Clinical biochamis, Second chapter 6

### Liver disease

إسراءيوسف ملكاوي "(لتفويغ شابترالسان)"

كيف نعل فوصل لا مع ما الم لحي معكنا شف الأوامد إ

### Liver

> The liver is the largest, most versatile organ in the body

> It consists of two main lobes that, together, weigh from 1400-1600 g in the normal adult 602 Lobricia 200

It has an abundant blood supply/receiving about 15 ml/minute from two major vessels: the hepatic artery and the portal vein

The hepatic artery a branch of the aorta, contributes 20% of the blood supply and provides most of the oxygen requirement

The portal vein, which drains the gastrointestinal tract, transports the most recently absorbed material from the intestine to the liver

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although only 1 L is excreted. The excretion of bile Total bile production averages about 3 L per day. احنا مطلع هاليوم على التر الك

The primary bile acids cholic acid and chenodeoxycholic acid, are formed in the liver from cholesterol. The bile acids are conjugated with the amino acids glycine or taurine, forming bile salts. - Lame

ب وال باليوم معك fold in the gallbladder During fasting and between meals, bile acid pool is concentrated up to 10at in power of secretality and the

and these secondary bile acids are subsequently absorbed the terminal ileum and colon, dehydration to secondary bile acids occurs, When the conjugated bile acids (salts) come into contact with bacteria in

felease,

of bile occurs 2-5 times daily where they are reconjugated and reexcreted. The enterohepatic circulation The absorbed bile acids enter the portal circulation and return to the liver,

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Bilirubin-p ) hap be a poly

glyc'm

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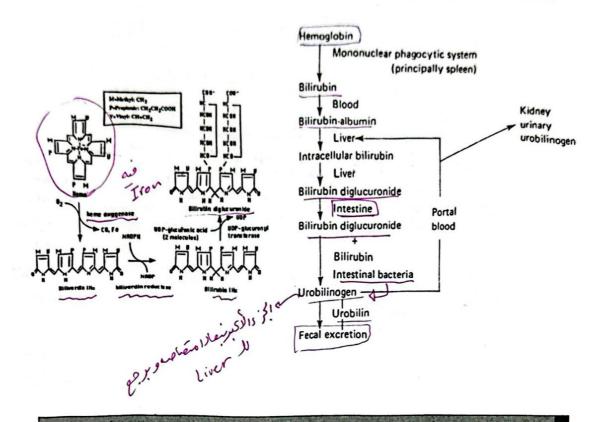
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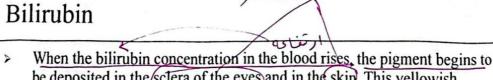
### Bilirubin-0 ) bap beau of

system, primarily in the spleen, liver, and bone marrow where it is conjugated with diglucuronide on it two carboxylic acids and when aged red blood cells are phagocytized by the reticuloendothelial body, The iron enters the body suron stores and is also reused. The Bilirubin is transported to the liver in the bloodstream bound to(albumin) When hemoglobin is destroyed The principal pigment in bile is derived from the breakdown of hemoglobin, porphyrin is broken down as a waste product and excreted the protein portion globin is reused by the الهيم وعلوين الل فيها ختيكسرل جيم وعلوبن خومول بالم Amino

excreted to the intestine The normal flora) in the colon oxidize it further where it is reabsorbed, تاحيره

Jensey Sills (6) urobilinogen, colourless) excreted to the stool (urobilin) reddish brown or excreted in kidney exceled. in stool بها د اللون





بكونوا عاجمهار

be deposited in the sclera of the eyes and in the skip. This yellowish pigmentation in the skin or sclera is known as jaundice, or icterus

The cause can be:

Prehepatic results when an excessive amount of bilirubin is presented to the liver for metabolism, such as in hemolytic anemia. This type of jaundice is characterized by unconjugated

hyperbilirubinemia
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appear in the urine in this type of jaundice.

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مارع رنا رفي البول لأنت مهرفي البول عابتنام ما د البوض

### Bilirubin

متأثرة خلاسا الكبر (متعالة بالكبرنفسه)

Hepatic: Impaired cellular uptake, defective conjugation, or abnormal secretion of bilirubin by the liver cell are the main causes of this kind of iaundice

ممكن بكون الكسيلم .. الإلى المنكالة و م

Posthepatic results from the impaired excretion of bilirubin caused by (mechanical obstruction of the flow of bile into the intestine. This may be due to gallstones or a(tumor) الورم العقصودهون 0 اد حمی

the head of When bile ceases to flow into the intestine, there is a rise in the serum level of conjugated bilirubin and the stool loses its source of normal pigmentation and becomes clay-colored

Conjugated bilirubin appears in the urine, and urine urobilinogen levels decrease,

رومل للأمعاء (نبكره) وبوقف MI bilel1 راع للافعاء

(يزيد ومه

### Major Synthetic Activity

- The liver plays an important role in plasma protein production, synthesizing albumin and the majority of the  $\alpha$  and  $\beta$ -globulins. All the blood-clotting factors (except VIII) are synthesized in the liver ك حاى الأثبياء فيصير في المان
- The deamination of glutamate in the liver is the primary source of ammonia, which is then converted to(urea)
- Glycogenesis and gluconeogenesis, lipogenesis, metabolism of cholesterol into bile acids, Very-low-density lipoproteins (transport TG into the tissues), Highdensity lipoproteins, phospholipids are all made in the liver

lipid 11@ fasting for wish 11:5 The formation of ketone bodies ووستحول الم وهاي محكن مصدر لللاكمة للدما

