Lecture 5[2hrs]

Vascular disorders of the liver include

1-Hepatic arterial disease

A-Liver ischemia

acute circulatory failure with hypoperfusion of the liver causing acute hepatocyte injury.

Causes include -any cause of acute hypotension, most commonly seen in acute myocardial infarction. Patients with pre-existing liver disease, especially alcoholic liver disease are more born to injury.

Investigations.

*Rapid rise in serum transaminase concentrations. Characteristically these return rapidly to normal within seven days if the underlying cause of hypotension is corrected.

*The serum bilirubin and alkaline phosphatase may rise slightly but also may be delayed simulating a cholestatic pattern.

*In difficult diagnostic cases the liver biopsy shows characteristic zone 3 injuries.

*Evidence of end-organ hypoperfusion especially acute renal injury.

Treatment.

Therapy aims to restore cardiac output and reverse the underlying cause.

B-Hepatic Artery Occlusion.

Cause includes

*Injury during biliary surgery

*Emboli, Neoplasms, Polyarteritis Nodosa, blunt trauma or radiation.

Clinical features include

*Severe upper abdominal pain with or without signs of circulatory shock.

*Raised transaminases (AST or ALT usually > 1000 U/L).

C-Hepatic Artery Aneurysms may be extrahepatic or intrahepatic.

causes include

*Atheroma*vasculitis*bacterial endocarditis *surgical or biopsy trauma.

They usually lead to bleeding into the biliary tree, peritoneum or intestine

Diagnoses- by angiography.

Treatment is radiological or surgical.

D-Hepatic Artery Thrombosis is a recognized complication of liver transplantation and typically occurs in the early post-transplant period.

***Clinical features** usually related to bile duct rather than liver ischemia because of the dominant role of the hepatic artery in extrahepatic bile duct perfusion. Manifested by

*Bile leak or the development of bile duct strictures due to bile duct anastomotic failure.

Diagnosis- by ERCP

Treatment -biliary stenting for bile duct injury.

E-Congestive Heart Failure

Patients with either acute or chronic congestive heart failure will commonly have biochemical and clinical features of liver disease that reflect passive hepatic congestion.

Clinical features of hepatic congestion include

*Tender hepatomegaly.

*Features of right heart failure include- raised JVP and positive hepato-jugular reflux.

*Pulsatile liver in tricuspid insufficiency.

*Ascites with high protein content.

Investigations

*Elevation of aminotransferases (< 300) and a mild elevation of alkaline phosphatase.

*The level of bilirubin elevation correlates well with the degree of heart failure and may be disproportionately elevated to the liver enzymes.

* Liver biopsy will reveal classical zone 3 changes with a central vein, sinusoidal dilatation, and hemorrhage.
Chronic cases may develop cirrhosis and PHT with hard irregular hepatomegaly and splenomegaly.
The prognosis is directly related to the severity of heart failure and its response to therapy.

2-Portal Vein Thrombosis. [PVT]

Portal venous thrombosis as a primary event is rare but can occur in any condition predisposing to thrombosis.
Causes of PVT in children include *Neonatal umbilical sepsis.
In adults cause include –
*Trauma, local inflammatory condition (pancreatitis), neoplasia (hepatoma), hypercoagulable conditions, and idiopathic.
*Portal vein thrombosis can arise as a secondary event in patients with cirrhosis and portal hypertension and may cause decompensation in patients with previously compensated cirrhosis.
Clinical Presentation
*Acute portal venous thrombosis causes abdominal pain and diarrhea and rarely leads to bowel infarction and perforation.
*Subacute thrombosis can be asymptomatic or lead to extrahepatic portal hypertension with massive hematemesis from bleeding esophageal varices.
*Encephalopathy is uncommon and bleeding episodes are better-tolerated
investigation
*Biochemical tests of the liver are normal. Because the liver function is usually preserved.
*Diagnosis by a Doppler ultrasound of the portal vein or a venous phase of hepatic angiography.
Treatment
*Anticoagulation for thrombosis*Surgery for bowel infarction.
Controlling the bleeding esophageal varices and prevention of bleeding EV
by *Variceal banding. *B-blockers.
*Placement of a transjugular intrahepatic portosystemic shunt (TIPS).
Because of normal liver parenchyma, mesocaval shunt generally better tolerated than by patients with chronic liver disease.

