia, etc.
Parasitic
Amebiasis, trichinosis, ascariasis
Irritative
Saline cathartics
Osmotic
Food intolerance, coarse food, vitamin deficiencies
Dietary
Drug or food sensitivity
Allergic
Tropical sprue, symptomatic sprue, celiac disease, nontropical sprue (adult celiac disease), Whipple disease
Malabsorptive
Regional enteritis
Ulcerative colitis
Inflammatory

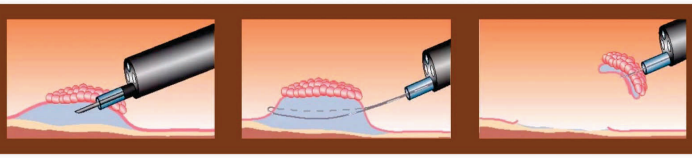
# Management of Gastric Polyps

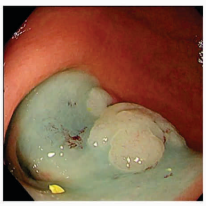


# Endoscopic Mucosal Resection (EMR)

Injection of solution into submucosal space to separate a mucosal lesion from muscularis propria followed by snare resection with electrosurgery





## Indication

Consider EMR for

- Lesions >10mm, with:
- No features of submucosal invasive cancer

## Goals of EMR

- ✓ Completely snare-resect lesion with minimum number of pieces
- ✓ Adequate margins to avoid recurrence
- ✓ Efficient and safe removal of colonic polyps

## Predictors of Poor Outcome

- Lesion >40mm
- Ileocecal valve location
- Prior resection attempt
- SMSA (size, morphology, site, access) level 4

## Review of Polypectomy Guidelines (ESGE, 2017)

**Intermediate**
**Size 10–19 mm**

Hot Snare Polypectomy (HSP)[4]

Submucosal injection prior to HSP should be considered to reduce the risk of deep thermal injury

**Large**
**Size ≥20 mm**

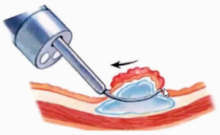
En bloc endoscopic mucosal resection (EMR) to achieve R0 resection[5]

Piecemeal EMR if en bloc not feasible or not safe

If lesion is sized >40 mm or complex[6] refer to expert center

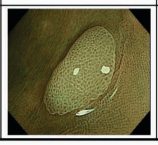
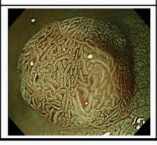
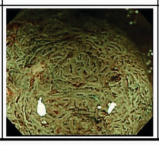
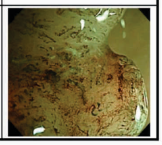
## Why Inject?

- Facilitate snare capture
- Protect the underlying muscle layer
- Aid in border delineation



| | Normal Saline | Gelofusin | Hyaluronic acid | Newer gels (Eleview/Orise) |
|---|---|---|---|---|
| Lift duration | + | ++ | +++ | +++ |
| Advantages | ✓ Cheap<br>✓ Availability<br>✓ Safe | ✓ Widely available<br>✓ Inexpensive<br>✓ Safe<br>✓ Fewer resections[1]<br>✓ Short procedure time[1] | ✓ En bloc resection [2]<br>✓ Non-toxic | ✓ Excellent lift<br>✓ Prefilled<br>✓ Less volume |
| Disadvantages | × Rapidly dissipates | Similar en bloc rates to NS[1] | × Expensive<br>× Limited availability<br>× Piecemeal | × Very expensive |

## Characterizing Lesions: JNET Classification

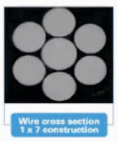
| | Type 1 | Type 2A | Type 2B | Type 3 |
|---|---|---|---|---|
| Vessel pattern | · Invisible ※1 | ·Regular caliber<br>·Regular distribution (meshed/spiral pattern) ※2 | ·Variable caliber<br>·Irregular distribution | ·Loose vessel areas<br>·Interruption of thick vessels |
| Surface pattern | ·Regular dark or white spots<br>·Similar to surrounding normal mucosa | ·Regular (tubular/branched/papillary) | ·Irregular or obscure | ·Amorphous areas |
| Most likely histology | Hyperplastic polyp/ Sessile serrated polyp | Low grade intramucosal neoplasia | High grade intramucosal neoplasia/ Shallow submucosal invasive cancer ※3 | Deep submucosal invasive cancer |
| Endoscopic image |  |  |  |  |

## Tips on Submucosal Injection

- Normal saline for en bloc excision of smaller polyps (<20mm)
- Viscous solutions for larger lesions
- Adjuncts: Adrenaline (stalked lesions), dye (border delineation and DMI identification), lidocaine (low rectal lesions)
- Dynamic injection technique to adjust the shape of the submucosal cushion for effective tissue resection

## How to Snare?

- Use dedicated snares
- Snare 1-2cm polyp pieces
- Work with 'natural polyp contour'
- Close snare slowly while deflecting scope tip down, pushing snare catheter slightly forward and aspiring air to pull polyp into snare
- If too much tissue in snare (feels hard – no compressibility), relax tension on the snare and then resnare




