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Chapter 1

Preoperative Evaluation and Management of Patients with Liver Disease

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Additional information is available at the end of the chapter

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Abstract

Patients with liver disease who undergo surgery have an increased risk of morbidity and mortality. Impairment of the liver functions increases the risks of surgery and anesthesia.The risk depends on the severity of liver disease, nature of the surgery and comorbid conditions. Patients with compensated cirrhosis and normal synthetic function have a low risk. Elective surgery should be postponed in patients with abnormal liver tests. All patients should have thorough preoperative evaluation, and their conditions are to be optimized before elective surgery. Thorough history and physical examination usually provide important informatation. Elective surgery can be rescheduled or cancelled once the severity of underlying liver disease is assessed. When surgery is mandatory, meticulous perioperative management is required, including hemodynamic stability, broad-spectrum antibiotics, correction of coagulopathy, improvement of nutritional status, avoidance of nephrotoxins and sedatives that could precipitate hepatic encephalopathy, and intensive care unit admission if needed.

Keywords: Liver, Liver failure, liver surgery, Liver tests, Liver functions, preoperative preparation

1. Introduction

Patients with liver disease who undergo surgery have an increased risk of morbidity and mortality [1-3].

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The optimal management of such patients requires the following:

1. Diagnosis of the underlying liver disease
2. Assessment and stratification of the risk of surgery
3. Estimation of functional hepatic reserve
4. Correction of underlying conditions if feasible
5. Hepatic hemodynamic evaluation and identification of the site of upper gastrointestinal hemorrhage, if present

Impairment of the liver functions increases the risks of surgery and anesthesia in several ways, including the following [4-6]:

1. Bleeding risk may increase because of coagulopathy
2. Susceptibility to infection is increased due to altered functions of the hepatic reticuloendothelial cells and changes in the immune system and portal hypertension
3. Reduced hepatic blood flow
4. Altered drug metabolism

1.1. Factors contributing decreased liver blood flow and hypoxia [7, 8]

1. Hyperdynamic circulation with increased cardiac output and decreased systemic vascular resistance
2. Systemic and splanchnic vasodilation, with subsequent activation of the sympathetic nervous system and neurohormonal axis in an attempt to maintain arterial perfusion pressure
3. Alterations in the systemic circulation due to arteriovenous shunting and reduced splanchnic inflow
4. Anesthetic agents may reduce hepatic blood flow
5. The compensatory inotropic and chronotropic response to pharmacologic and physiologic stress, including surgery, is blunted

Induction of anesthesia hypotension, intermittent positive-pressure ventilation, pneumoperitoneum during laparoscopic surgery, traction on abdominal viscera, hemorrhage, hypoxemia, vasoactive drugs, surgical maneuver, and even positioning of the patient may all result in intraoperative and perioperative hepatic hypoxemia and further increase in the hepatic dysfunction. Risk factors for hepatic hypoxemia include ascites, hepatic hydrothorax, and hepatopulmonary syndrome [7, 17]

1.2. Altered drug metabolism

The duration of action of many drugs can be increased as a result of [10]

1. altered metabolism by cytochrome P450 enzymes,

2. decreased concentration of plasma-binding proteins,

3. decreased biliary excretion.

Hepatic dysfunction can significantly impair the metabolism of certain medications. Examples are as follows [10, 11]:

1. The volume of distribution of nondepolarizing muscle relaxants is increased, and larger doses may be required to achieve adequate neuromuscular block.

2. Sedatives, narcotics, and intravenous induction agents must be used with caution, as they may result in prolonged depression of consciousness and may lead to hepatic encephalopathy. The perioperative use of opioids as morphine should be avoided, as their bioavailability is increased.

3. Benzodiazepines should be avoided, and, if necessary, remifentanil and oxazepam are the preferred narcotic and sedative because their metabolism is not affected by liver disease.

4. Isoflurane is the recommended volatile anesthetic because it does not impair hepatic blood flow and undergoes the least amount of hepatic metabolism.

2. Preoperative assessment

If liver dysfunction is suspected, elective surgery should be deferred until extensive evaluation is made. Evaluation will include the following items [12].

2.1. History and physical examination [1-3, 13]

Thorough history and physical examination usually provide important informatation.