3-Budd – Chiari Syndrome [BCS].

*BCS is a disorder characterized by obstruction of hepatic venous outflow, this obstruction may be at the level of the small hepatic veins, large hepatic veins or the suprahepatic portion of the inferior vena cava (IVC)
*BCS is more common in women and usually presents in the third or fourth decade.
BCS maybe
***Primary**- When the obstructing process arises from the venous wall (phlebitis or fibrous thickening) or the venous lumen (thrombosis)**.**
***Secondary** -When the veins are compressed or invaded by a lesion arising outside the vein.
Causes include
* Myeloproliferative disorder (polycythemia vera, essential thrombocytosis, myeloid metaplasia) with its associated hypercoagulability

*Paroxysmal nocturnal hemoglobinuria
*Protein C and S deficiency
*Oral contraceptives *SLE, *Behcets disease, *Polycystic liver disease and *Adrenal or Renal cell carcinoma * pregnancy
*Infections and mechanical obstructive lesions such as tumors, membranous vascular webs.

Clinical Presentation

depend on the extent and the rapidity of the vascular occlusion.

In the acute disease patients present with rapidly developing
Tender hepatomegaly and Marked ascites *Liver failure with jaundice and coma *Negative hepatic – jugular reflux.

The subacute and chronic disease-

*Features of PHT like *ascites *variceal bleeding, *Veins distended over the abdomen, flanks, back. *Leg edema occurs when I.V.C is blocked.

Investigation

*Elevation of AST and ALT *hyperbilirubinemia *increasing INR.
*The serum-ascites fluid albumin gradient is high, with the total protein level in the ascitic fluid > 2.5 g/dL.
*Doppler ultrasound is the diagnostic procedure of choice. The typical ultrasound features include the inability to visualize normal hepatic venous connections to the vena cava, absence of any waveform in the hepatic vein, and Caudate lobe hypertrophy.
*In difficult diagnostic cases, contrast CT scan, MRI or IVC hepatic venography
*Isotope imaging may show preservation of the caudate lobe.

Management:-

*Predisposing causes should be treated.
*Anticoagulant for Hyper coagulation.
*Venosection + cytotoxic therapy for Polycythemia treated.
*Streptokinase followed by heparin and oral anticoagulation for Recent thrombosis.
*Low sodium diet, Diuretics and paracentesis for Ascites.
*Surgery or dilatation for I.V.C web or stenosis.
*Liver transplantation for progressive liver failure.

Prognosis-generally poor when the onset is sudden. 2/3 of patients die within a year and few live > 5 years, some survive to develop cirrhosis

4-Veno – Occlusive Disease (VOD):

is a rare condition characterized by widespread occlusion of the small central hepatic vein?

Causes include- pyrrolizidine alkaloids, hepatic irradiation, Azathioprine, and graft-versus-host disease related to bone marrow transplantation.

The presentation In the acute form includes *Hepatomegaly, *Ascites, and *Hyperbilirubinemia.

The chronic form leads to cirrhosis and portal hypertension with esophageal varices.

Treatment

*Supportive as 70-85% recover spontaneously.
*Control of the ascites by sodium restriction and use of diuretics.
*TIPS for refractory ascites. *Liver transplantation

Pregnancy-Associated Liver Disease

Spectrum of liver disease occurs during gestation and the postpartum period that result in abnormal liver function test and hepatobiliary dysfunction**.**
The following conditions occur only during pregnancy, may recur in subsequent pregnancies and resolve after delivery of the baby.

1-Hyperemesis gravidarum

*It occurs in ~0.5% of pregnancies, and is more common in nulliparous women and twin pregnancy. It presents early in the first trimester.

Characterized by

*Nausea and vomiting.
* Weight loss, *Dehydration occurs in severe cases
* Jaundice (usually mild), *Excessive salivation
*Transient hyperthyroidism may occur because human chorionic gonadotrophin (hCG) having thyroid-stimulating activity.

Investigations

* Mild elevation of liver enzymes usually <250 U/L.
*Ketonuria reflecting more severe starvation and dehydration.