### Braided snares

- ❑ Shape memory
- ❑ Strength with flexibility
- ❑ Medium stiffness
- ❑ "good feel", low risk


## To Clip or Not to Clip After Colonic EMR?

*Lesions that benefit from prophylactic clipping*

- Delayed post-polypectomy bleeding
  - ≥20mm, proximal to splenic flexure
  - Complete defect closure reduces rate of bleeding
- Delayed perforation risk
  - Muscularis propria injury (Sydney 2 and above)

*Lesions that do not have evidence for prophylactic clipping*

- Distal to splenic flexure
- <20mm
- Piecemeal cold EMR

*Scenarios that may benefit rom clipping but lack evidence*

- Hot EMR <2cm or cold snare/EMR on anti-thrombotics
- Clinical judgement
  - Advanced age and multiple patient comorbidities
  - Lesions with difficult access
- Hemostatic agents may have a role, particularly in large defects that cannot be closed with clips

# Fluid Resuscitation in Acute Pancreatitis

**HYPOVOLEMIA CRITERIA**

**One criterion or more:**

A. Baseline creatinine >1.1 mg/dL or blood urea nitrogen (BUN) >20 mg/dL, equivalent to urea >43 mg/dL
B. Hematocrit >44%
C. Increase in creatinine and/or BUN and/or urea from the previous value
D. Urine output <0.75 mL/kg/h
E. Systolic blood pressure <90 mmHg without other explanation than hypovolemia
F. Signs and/or symptoms of dehydration (intense thirst, dehydrated oral mucosa, decreased skin turgor–skin pinch)

**Lactated Ringer Solution: LR**

Potassium 40 mEq/day was administered (LR has potassium 4 mEq/L)

After the #WATERFALLtrial

Hypovolemia

Yes

LR 10 ml/kg bolus over 2hr

No

LR infusion 1.5 ml/kg/hr

12h

Oral feeding if pain < 5 over 10 and the patient is willing to eat*

Checkpoints in the WATERFALL study: 12, 24, 48 and 72 hr

Volemia?

**Suspicion of fluid overload**

Decrease or stop fluid resuscitation, treat fluid overload

**Normovolemia**

LR infusion 1.5 ml/kg/hr

**Hypovolemia**

LR 10 ml/kg bolus over 2h, it can be repeated in case of SBP<90 mmHg or urine output <0.5ml/kg/hr

Oral feeding tolerated for 8 hr

Stop fluid resuscitation

LR infusion 1.5 ml/kg/hr

**FLUID OVERLOAD**

**Suspicion of fluid overload was defined by the presence of at least 1 of the following 3 criteria, definitive fluid overload at least 2:**

**Criterion 1. Hemodynamic-imaging evidence (at least one):**
A. Non-invasive diagnostic evidence of heart failure (i.e., echocardiographic)
B. Radiographic evidence of pulmonary congestion
C. Invasive cardiac catheterization suggesting evidence of heart failure [i.e., pulmonary capillary wedge pressure (or left ventricular end-diastolic pressure) >18 mmHg, right arterial pressure (or central venous pressure) >12 mmHg, or cardiac index <2.2 L/min per m2]

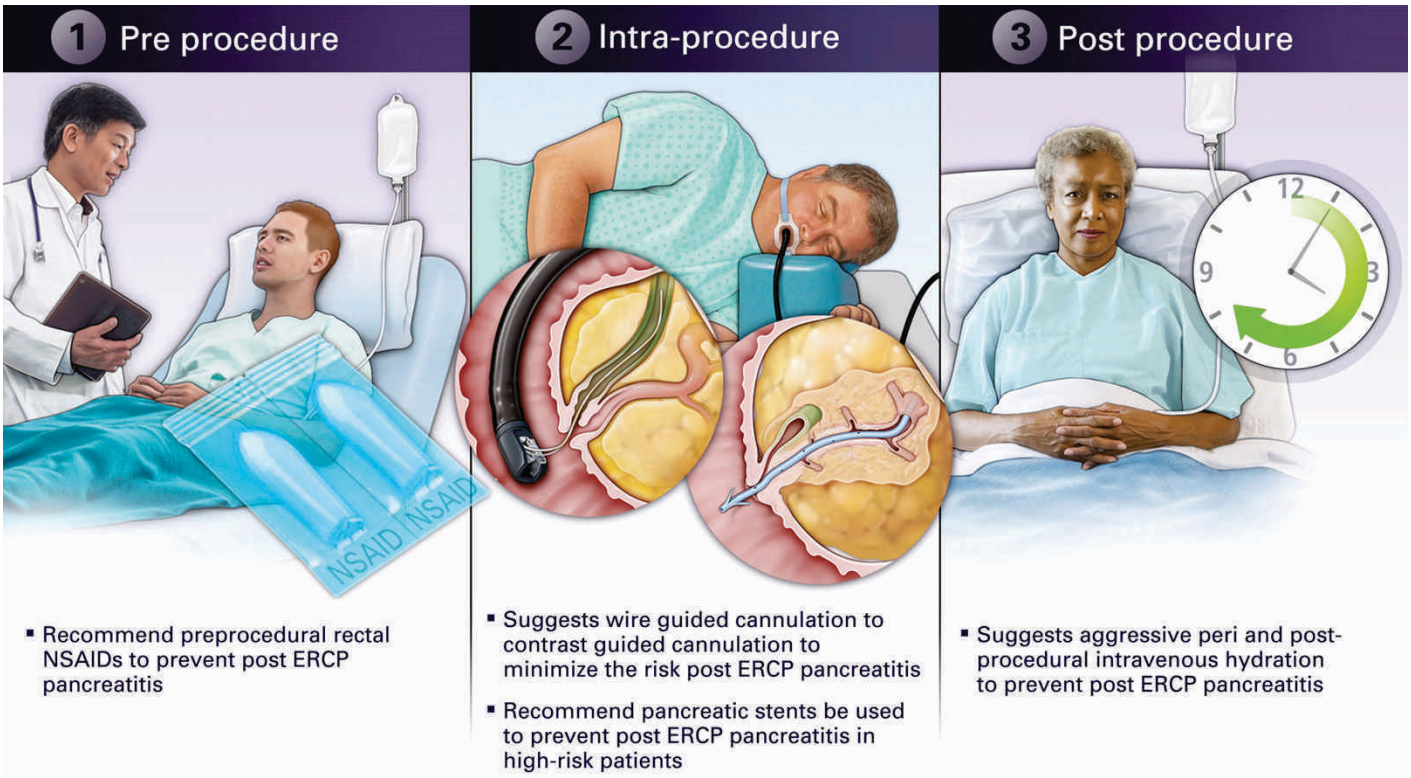
**Criterion 2. Heart failure symptoms:**
A. Dyspnea