1. History of previous blood transfusions, drug abuse, or excessive alcohol intake.

2. Family history of jaundice, anemia, hereditary liver disease, and prior adverse reactions to anesthesia.

3. Medication history includes the use of analgesics and alternative medications.

4. Physical examination may identify signs of underlying liver disease, as temporal wasting, jaundice, palmar erythema, spider nevi, ascites, or hepatosplenomegaly.

2.2. Laboratory tests [1-18])

The term "liver function tests" is a misnomer and can be misleading. Because of the complexity of liver functions, the ideal liver function test has not been invented yet. A successful liver function test, to assist with preoperative assessment of liver function, should be safe, reproducible, and easily performed.

The aims of the tests are as follows:

1. To determine the presence or absence of hepatic injury
2. To decide whether the injury is cell necrosis or cholestasis
3. To specify the particular disease
4. To determine its severity
    - Markers of hepatocellular injury include aminotransferases and lactate dehydrogenase
    - Markers of cholestasis include alkaline phosphatase, gamma glutamyl transpeptidase, 5′-nucleotidase, and bilirubin
    - Markers of synthetic functions of the liver are prothrombin time and albumin

2.3. Aminotransferases

Alanine aminotransferase (ALT) (normal range: 10-55 U/L)

Aspartate aminotransferase (AST) (normal range: 10-40 U/L)

- Serum level rises as a result of leakage from damaged tissue
- Mild to moderate elevations occur in many types of liver disease
- Marked elevations occur in hepatitis (viral, toxic, autoimmune, and ischemic)
- AST/ALT >2 suggests alcoholic liver disease or cirrhosis of any etiology
- ALT is more specific than AST for hepatic injury
- AST is nonspecific and can originate from skeletal muscle, red blood cell, kidney, pancreas, brain, and myocardium

2.4. Alkaline phosphatase (AP)

- Normal range 45-115 U/L
- Serum level rises as a result of increased production and leaks into the serum
- Moderate rises occur in many liver diseases
- Marked rises occur in extra- and intrahepatic cholestasis, diffuse infiltrating disease (e.g., liver neoplasms), and rarely alcoholic cirrhosis
- Considerable rises occur in bone diseases (e.g., tumor, fracture, Paget's disease)
- It also originates from the intestine, placenta, and some neoplasms

2.5. Gamma Glutamyl Transpeptidase (GGTP)

- Normal range: 0-30 U/L
- Serum level rises as a result of overproduction and leakage into serum, as for AP; induced by ethanol and drugs

- GGTP/AP >2.5 suggests alcoholic liver disease
- Kidney, spleen, pancreas, heart, lung, and brain are other sources

2.6. 5′-Nucleotidase

- Normal range:: 0-11 U/L
- Serum level rises as a result of overproduction and leakage into serum, as for AP
- Found in many tissues, but serum elevation is relatively specific for liver disease

2.7. Bilirubin

- Normal range::0.0-1.0 mg/dL
- Unconjugated hyperbilirubinemia

The mechanisms that result in elevation of serum unconjugated bilirubin levels include increased production (increased breakdown of hemoglobin (resulting from hemolysis, disordered erythropoiesis, and resorption of hematoma) or myoglobin (resulting from muscle injury)) and defects in hepatic uptake or conjugation.

- Conjugated hyperbilirubinemia

The mechanisms that result in the elevation of serum-conjugated bilirubin levels are hepatobiliary diseases, including extrahepatic and intrahepatic bile duct obstruction, viral, alcoholic or drug-induced hepatitis, and inherited hyperbilirubinemia.

2.8. Prothrombin Time (PT) (10.9-12.5 s), International Normalized Ratio [INR]: (0.9-1.2)

- All clotting factors except factor VIII are synthesized by hepatocytes; factor VIII is produced by vascular endothelium and reticuloendothelial cells.
- Serum values rise as a result of the following:
  1. Decreased synthetic capacity as in acute or chronic liver failure (prolonged PT unresponsive to vitamin K)
  2. Biliary obstruction (prolonged PT usually responsive to vitamin K administration)
  3. Vitamin K deficiency (secondary to malabsorption, malnutrition, and antibiotics) and consumptive coagulopathy

2.9. Albumin

- Normal range: 3.5-5.0 g/dL
- Serum level decreases as a result of decreased synthesis; or increased loss as in
  1. chronic liver failure
  2. nephrotic syndrome, protein-losing enteropathy, and vascular leak

2.10. Markers of viral hepatitis

2.10.1. Hepatitis A

Acute infection is confirmed by detection of IgM anti-hepatitis A antibody (IgM HAV), which appears early in the course of infection and has high sensitivity and specificity. IgG anti-HAV predominates in convalescence and persists throughout life.