Treatment-

*****Supportive care, IVF, electrolyte replacement, vitamin supplementations [Thiamine deficiency can occur and needs correction,]
*Antiemetic like [promethazine, metoclopramide, ondansetron,]

Complications include esophageal rupture, Wernicke's encephalopathy, central pontine myelinolysis, retinal hemorrhage, and spontaneous pneumomediastinum.

2-Intrahepatic Cholestasis of Pregnancy (ICP).

*Usually occurs in the second or third trimester of pregnancy. characterized by new onset pruritus in the second or third trimester, elevated bile acid and Mild jaundice (bilirubin < 5 mg/dL).
*The most common liver disease specific to pregnancy
Risk factors Multiple gestations (e.g., twin pregnancy), multiparity, metabolic syndrome, HCV infection, personal or family history of ICP.
*Fetal Risk-May cause intrauterine growth retardation and premature birth if the pregnancy goes beyond 36 weeks of gestation**.**

Treatment-

*UDCA modifies the bile acid pool by increasing hydrophilic and less cytotoxic.
*Delivery leads to resolution of the condition

3-Acute fatty liver of pregnancy [AFLP]

*It typically presents in the third trimester between 31 and 38 weeks of pregnancy or postpartum
*This is more common in twins and first pregnancies.
*More frequently when the fetus is male.

Pathogenesis - a defect in beta-oxidation of fatty acids in the mitochondria [Deficiency of LCHAD] that leads to the formation of Microvesicular fatty liver**.**

Clinical presentation

*Vomiting and abdominal pain followed by jaundice. *Coagulopathy* Encephalopathy and ascites.

Investigations

*US shows fatty liver and blood tests
*Leukocytosis, increase transaminase, ↑ammonia, ↑bilirubin, ↑urate, low blood sugar ↑creatinine,
*Liver Biopsy shows micro vesicular fatty infiltrations.

Treatment- Early delivery of the fetus. And molecular testing for LCHAD

4-Toxaemia of Pregnancy and HELLP Syndrome.

*Usually occurs in Second or third trimester of pregnancy or postpartum
Risk factors include hypertension, DM, Multiparity and Age > 35
Clinical presentation.
*Abdominal pain, nausea, vomiting
*Pre-eclampsia [hypertension, edema, proteinuria]. Pre-eclampsia can progress to eclampsia (pre-eclampsia + seizures), AFLP and HELLP Syndrome.
*Maybe association with **(HELLP**) syndrome.
[**HELLP**] **H**aemolysis, **E**levated Liver enzyme, and **L**ow **P**latelets
Investigations-
***H**emolysis (↑ LDH, ↑indirect bilirubin) *Elevated serum transaminases*Low platelet
*Low hemoglobin, with fragmented red cells,
*Raised D-dimers.
Complication.
*Hepatic infarction and rupture.
*Disseminated intravascular coagulation and placental abruption.
*Maternal mortality is 1% and perinatal mortality can be up to 30%.
Treatment-
***D**elivery and platelet transfusion [when platelet level < 40,000– 50,000 cells/µl].
the disease recurs in <5% of subsequent pregnancies

Acute Liver Failure [ALF]

*Characterised by the onset of severe acute hepatic synthetic dysfunction associated with encephalopathy and coagulopathy [INR ≥1.5] within 6 m of onset of symptoms in patients without pre-existing liver disease**.** *Acute liver failure is an uncommon but serious syndrome.
Classifications of ALF
Old Classification
The interval between onset of jaundice and encephalopathy divides acute liver failure into
1-Fulminant hepatic failure- development of encephalopathy within 2 W of the onset of jaundice
2-Sub- fulminant hepatic failure –development of encephalopathy >2 W to 3 months after the onset of jaundice
Recent Classification
The interval between onset of jaundice and encephalopathy divides acute liver failure into
1-Hyperacute-The encephalopathy develops within 7 days of the onset of jaundice.
2-Acute-The encephalopathy develops between 8- 28 days after the onset of jaundice.
3-Subacute [-the encephalopathy develops between 4-12 weeks after the onset of jaundice.
Causes include. * viral hepatitis A, B, C, D, E * drugs [Paracetamol, Halothane, Antituberculous] *Herbal remedies *Amanita phalloides (mushroom) poisoning* Wilson's disease *acute circulatory failure, *Budd-Chiari syndrome, *veno occlusive disease *heat stroke* Leptospirosis *autoimmune hepatitis *Massive infiltration of the liver with a tumor. * Lymphoma. *Acute fatty liver of pregnancy *Reyes syndrome, *cytomegalovirus, herpes simplex and herpes zoster viruses in immunosuppressed patients].
*10% of cases are a cryptogenic acute liver failure**.**
Clinical assessment

1-Hepatic Encephalopathy is the cardinal manifestation of acute liver failure, but in the early stages, this can be mild and episodic.