**Criterion 3. Heart failure signs (at least one):**
A. Peripheral edema
B. Pulmonary rales
C. Increased jugular venous pressure, hepatojugular reflux, or both

**WATERFALL TRIAL (NEJM 2022):** https://www.nejm.org/doi/10.1056/NEJMoa2202884

* #WATERFALLtrial was not designed to study the best time to start oral feeding. Early oral feeding is encouraged, even <12h

# Prevention of Post ERCP Pancreatitis




- Recommend preprocedural rectal NSAIDs to prevent post ERCP pancreatitis

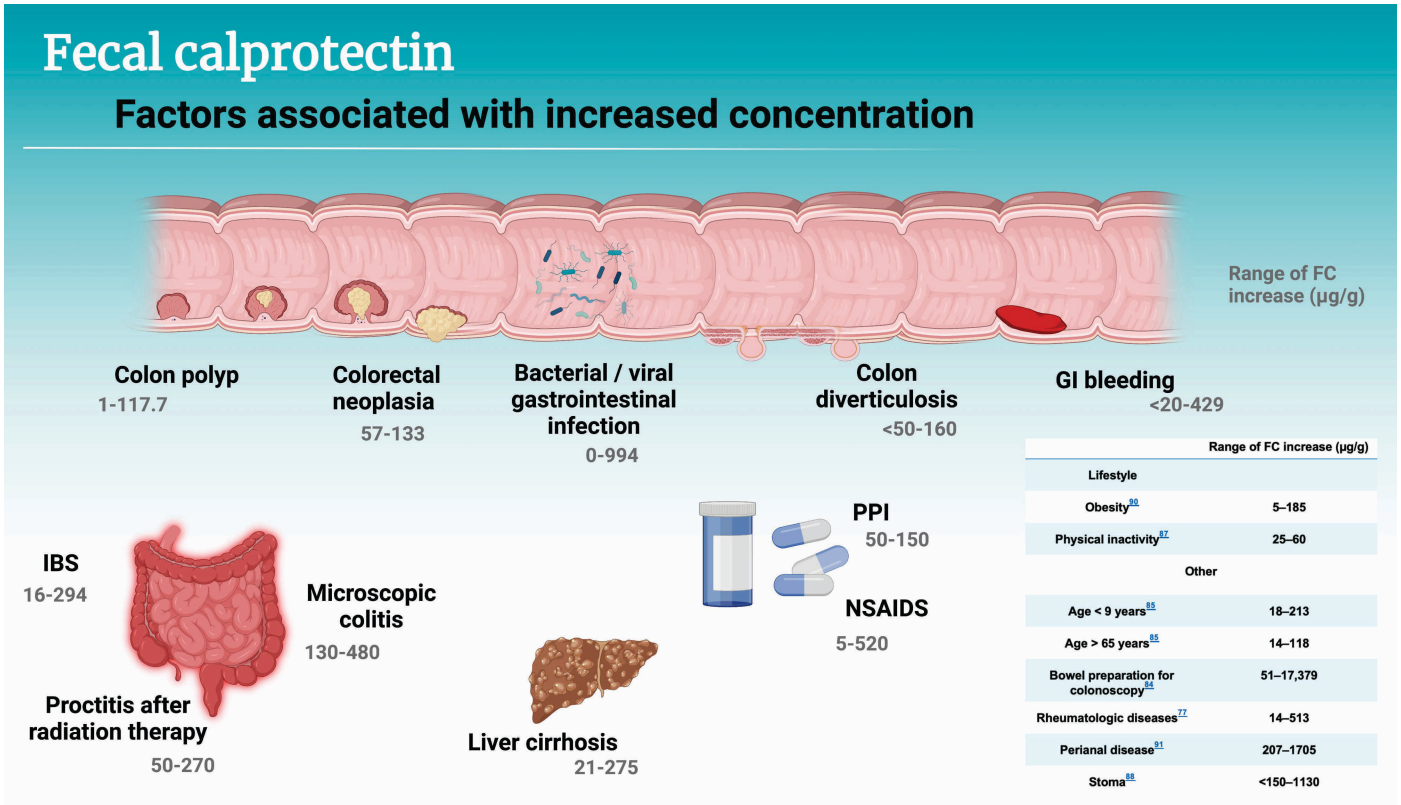
- Suggests wire guided cannulation to contrast guided cannulation to minimize the risk post ERCP pancreatitis
- Recommend pancreatic stents be used to prevent post ERCP pancreatitis in high-risk patients