2.10.2. Hepatitis B

- Acute infection is associated with the presence of hepatitis B surface antigen (HBsAg).
- Detection of HBsAg precedes serum aminotransferase elevations.
- HBsAg becomes undetectable 1-3 months after jaundice.
- Some time after HBsAg disappears; HBsAg antibody (anti-HBs) appears and persists for life.
- In the interval between disappearance of HBsAg and appearance of anti-HBs, hepatitis core antigen antibody (anti-HBc) is present and helps as a marker for current or recent HBV infection.
- Anti-HBc may remain for years after infection longer than anti-HBs.
- IgM anti-HBc distinguishes recent from remote infection

2.10.3. Hepatitis C

- Hepatitis C antibodies are detected relatively late in the course of the HCV infection.
- False-positive test is a problem.
- Reverse transcriptase polymerase chain reaction and branched amplification assays are the most sensitive and specific.

2.11. Liver function quantitative tests

These tests offer attractive means to assess the liver functions. However, they have limitations, including expense, availability, invasiveness, and lack of validity.

2.11.1. Indocyanine green clearance

This dye is taken up almost exclusively by hepatocytes and excreted unchanged into the bile. It is measured photometrically in blood samples taken at regular intervals after a bolus intravenous injection (0.5 mg/kg). Clearance of the dye decreases with loss of hepatocyte mass.

2.11.2. Aminopyrine breath test

Radioactivity ($14CO_2$) is measured in breath at 15-min intervals for 2 h after oral or intravenous administration of 14C-labeled methyl aminopyrine. It may predict death and histology in chronic hepatitis.

2.11.3. Monoethylglycinexylidide (MEGX)

This lidocaine metabolite is measured in blood samples 15 min after intravenous administration of lidocaine (1 mg/kg). It may predict death and complications before and after liver transplantation

2.12. Ultrasound

Ultrasound is useful for assessing liver size, spleen size, intra- and extrahepatic biliary tree, and the presence of liver masses. It can also detect ascites in its earliest stages (≥100 mL). Doppler ultrasonography is helpful in assessment of portal venous patency, direction of portal flow.

3. Risk estimation [10-30]

The risk of surgery in patients with impairment of liver functions depends on the severity of liver disease, nature of the surgery, and comorbid conditions. Patients with compensated cirrhosis and normal synthetic function have a low risk. The risk increases for patients with decompensated liver cirrhosis. Patients with advanced liver disease may benefit from non-surgical therapy when appropriate.

3.1. Contraindications to elective surgery

1. **Acute hepatitis:** Patients with acute hepatitis of any cause have an increased operative risk.
2. **Alcoholic hepatitis:** Alcoholic hepatitis greatly increases perioperative mortality.
3. **Acute liver failure:** For acute liver failure (the development of jaundice, coagulopathy, and hepatic encephalopathy within 2-6 weeks without preexisting liver disease), all surgery other than liver transplantation is contraindicated.
4. **Decompensated cirrhosis.** Elective surgery is contraindicated in patients with Child's class C cirrhosis; these patients should be considered for surgery only in life-threatening situations, such as an incarcerated hernia, gangrenous cholecystitis, or bowel infarction.

Elective surgery should be postponed in patients with abnormal liver tests. All patients should have thorough preoperative evaluation, and their conditions are to be optimized before elective surgery. Elective surgery can be rescheduled or cancelled once the severity of underlying liver disease is assessed.

When surgery is mandatory, meticulous perioperative management is required, including hemodynamic stability, broad-spectrum antibiotics, correction of coagulopathy, improvement of nutritional status, avoidance of nephrotoxins and sedatives that could precipitate hepatic encephalopathy, and intensive care unit admission if needed.

4. Assessment of the risk factors

4.1. Severity and nature of the underlying liver disease

Operative risks are markedly influenced by the severity and nature of the underlying liver disease.

Obstructive jaundice: Obstructive jaundice markedly increases perioperative mortality.

Acute hepatitis: Acute hepatitis is associated with increased morbidity and mortality associated with surgery.