Clinical grade of Hepatic Encephalopathy

Grade 1: Poor concentration, slurred speech, slow mentation, disordered sleep rhythm

Grade 2: Drowsy but easily arousable, occasional aggressive behavior, lethargic

Grade 3: Marked confusion, drowsy, sleepy but a response to pain and voice, gross disorientation

Grade 4: Unresponsive to voice, may or may not respond to painful stimuli, unconscious.

2-Cerebral edema is the most common cause of death in acute liver failure occur due to increased intracranial pressure.

Examination-

Unequal or abnormally reacting pupils fixed pupils *hypertensive episodes*bradycardia, *Hyperventilation* Profuse sweating*local or general myoclonus*Focal fits

*Decerebrate posturing.

*Papilloedema is a late sign

*Jaundiced except in Reye's syndrome when jaundice is rare.

Occasionally death may occur in fulminant cases of acute liver failure before jaundice develops.

* Fetor hepaticus can be present.

*The liver is usually of normal size but later becomes smaller, Hepatomegaly is unusual.

* The Presence of a sudden onset of ascites occurs in (Budd-Chiari syndrome).

*Splenomegaly is uncommon in acute liver failure.

*Ascites and edema are late sings s and may be a consequence of fluid therapy.

Investigations include.

*Toxicology screen of blood and urine.

*HBsAg, IgM anti-HBc *IgM anti- HAV *Anti-HEV, HCV, cytomegalovirus, herpes simplex, Epstein-Barr virus.

*Ceruloplasmin, serum copper, urinary copper, slit-lamp eye examination.

*Autoantibodies: ANF, ASMA, LKM.

*Immunoglobulins *Ultrasound of liver and Doppler of hepatic veins.

*Plasma aminotransferase is high after paracetamol overdose, reaching 100-500 times normal, but falls as liver damage progresses and does not help determine prognosis.

*The prothrombin time rapidly becomes prolonged as coagulation factor synthesis fails; this is the laboratory test of greatest prognostic value and should be done at least twice daily.

*Factor V levels can be used instead of the prothrombin time to assess the degree of liver impairment.

*Plasma albumin remains normal unless the course is prolonged.

*Percutaneous liver biopsy is contraindicated because of the severe coagulopathy, but a biopsy can be undertaken using the transjugular route.

Management

*Patients with acute liver failure should be treated in the intensive care unit.

*Conservative treatment to maintain life in the hope that hepatic regeneration will occur.

*Early transfer to a specialized transplant unit should always be considered.

*N-acetylcysteine therapy may improve outcomes, particularly in patients with acute liver failure due to paracetamol poisoning. N-acetylcysteine 150mg/kg IV in 200 ml D5W over 15 mint followed by 50mg/kg in 500 ml D5W over 4 hrs and 100 mg /kg in 1000D5W over 16 hrs.

*Mushroom poisoning treated –by iv penicillin G 300.000-1million units/kg per day

Treatment of Complications of Acute Liver Failure

1-Treatment of Encephalopathy -Search for treatable causes like (e.g., hypoglycemia, sepsis, gastrointestinal bleeding, electrolyte imbalance, decreased PO2, increased PCo2* sedative drugs.

2-Treatment of Cerebral edema. The goal is to maintain an intracranial pressure of less than 20 mm Hg. control of agitation, the head elevation of 20 to 30 degrees, hyperventilation,

administration of

*Mannitol 0.25-0.5 mg /kg bolus hourly until ICP improved,

*Barbiturate-induced coma.

Hypoglycemia is a common complication of liver failure resulting from impaired hepatic gluconeogenesis and insulin degradation

3-Treatment of Hypoglycemia.