- Suggests aggressive peri and post-procedural intravenous hydration to prevent post ERCP pancreatitis

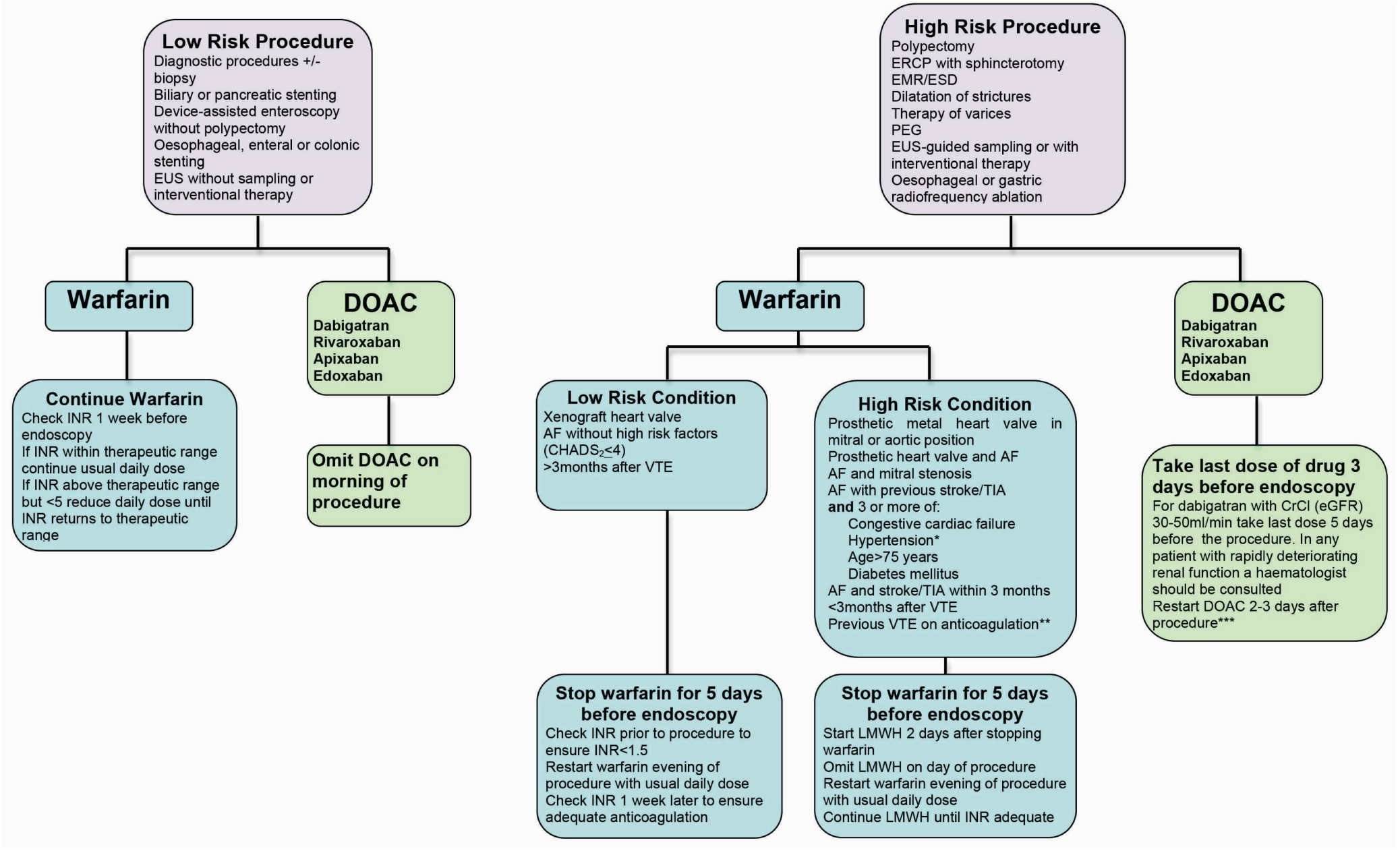
# CA 19.9 Elevation - Benign Causes

| Aetiology | Serum CA19.9 level | |
|---|---|---|
| | Mild elevation (<200 U/ml) | High levels (>1000 U/ml) |
| Obstructive jaundice[2 3] | | + |
| Acute liver failure and acute hepatitis[4 5] | | + |
| Chronic liver disease[4] | + | |
| Alcoholic liver disease[4–6] | + | + |
| Non-alcoholic liver disease[4 5] | + | |
| Pancreatitis | | |
| Acute[8 9] | + | + |
| Chronic[8 9] | + | |
| Diabetes mellitus[10] | + | |
| Interstitial pulmonary disease[11] | + | |
| Collagen vascular diseases[12] | + | |