Cirrhosis: The perioperative risk is influenced by the degree of hepatic dysfunction, portal hypertension, and its complications as ascites, intra-abdominal varices, renal impairment, and portopulmonary hypertension.

The amount of perioperative risks is related to the degree of liver decompensation. An accurate assessment of the degree of liver decompensation is important for determination of the perioperative risk.

4.2. Child's classification and its modifications

This is based on the patient's serum bilirubin and albumin levels, prothrombin time, and severity of encephalopathy and ascites.

Child–Pugh scoring system

Points	**1**	**2**	**3**
Ascites	None	Small or diuretic controlled	Tense
Encephalopathy	Absent	States I–II	States III–IV
Albumin (g/L)	>3.5	2.8–3.5	<2.8
Bilirubin (mg/dL)	<2	2–3	>3
PT(sec above control), or INR	<4 <1.7	4–6 1.7–2.3	>6 >2.3

In general, elective surgery is well tolerated in patients with Child class A, permitted with careful preoperative preparation in patients with Child class B, and contraindicated in patients with Child class C.

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Score	Child–Turcotte-Pugh Class
5–6	A
7–9	B
10–15	C

Other factors can also increase the perioperative risk beyond the Child classification. The perioperative risk is increased if there is portal hypertension. Emergency surgery is associated with a higher mortality rates.

Child score for estimating the perioperative risks has been shown to be quite variable. This may be explained by the following:

1. Patients with class A may have ascites, hyperbilirubinemia, and portal hypertension.
2. The variables (ascites and hepatic encephalopathy are graded subjectively) are operator dependent.
3. It is unable to stratify patients with severely decompensated liver disease.

For this reason, alternative systems have been sought

4.3. Model for end-stage liver disease (MELD) score

The MELD score is a linear regression model based on a patient's serum bilirubin and creatinine levels and international normalized ratio (INR).

4.3.1. MELD scoring equation

MELD score for TIPS = 0.957 × loge (creatinine [mg/dL]) + 0.378 × loge (bilirubin [mg/dL]) + 1.120 × loge (INR) + 0.643 (cause of liver disease)

MELD score for liver transplantation = 0.957 × loge(creatinine [mg/dL]) + 0.378 × loge(bilirubin [mg/dL]) + 1.120 × loge(INR) + 0.643

It was created to predict mortality after TIPS, then to stratify the risks in patients awaiting liver transplant, and recently used to predict perioperative mortality. It has several distinct advantages over the Child classification, being objective, and does not rely on cutoff values.

The general guidelines are as follows:

- Patient with an MELD score below 10 can undergo elective surgery.
- Patient with an MELD score of 10-15 should be managed with caution.
- In patients with an MELD score above 15, elective surgery should be avoided and the patient should be considered for liver transplantation.

These guidelines should be modified for specific circumstances.

4.4. Type of surgery

The operative risk is higher with certain types of surgery, such as hepatic resection, biliary surgery, gastric surgery, colectomy, and cardiac surgery.

Emergency surgery carries higher mortality in hepatic patients than patients with normal hepatic function.

Abdominal surgery as cholecystectomy, gastric bypass, biliary procedures, peptic ulcers, and colon resection is associated with increased morbidity and mortality risks in patients with cirrhosis.

Biliary tract surgery: Patients with obstructive jaundice have increased risk of infections, disseminated intravascular coagulation, gastrointestinal bleeding, delayed wound healing, wound dehiscence, incisional hernias, and renal failure. Patients with cirrhosis are at increased risk of gallstones and their complications. For Child class C patients, cholecystostomy, rather than cholecystectomy, is considered. For patients with obstructive jaundice, nonsurgical approaches to decompression via endoscopic retrograde cholangiopancreatography or percutaneous transhepatic cholangiography are preferred.

Cardiac Surgery: Procedures that require cardiopulmonary bypass are associated with greater mortality in patients with cirrhosis.

Hepatic Resection: Hepatectomies in cirrhotic patients are associated with increased risks. The extent of hepatectomy is a predictor of mortality.