*All patients should receive 10% glucose intravenous infusions with frequent monitoring of blood glucose levels.

4-Treatment of Metabolic abnormalities commonly occur, including hyponatremia, hypokalemia, respiratory alkalosis, and metabolic acidosis. Thus, frequent monitoring of blood electrolytes and pH is indicated.

5-Treatment of Renal failure by - iv fluid if the patient is hypovolemic, dopamine infusion, hemodialysis, or continuous hemofiltration

6-Treatment of Coagulopathy, thrombocytopenia, aplastic anemia, sepsis

7-Treatment of Acute Respiratory distress syndrome

8-Liver Transplantation

Adverse Prognostic Criteria in Acute Liver Failure due to paracetamol overdose include

*(pH < 7.3) at or beyond 24 hrs following the overdose Or Serum creatinine ≅ 3.38 mg/dL) plus prothrombin time > 100 secs plus encephalopathy grade 3 or 4

*** Non-paracetamol acute liver failure include**.

Prothrombin time > 100 secs or • Any three of the following: *Jaundice to encephalopathy time > 7 days *Age < 10 or > 40 yrs.*Indeterminate or drug-induced causes *Bilirubin ≅ 17.6 mg/dL) *Prothrombin time > 50 secs or *Factor V level < 15% and encephalopathy grade 3 or 4.

Chronic Hepatitis

*Means active, ongoing inflammation of the liver that persists for more than six months.

*Chronic hepatitis can develop in patients with HBV, HCV, and HDV infection. autoimmune hepatitis, drug-induced hepatitis, Wilson's disease,1-antitrypsin deficiency and steato-hepatitis, Primary biliary cirrhosis, and primary sclerosing cholangitis.

*Chronic hepatitis characterized by increasing in the serum aminotransferases (AST and ALT), with minimal elevation of the alkaline phosphatase (AP).

*Chronic hepatitis may progress to cirrhosis with PHT and its complications.

*Liver biopsy is the gold standard to evaluate the grade (degree of inflammation) and stage (degree of fibrosis/cirrhosis) of chronic viral hepatitis.

*The most commonly used system for grading and staging of hepatitis is the METAVIR system Histologic or inflammatory activity (A score) is determined by the amount of portal and lobular inflammation and necrosis into a score from A0-A3.
The degree of fibrosis (F score) is evaluated separately to obtain the stage of disease and ranges from F0-F4.

Histologic

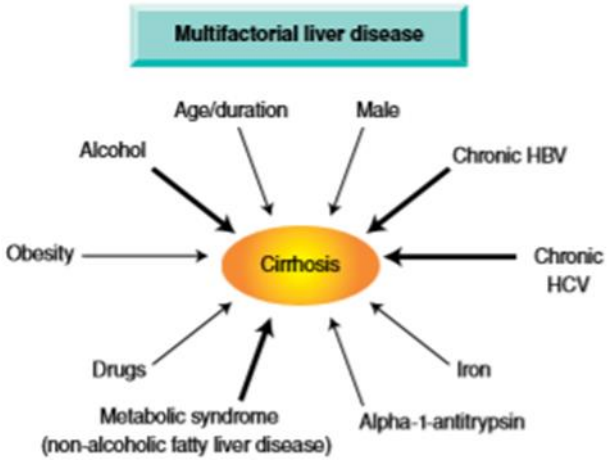
A0 = no activity
A1 = mild activity
A2 = moderate activity
A3 = severe activity
F0 = no fibrosis
F1 = portal fibrosis without septa
F2 = portal fibrosis with few septa
F3 = numerous septa without cirrhosis
F4 = cirrhosis
*Also The stage of fibrosis can be determined by fibroscon

Liver Cirrhosis

*Cirrhosis is the final pathway for a wide variety of chronic liver diseases causing diffuse hepatic fibrosis with the replacement of the normal liver architecture by regeneration nodules that disrupts the structure and function of the liver.
*The rate of progression of chronic liver disease to cirrhosis may be variable from weeks in patients with complete biliary obstruction to decades in patients with chronic hepatitis C.
*Cirrhosis can occur at any age.
*Cirrhosis interferes with liver blood flow and function. This derangement produces the clinical features of portal hypertension and impaired liver cell function.