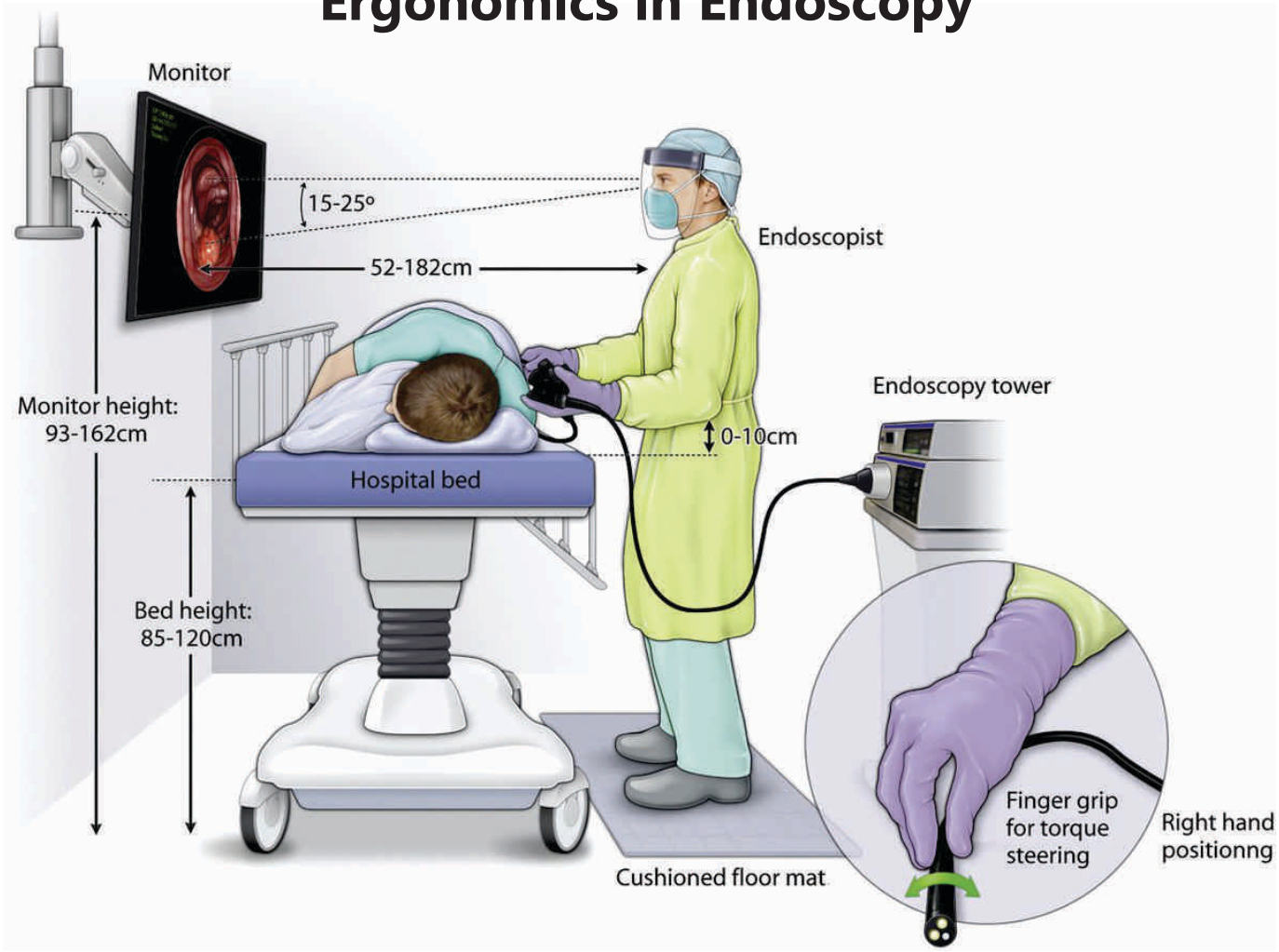
Anecdotally reports: hydronephrosis,[13] endometriosis,[14] splenic cyst,[15] bronchogenic cyst,[16] sigmoid diverticulitis,[17] and hypothyroidism.[18]