5. Preoperative care of patients with liver disease [28-48]

5.1. Aims

1. Prophylactic measures to prevent complications
2. Early recognition and treatment of complications

5.2. Complications of liver diseases

1. Refractory ascites
2. Spontaneous bacterial peritonitis (SBP)
3. Fluid and electrolyte disturbances
4. Hepatorenal syndrome (HRS)
5. Portal hypertensive bleedings
6. Hepatic encephalopathy (HE)
7. Hepatocellular carcinoma (HCC)

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8. Malnutrition
9. Progress of other medical diseases

5.3. Tests to assess the complications of liver disease

1. Liver imaging and AFP, CA19-9 to exclude neoplasms
2. Doppler ultrasound: to exclude portal vein thrombosis
3. Upper GI endoscopy: to assess portal hypertension
4. Bone densitometry: in selected patients
5. Neuropsychologic testing: selected patients
6. ABG: to exclude hypoxemia-hepatopulmonary syndrome

Particular attention needs to be paid to the management of common complications of advanced liver disease, as coagulopathy, thrombocytopenia, ascites, renal insufficiency, encephalopathy, and malnutrition, as well as to disease-specific factors.

5.4. Coagulopathy

The cause of coagulopathy is multifactorial. It may result from poor absorption of vitamin K due to cholestasis or impaired synthesis of coagulation factors.

- Parenteral vitamin K and transfusions of fresh frozen plasma can be used before surgery.
- Intravenous cryoprecipitate may be infused with a minimal volume load. It contains large amounts of fibrinogen and von Willebrand factor together with clotting factors.
- Intravenous recombinant factor VIIa is a safe and effective in correcting coagulopathy and normalizing the INR.
- For patients with thrombocytopenia, platelet transfusion of may be recommended.
- Prolonged bleeding time can be corrected bydesmopressin acetate.

5.5. Ascites

Grades of ascites:

Grade 1: ascites only detected by ultrasound

Grade 2: moderate with symmetrical distention of the abdomen

Grade 3: large or tense with marked abdominal distension

- Fluid restriction is not necessary in most patients.
- Due to hyperkalemia, spironolactone as a single agent is recommended only in minimal fluid overload.

- The usual regimen is a single morning dose of 100 mg spironolactone and 40 mg furosemide. The dose can be increased every 3-5 days, if weight loss is not satisfactory. Maximum doses are 600 mg/day spironolactone and 200 mg/day furosemide.
- Side effects include volume depletion, which may precipitate encephalopathy, or renal failure.
- Weekly monitoring of electrolytes and weight must be undertaken when initiating or changing therapy.
- Encephalopathy, serum Na <125 mmol/L, creatinine >1.7mg/dL, should lead to cessation of diuretic use.

Refractory ascites is that which is unresponsive to high-dose diuretics and a Na-restricted diet and tends to rapidly recur following paracentesis. Prior to labeling a patient as having refractory ascites, an in-hospital trial of dietary management and diuretic therapy should be attempted.

Treatment options include the following:

1. Paracentesis with albumin replacement remains the first treatment option for patients on the waiting list and are likely to undergo LT within a few months.
   - For large volume paracentesis, an albumin infusion of 8-10 g/L of fluid removed should be considered.
   - Paracentesis increases the risk of peritonitis.

2. TIPS is considered for the following:
   - Cases where the frequency of paracentesis is >3 times/month
   - Patients not tolerating large-volume paracentesis
   - Large-volume paracentesis is ineffective due to multiple adhesions or loculated ascites
   - Refractory hepatic hydrothorax

The major disadvantages are shunt stenosis and HE.

3. Peritoneovenous shunt: for historical interest only

4. Surgical shunts are rarely indicated

5.6. Spontaneous bacterial peritonitis

Definition: infection of the ascitic fluid in the absence of any known intra-abdominal source.

Diagnosis: positive ascites culture and/or polymorphonuclear cell count ≥25 0 cells/mm^3.

Its prevalence justifies diagnostic paracentesis in cirrhotics with ascites admitted to the hospital. Norfloxacin (400 mg/day) significantly reduces the probability of SBP.

Secondary long-term prophylaxis is recommended for all patients with a history of SBP. Antibiotic prophylaxis is recommended in patients with an upper GI bleed irrespective of the presence or absence of ascites.

5.7. Renal impairment

Patients with ESLD are at increased risk to develop renal failure (RF), either spontaneously (hepatorenal syndrome [HRS]) or iatrogenically (diuretics, nephrotoxic drugs). Preoperative renal function significantly affects postoperative survival.

HRS can only be diagnosed after other causes of renal failure are excluded: obstruction, volume depletion, ATN, and drug-induced nephrotoxicity. All diuretics should be stopped. Fluid challenge with 1.5 L of isotonic saline should be administered to exclude volume depletion.