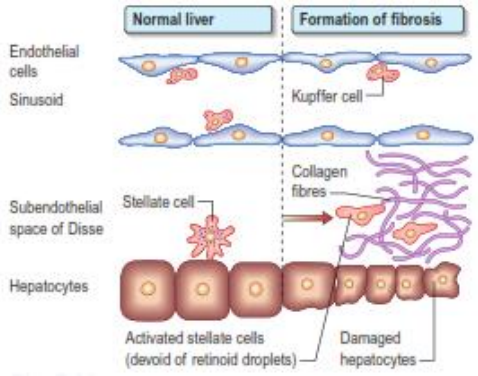
Causes of Liver Cirrhosis include

*Viral hepatitis B, C, D*Alcohol*Metabolic syndrome, [NASH] *Hemochromatosis *Wilson's disease, *Alfa 1-antitrypsin deficiency *Autoimmune Sclerosing cholangitis*Primary biliary cirrhosis* Autoimmune Hepatitis * Drug-induced, *Congestive Cardiac failure *Budd-Chiari syndrome *Cystic fibrosis *Secondary biliary cirrhosis [Stone, Stricture] *Galactosemia and Tyrosinemia. About 5-10% cryptogenic cirrhosis [when the cirrhosis has no known cause].



Pathogenesis

*Following liver injury stellate cells in the space of Disse are activated by cytokines produced by Kupffer cells and hepatocytes. This transforms the stellate cell into a myofibroblast-like cell, capable of producing collagen.
*Cirrhosis It evolves over years as progressive fibrosis and widespread hepatocyte loss lead to distortion of the normal liver architecture that disrupts the hepatic vasculature, causing portosystemic shunts. These changes usually affect the whole liver, but in biliary cirrhosis, they can be patchy.



Pathology
The characteristic feature of cirrhosis is regenerating nodules separated by fibrous septa and loss of the normal Lobular architecture within the nodules.
Types of Cirrhosis include
*Micro nodular: Characterized by Regenerative nodule < 1 mm in diameter.
This type is caused by ongoing alcohol damage or biliary tract disease
*Macro nodular: Characterized by nodules of variable size.
This type is seen following chronic viral hepatitis.
*Mixed macronodular and micro nodular.
Clinical Features due to cirrhosis itself and its Complication
1-Features due to portal hypertension.
2-Features due to hepatocellular failure.
3-Features due to a combination of hepatocellular failure and portal hypertension.

1-Features due to Portal hypertension (portal venous pressure >10 mmHg) include -

*Prominent abdominal vein with/without caput-medusae. Much prominent collateral vein radiating from umbilicus giving the picture of spider leg it is due to collateral anastomosis between para-umbilical vein (a tributary of the portal vein) with the superior epigastric, lateral thoracic, superficial epigastric inferior epigastric, posterior intercostal and lumbar vein (a tributary of vena cava).

*Cruveilhier-Baumgarten syndrome—a venous hump may be audible over the umbilicus over the caput due to extensive collateral connections.

*Splenomegaly—When massive it leads to heaviness of LUQ of the abdomen, early satiety, splenomegaly may contribute to anemia and/or thrombocytopenia.

* Hematemesis and melena or hematochezia may result from rupture of dilated esophageal varices or rectal hemorrhoids

2-Features due to hepatocellular failure include

A-Features due to hyperestroginism include

*Spider nevi. *Palmar erythema. * Gynecomastia. *Loss of body, axillary and pubic hair. * Loss of libido. * Testicular atrophy and infertility. *Menstrual irregularity in the female. This is mostly due to decreased metabolism of estrogen precursor(androstenedione) in the liver giving the feature of hyperestroginism.

B- Features due to non-detoxification of NH3 and related compounds.

*Fetor hepaticus * Hepatic flap (asterixis) * Hepatic encephalopathy.

C-Features due to non-Synthesis of albumin

* Alteration of albumin and globulin ratio[A: G].

*White nails (leukonychia)—Nails become white and brittle due to Hypoalbuminemia.

D- Features due to non-synthesis of Clotting factors and Thrombocytopenia

* Increased bleeding* Increased clotting time and *INR.