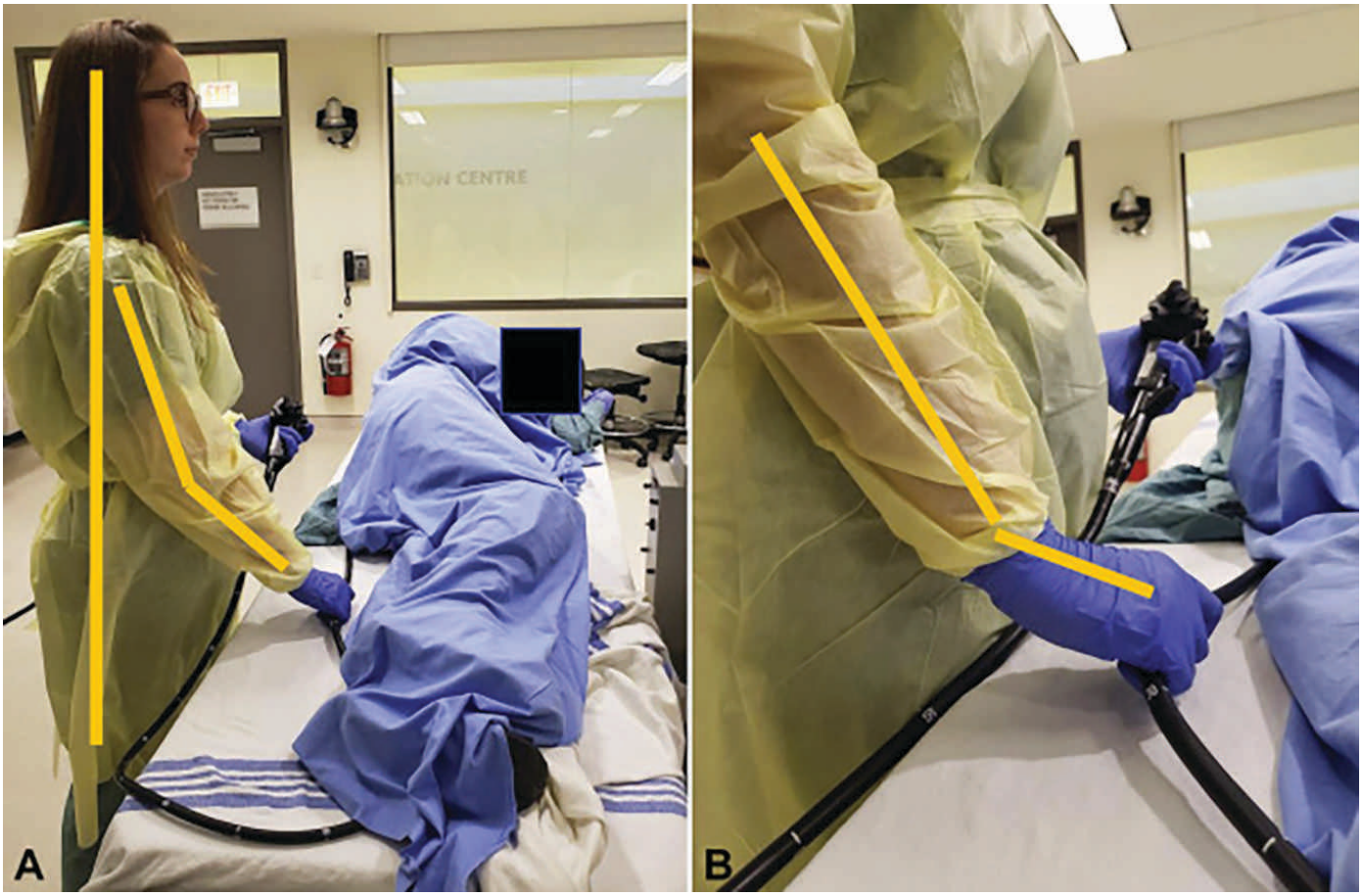


# Management of Anticoagulation in Endoscopy



# Ergonomics in Endoscopy



# Frequently Asked Questions (FAQs) related to bowel and liver diseases in Sri Lanka

**"Why does everyone have 'gastritis'?"**

Symptoms of pain or 'burning sensation' in the upper abdomen or abdominal fullness after meals or early satiation are expected in up to 30-50 % of the population worldwide. However, these are common symptoms and do not necessarily mean an upper gastrointestinal endoscopy (UGIE) is needed.

However, patients who also have 'sinister' symptoms of persistent loss of appetite, unintentional weight loss, difficulty swallowing food, recurrent vomiting, evidence of blood loss or anaemia etc., need UGIE to look for an underlying sinister cause.

Alternatively, stress or even depression may present with similar 'gastritis' symptoms and should be addressed accordingly and may not resolve entirely within addressing these psychological issues.

**"Screening for large bowel carcinoma? "**

Colorectal or large bowel carcinoma (CRC) is the third most common cancer worldwide.

CRC start as a polyp (small fleshy growth in the colon). If detected early and removed, polyps can be prevented from becoming CRC.

Some countries screen asymptomatic (those without symptoms) adults (45-75 years) by testing the stools for blood (stool for occult blood on three consecutive days) to detect patients who are more likely to have colonic polyps or early CRC. These patients with positive stool occult blood are invited for screening colonoscopy to detect and treat polyps.

The presence of persistent loss of appetite, unintentional weight loss coupled with a change in bowel habits or passage of blood in their stools may be suggestive of CRC. Therefore, if someone has these symptoms, they should be investigated with a colonoscopy, especially as one grows older.

**"What's fatty liver disease, and what does it mean if found in my scan."**

Foods rich in calories, unhealthy fats and sugars are becoming more common in Sri Lanka. More people are adopting a sedentary lifestyle with reduced day-to-day physical activity. When we consume more calories than we use in our daily activities, our bodies save them up and store them as fat. Not only do we gain weight and become fatter, but the excess fat also gets deposited in our livers, which is called 'non-alcoholic fatty liver disease.