5.7.1. Types of HRS

Type I HRS: rapidly progressive renal failure with an increase in the serum creatinine to more than 2.5 mg/dL within 14 days and marked oliguria.

Type II HRS: stable or slowly progressive impairment in renal function in patients with refractory ascites.

Management:

1. Combination of

- vasoconstrictor drugs, such as vasopressin analogues, noradrenalin, and the combination of midodrine and octreotide together,
- plasma volume expansion with albumin (1 g/kg intravenously on day 1, 20-40 daily thereafter).

Hemodialysis as a bridge to liver transplant might be useful in patients who fail to respond to medical treatment.

Nephrotoxic drugs should be used with cautious, and overtreatment with diuretics should be avoided. It is recommended to stop diuretics if serum creatinine is >1.7 mg/dL.

5.8. Dilutional hyponatremia

Definition: Serum sodium <130 mmol/L.

Cause: impaired free water clearance by the kidneys due to nonosmotic hypersecretion of ADH.

- It represents a late event and indicates poor prognosis. Occurs months after the onset of Na retention.
- It has been proposed to incorporate serum Na concentration in the MELD score; however, this remains controversial.

Management

- As long as the serum Na remains >125 mmol/L, no specific measures are required.
- If the serum Na level is <125 mmol/L, the following should be considered:
  1. Diuretics should be stopped.
  2. Infusion of albumin (100 g/24 h) or red blood cells is instituted attempting at expanding the effective circulating blood volume.

     Na level will increase as a result of turning off ADH secretion by the increased blood volume. Once the serum sodium starts to rise, the albumin infusion is tapered.
  3. Free water restriction.
- Attempts to rapid correction with hypertonic saline can lead to more complications.

5.9. Hepatic Encephalopathy (HE)

HE is a diagnosis of exclusion. Other etiologies as space-occupying lesions, vascular events, metabolic disorders, and infectious diseases should be excluded.

Stages of hepatic encephalopathy

1. Slowing of consciousness
2. Drowsiness
3. Confusion, reactive only to vocal stimuli
4. Coma

Precipitating factors

1. Renal and electrolyte abnormalities
2. Gastrointestinal bleeding
3. Infection
4. Constipation
5. Benzodiazepines, narcotics, or other sedatives
6. Excessive dietary protein intake
7. Worsening liver function, e.g., portal vein thrombosis
8. Noncompliance with medications, especially lactulose

Therapy

1. The mainstay is correcting the precipitating event.
2. Intubation has to be considered to prevent aspiration, depending on the level of consciousness.

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3. Nasogastric tube should be placed.
4. Nonabsorbable disaccharides such as lactulose: The usual starting dose is 20 mL, 3-4 times daily with the aim of achieving 2-4 soft bowel movements per day.
5. Neomycin 3-6 g/day in divided doses might be added. Alternatively, metronidazole can be used.
6. Low-protein diet (minimum 30 g/day).
7. Gluconeogenesis is a significant source of production of endogenous ammonia and may result in worsening of the encephalopathy. Patients should be provided with at least of 400 calories daily in the form of IV glucose to reduce gluconeogenesis.

- Once the patient recovers, a moderate amount of protein (40 g/day) is given and increased to the maximum tolerated dose within few days.
- It is important to avoid protein restriction for a long time to prevent worsening of the nutritional state.
- The role of ornithine-aspartate, sodium benzoate, and branched-chain amino acids is questioned.
- Ammonia level is a poor predictor of the degree of encephalopathy. Changes in ammonia levels should not be considered an indicator of therapeutic benefit; improvement in mental status is the therapeutic end point.

5.10. Portopulmonary Hypertension (PPHTN)

Definition: portal hypertension (clinical diagnosis), mean pulmonary artery pressure (MPAP) >25 mm Hg, pulmonary artery occlusion pressure (PAOP) 15< mm Hg**,** pulmonary vascular resistance (PVR) >240 dyne/s/cm^{-5}.

The detection of PPHTN is crucial as it increases the perioperative and long-term risks.

The most common presenting symptom is progressive dyspnea on excretion; however, patients with even severe PPHTN can be completely asymptomatic. Echocardiography is the screening method of choice. A systolic right ventricular pressure (RVsys) of >50 mm Hg as a cutoff is used. Only these patients need to undergo right heart catheterization to characterize pulmonary hemodynamics.