3-Combined features of hepatocellular failure and portal hypertension

*Ascites *Fetor hepaticus*Encephalopathy* Hepatorenal syndrome

2-Laboratory tests with deteriorating hepatic function include

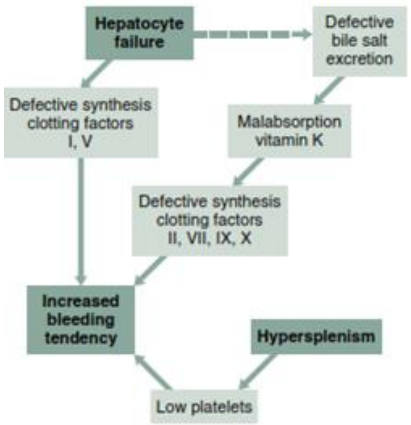
*Low albumin, *increase serum bilirubin level, *Alkaline phosphatase is usually raised

*Normochromic, normocytic anemia is found with target cells noted in the blood smear.

* if gastrointestinal bleeding occurs the anemia may be microcytic as a result of iron loss

*Liver enzymes may be within the normal range despite the severe liver disease

*Prolonged prothrombin time *Low leukocyte and platelet counts may be present secondary to hypersplenism.



*The urine often contains urobilinogen and bilirubin if the patient is jaundiced.

*Reduction in urinary sodium excretion in patients with ascites.

3-imaging Features include

***Ultrasound examination**.:

*Ultrasound of the abdomen reveals an inhomogeneous nodular liver with splenomegaly and distortion of the arterial vascular architecture.

*The patency of the portal and hepatic veins can be evaluated.

*US is useful in detecting hepatocellular carcinoma.

***Fibroscan or magnetic resonance elastography** [MRE for Grading of fibrosis].

***CT scan**: shows hepato-splenomegaly, and dilated collaterals. Arterial phase-contrast-enhanced Scans are useful in the detection of hepatocellular carcinoma

***Endoscopy**-

***Gastroduodenoscopy**-performed for the detection and treatment of varices, and portal hypertensive. gastropathy.

*Colonoscopy is occasionally performed for colopathy**.**

***MRI Scan**. This is useful in the diagnosis of benign tumors such as hemangiomas**.**

***MR Angiography** can demonstrate the vascular anatomy.

***MR Cholangiography** for the biliary tree visualization.

Management include

*Treatment of the Underlying cause.

*Maintenance of Nutrition

*Treatment of Complications [ascites, hepatic encephalopathy, portal hypertension, and Bleeding varices.]

*Once the diagnosis of cirrhosis is made endoscopy should be performed to screen for esophageal varices and repeated every 2 years.

*Screening for hepatocellular carcinoma by - [Alfa-fetoprotein and US every 6 M] because the risk of HCC increased in patients with cirrhosis.

Drugs to be avoided in cirrhosis include [NSAIDs, ACE inhibitors, Codeine Narcotics and Anxiolytics

Prognosis.

Prognosis is poor but more favorable when the underlying cause of cirrhosis can be corrected ex [when the alcoholic patient stops alcohol consumption when the iron can be removed by venesection in the patient with hemochromatosis when excessive copper has removed inpatient with Wilson's disease].

Laboratory tests give only a rough guide to prognosis in individual patients.

*Deteriorating liver function, as evidenced by jaundice, ascites, or encephalopathy *Increasing bilirubin, albumin concentration < 30 g/L), marked hyponatremia (< 120 mmol/L) not due to diuretic therapy, and a prolonged PT is all bad prognostic features.

The commonest scoring system used for assessment of the severity of hepatic dysfunction is the *Child–Turcotte–Pugh score. Based on this score, patients are grouped into three severity levels: A, B, and C are predictive of prognosis and useful in determining the required strength of treatment and the necessity of liver transplantation

*Another specialized pretransplantation assessment is the Model for End-stage Liver Disease (MELD) score

Child-Pugh – classification of prognosis in liver cirrhosis.

Score	1	2	3
Encephalopathy	none	1-2	3-4
Bilirubin [mmol/L]	<34	>34	50
Albumin [g/L]	35	28-35	28

P T[s prolonged]	4	4-6	6
Ascites	none	mild	marked

	Score	Survival at 5 years
Childs A	< 7	45%
Childs B	7-9	20%
Childs C	> 9	20%