Fatty liver disease may be detected incidentally when an abdominal scan is done for another reason or on routine blood tests with elevated liver enzymes.

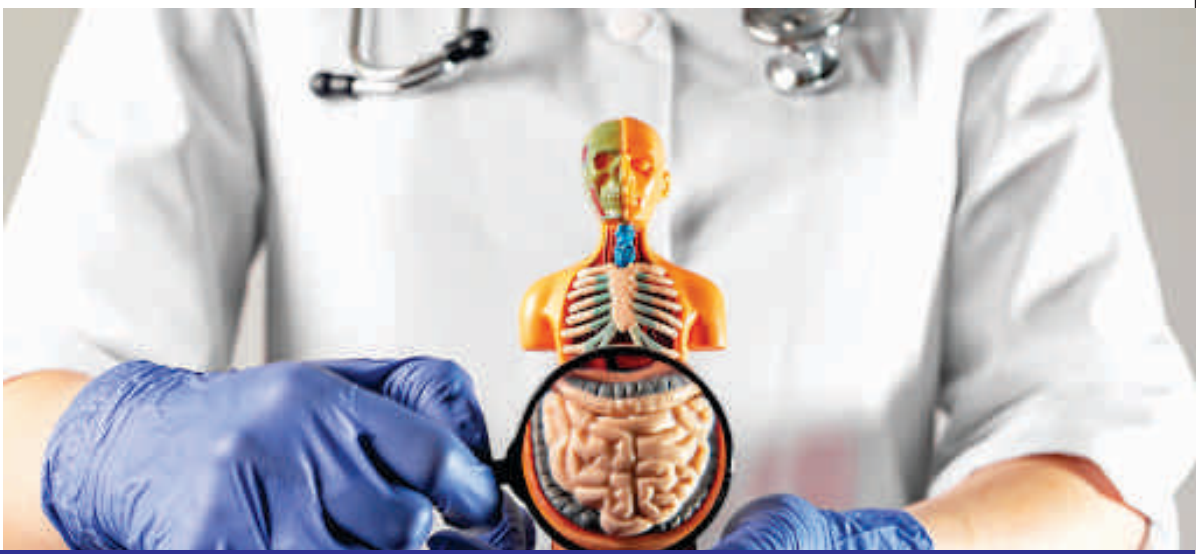
Usually, the fatty liver disease does not cause any symptoms. However, fatty liver disease is commonly associated with obesity, diabetes, high blood pressure and abnormal blood lipids. Therefore, if not detected and left untreated, fatty liver disease can lead to cardiovascular complications (heart attacks and stroke), liver cirrhosis, and liver cancer.

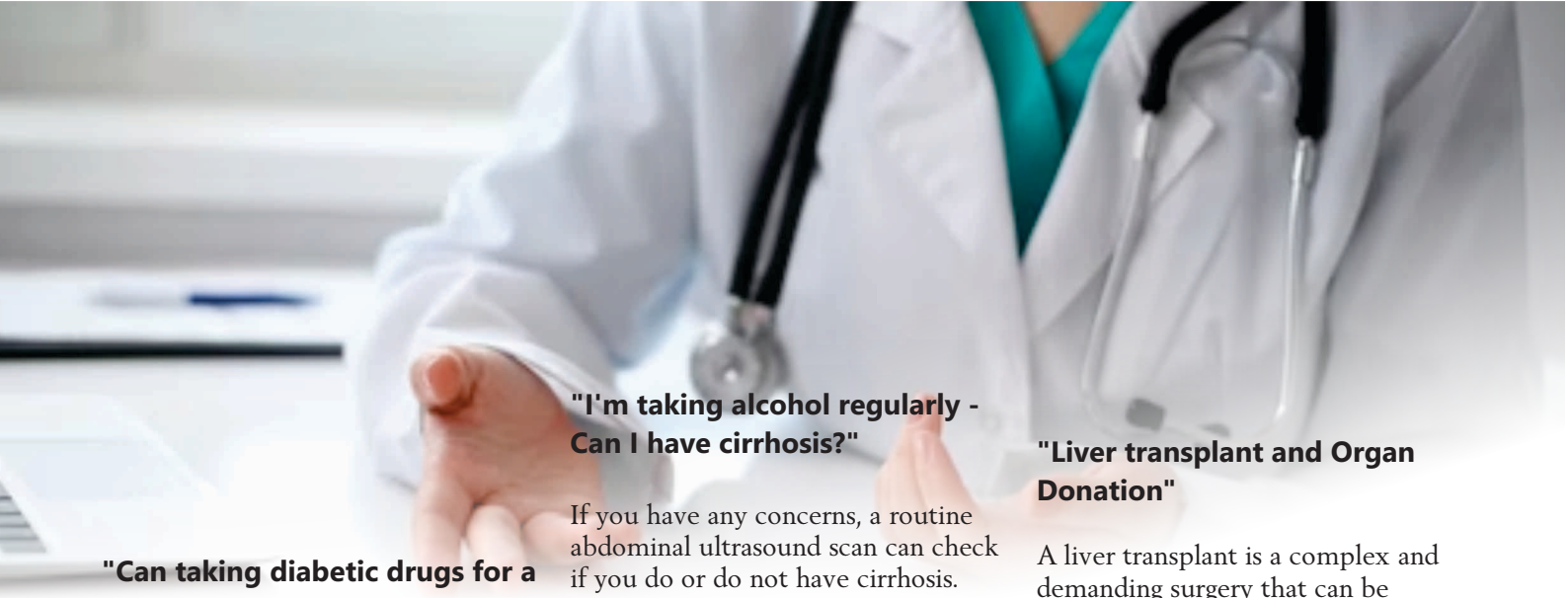
**"Do all patients with fatty liver lead to cirrhosis long term?"**