5.11. Hepatopulmonary syndrome

This is defined as a triad of the following:

1. Chronic liver disease
2. Hypoxemia (PaO_2 <70 mm Hg or alveolar to arterial oxygen gradient >20 mm Hg)
3. Intrapulmonary arteriovenous dilatation or shunts as detected by contrast echocardiography, lung perfusion scanning, or pulmonary angiography

Hypoxemia at rest is the prerequisite for the diagnosis. Medical management is disappointing, and liver transplant is advocated as the treatment of choice.

5.12. Malnutrition

Malnutrition is common with liver impairment and is a risk factor for mortality following LT. Nutritional supplementation has not been proven to affect outcome. The total amount of calories provided should be at least 30-35 kcal/kg/day. Adults can receive daily 1-2 g protein/kg of dry body weight. Patients should take daily multivitamin and other supplements as needed. Specific fat-soluble vitamin supplements are provided if a deficiency is present.

5.13. Psychosocial stress

The preoperative period can be extremely stressful. Declining health, uncertainty about the results, and inability to continue working and participating in daily activities may increase the risk of depression and/or anxiety. Patients with chronic HCV have a greater incidence of depression and anxiety. Patients who experience significant psychological distress have increased complications.

6. Preoperative checklist

The preoperative evaluation concludes with a review of all pertinent studies and information obtained from investigative tests.

1. Informed consent after discussion with the patient and family members regarding the indication for the anticipated surgical procedure, as well as its risks and proposed benefits
2. Review the need for β-blockade, DVT prophylaxis
3. Antibiotic prophylaxis: The appropriate antibiotic is chosen before surgery and administered before the skin incision is made
4. Preoperative mechanical bowel cleansing, whenever indicated
5. Revision of medications
    - Careful review of the patient's medications is important.
    - The aim is to judiciously give medications that control the patient's illnesses and at the same time minimizing the risk associated with anesthetic and other drugs interactions.
    - In general, patients taking cardiac drugs, pulmonary drugs or anticonvulsants, antihypertensives, or psychiatric drugs are advised to take their medications with sips of water on the morning of operation.
    - Parenteral medications are used if the patient remains NPO for any significant period postoperatively.

- It is important to restitute patients to their usual medications as soon as possible.
- Drugs affecting platelet function are withheld for variable time: aspirin and clopidogrel (Plavix) are withheld for 7-10 days, while NSAIDs are withheld depending on the drug's half-life between 1 day (ibuprofen and indomethacin) and 3 days (naproxen and sulindac).

6. Preoperative fasting

7. Postoperative monitoring

- The patients are monitored for signs of hepatic decompensation, such as ascites, worsening jaundice encephalopathy, coagulopathy, and renal impairment.
- If any of these occur, supportive therapy is started immediately.
- Prothrombin time is the single best indicator of the synthetic function of the liver.
- Elevated serum bilirubin level may result from worsening of the liver function and also may be elevated because of other conditions, as blood transfusions, blood extravasation, or infection.
- Renal function must be monitored closely. If renal impairment occurs, the cause should be suspected and treatment started.
- In cases of severe impairment of the liver functions, hypoglycemia may occur as a result of depletion of liver glycogen stores and impaired gluconeogenesis. Serum levels of glucose should be monitored closely if postoperative liver failure is suspected.
- Careful attention should be paid to the IV fluid infusions.

Intravascular volume maintenance minimizes the risk of hepatic and renal underperfusion.

At the same time, crystalloid overinfusion results in liver congestion, venous oozing and pulmonary congestion and edema, ascites, peripheral edema, and wound disruption.

8. Bottom lines

- Accurate preoperative identification of patients with liver disease allows their treatment plans to be adjusted.
- In patients with acute liver disease, elective surgery should be postponed until symptoms resolve.
- Elective surgery should be avoided in patients with acute liver diseases such as acute viral hepatitis or alcoholic hepatitis, if there is evidence of ongoing hepatic injury.

- In cases of chronic liver diseases, it is mandatory to assess the severity of underlying disease before deciding whether to proceed with surgery.
- MELD and CTP scores can be used to stratify the risks of surgery for patients with chronic liver disease.
- Optimal preoperative management can reduce the risk of postoperative morbidity and mortality.
- Preoperative management of complications related to patients' underlying liver disease is essential to optimize their outcomes.

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