Not everyone who has fatty liver will progress to develop cirrhosis. The majority will not move to cirrhosis. However, a minority may progress to cirrhosis slowly, usually over decades.

The risk of progression to cirrhosis in higher among those with liver fibrosis (scarring of the liver). This risk may be greater with age, obesity or increased weight, poorly controlled diabetes, hypertension, high cholesterol and a family history of cirrhosis and associated alcohol consumption.

Controlling these factors, losing weight through regular physical exercise and maintaining a diet low in calories long-term can reduce or prevent someone with fatty liver from progressing to cirrhosis.





**"Can taking diabetic drugs for a long period cause cirrhosis?"**

No, this is a myth. Drugs prescribed for diabetes, hypertension or high cholesterol never cause cirrhosis. It is quite the opposite. These drugs, by helping to control the disease, hypertension or high cholesterol, will help prevent the development of cardiovascular complications (heart attacks and stroke) related to fatty liver disease.

**"What is cirrhosis of the liver?"**

Cirrhosis is a common term referring to permanent liver damage in the presence of long-term liver injury. However, the extent of the damage and manifestations is variable. Those with early cirrhosis may not have symptoms or complications for a long time. Those who have advanced cirrhosis have a worse outcome and ideally need a liver transplant.

**"Is cirrhosis always due to alcohol?"**

Although long-term, unsafe alcohol intake may lead to cirrhosis, over half of the patients with cirrhosis in Sri Lanka have "non-alcoholic" cirrhosis. These patients have either not consumed any alcohol or consumed only occasional safe amounts. Many of these patients have a long history of obesity, diabetes, hypertension and high cholesterol and likely have cirrhosis related to non-alcoholic fatty liver disease. In addition, rare genetic conditions, autoimmune conditions of the liver and chronic viral hepatitis (hepatitis B & C) may also cause cirrhosis, so cirrhosis is not always due to alcohol!

**"I'm taking alcohol regularly - Can I have cirrhosis?"**

If you have any concerns, a routine abdominal ultrasound scan can check if you do or do not have cirrhosis. Otherwise, you can even do a simple blood test - complete blood count (CBC) and liver enzymes (ALT and AST) so the doctor can calculate the risk of having significant liver fibrosis (early cirrhosis). Those with abnormal values on this calculation (FIB-4 score) will need a special liver scan (FibroScan) to detect and estimate liver stiffness. A FibroScan accurately "rule in" or "rule out" significant liver fibrosis and see those with advanced liver fibrosis or early cirrhosis.

**"If someone has cirrhosis, do they have to abstain from alcohol completely?"**

If you or a loved one has cirrhosis, it is essential that they completely stop taking any alcohol. In cirrhosis, any alcohol consumption quickly causes liver disease progression.

**"Do all patients with cirrhosis need liver transplants?"**

Patients with early cirrhosis do not need a transplant. However, there is a tendency for liver fibrosis to progress gradually over a while. It is difficult to predict this; in some patients, the disease may remain very stable for a long time.

However, suppose the liver damage has progressed to advances stages with the development of symptoms (yellow discoloration of eyes, swelling of the abdominal and ankles) complications of cirrhosis (vomiting of blood, passage of dark tarry stools, excessive drowsiness, confusion or episodes of unconsciousness or early liver cancer). In that case, a liver transplant is the only treatment that will cure the disease.

**"Liver transplant and Organ Donation"**

A liver transplant is a complex and demanding surgery that can be selectively offered to suitable patients. After successful transplantation, such selected patients can live a 'near normal' life.

There are two sources of organs for transplantation. The liver can be received from a brain death donor such as those after a massive brain injury after a road traffic collision, where there is absolutely no possibility of recovery, but where in such situations the patient was connected to a breathing machine or "ventilator" and as a result, their organs such as their liver, were in good condition. Donation of such organs by the brain-dead person's family can potentially save the life of a patient who is waiting for transplantation and has no hope of otherwise surviving. Worldwide there is a huge shortage of such organs as there will usually be more patients who need a transplant, and only a few organs available for transplant. Hence, good public awareness to help promote organ donation is vital for a country to have a successful transplant program.

Alternatively, part of the liver can be donated from a living donor as around two-thirds of a healthy liver can be donated, and the remaining liver can "grow back" relatively soon. This is commonly practised in pediatric patients with cirrhosis, where their mother or father may donate part of their liver. Donations of such organs by living donors should always be made purely with altruistic intentions without any financial interests etc.

**Dr. Mananjala Senanayake**

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