The liver is the storage site for all fat-soluble vitamins (A, D, E, and K) and several water-soluble vitamins, such as (B12) and is responsible for the conversion of carotene into vitamin A

لح ذ اجد عنه و مشكله عنه صمل الكسرها و كل الوظائف صفيعي

### Synthesis of liver enzymes

Many enzymes are synthesized by liver cells, but not all of them have been found useful in the diagnosis of hepatobiliary disorders, this includes:

Aspartate aminotransferase (AST) and alanine aminotransferase

(ALT) which escape into the plasma from damaged liver cells

Alkaline phosphatase (ALP) and 5'-nucleotidase (5NT): induced or released when the canalicular membrane is damaged and biliary obstruction occurs

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### Detoxification and Drug Metabolism متصرفانا

- The <u>liver protects the body from potentially injurious substances</u> absorbed from the intestinal tract and toxic by-products of metabolism.
- The most important mechanism in this detoxification activity is the microsomal drug-metabolizing system of the liver. It is responsible for many detoxification mechanisms, including oxidation, reduction, hydrolysis, hydroxylation, carboxylation, and demethylation that convert many insoluble compounds into other forms that are less toxic or more water-soluble and, so excretable by the kidney.
- Conjugation with moieties, such as glycine, glucuronic acid, sulfuric acid, glutamine, acetate, cysteine, and glutathione, occurs mainly in the cytosol or smooth ER. This mechanism is the mode of bilirubin and bile acid excretion.

Endoplasmic Reticulum

### Disorders of the liver jaundice

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- Jaundice or icterust is the yellowish discoloration of the skin and sclerae resulting from hyperbilirubinemia
- Although the upper limit) of normal for total serum bilirubin is 1 mg/dL, jaundice is not clinically apparent until the bilirubin level exceeds 2-3 mg/dL
- In African American or Asian patients, yellowing of the sclerae may be the only clinical evidence of jaundice

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- Except in infants, hyperbilirubinemia is generally well tolerated
- In infants, hyperbilirubinemia (>15-20 mg/dL) may be associated with kernicterus (serious disorder of the CNS resulting from increased bilirubin levels) it only occurs in infants because the immature CNS does not have a well-developed blood-brain barrier

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### Jaundice

- Although all cases of jaundice result from hyperbilirubinemia not all are caused by hepatic dysfunction.
- hyperbilirubinemia may also result from erythrocyte destruction, or hemolysis in patients with normal liver function

Hypercarotenemia (excessive ingestion of vitamin A) may produce skin discoloration indistinguishable from that of hyperbilirubinemia. In hypercarotenemia, the sclerae are usually not discolored.



### Cirrhosis



- Cirrhosis refers to the irreversible scarring process by which normal liver architecture is transformed into abnormal nodular architecture
- One way to classify cirrhosis is by the appearance of the liver (by the size of the nodules). These conditions are referred to as macronodular and micronodular cirrhosis, although mixed forms occur
- In the USA, Canada, and Western Europe, the leading cause of cirrhosis is alcohol abuse, which leads to a micronodular type of cirrhosis
- > Other causes of cirrhosis include hemochromatosis, postnecrotic cirrhosis (occurs as a late consequence of hepatitis), and primary biliary cirrhosis (an autoimmune disorder).

### معنا عفاته فطرة و Cirrhosis

Cirrhosis is a serious disorder and one of the ten leading causes of death in the United States. It causes many complications:

Portal hypertension results when blood flow through the portal vein is obstructed by the cirrhotic liver. This may result in splenomegaly and esophageal varices (may rupture and lead to fatal hemorrhage)

عت معمل مزيعة داملي ويوي دوالي و الري تعالي التي وساموس The synthetic ability of the liver is reduced, causing

hypoalbuminemia and deficiency of the clotting factors, which may lead to hemorrhage

Ascitic fluid may accumulate in the abdomen

Although some patients with cirrhosis are capable of prolonged survival, generally this diagnosis is an ominous one

Tumors Secontary

On a worldwide basis, primary malignant tumors of the liver, known as hepatocellular carcinoma are an important cause of cancer mortality

In the United States, these tumors are relatively uncommon. Most cases of hepatocellular carcinoma can be related to previous infection with a hepatitis virus.

معكن سخف مارعده كاسترفي مكان زي القولوذ أو البكر المور ومما Liver is frequently involved secondarily by tumors arising in other organs. Metastatic tumors to the liver from primary sites, such as the lung, pancreas, gastrointestinal tract, or ovary, are common. Benign tumors of the liver are relatively uncommon

Whether primary or secondary any malignant tumor in the liver is a serious finding with a poor prognosis mys in secontry 1 Primary 1

The only hope For cure relies on surgical resection, which is usually impossible. Patients with malignancies of the liver usually have a survival measured in months ياروب بعشوا الأسهو أقل

Reye's Syndrome of my house in techion wired in techion of the syndrome is a disorder of unknown cause, involving the liver and

arising primarily in children,

- It is a form of hepatic destruction that usually occurs following recovery from a viral infection, such as varicella (chickenpox) or influenza.
- It has been related to aspirin therapy. Shortly after the infection, the patient develops neurologic abnormalities, which may include seizures or coma
- Liver functions are always abnormal, but the bilirubin level is not usually elevated
- Without treatment, rapid clinical deterioration, leading to death, may occur

Drug- and Alcohol-Related Disorders\_ , Statin 11 The wall wish girls of Many drugs and chemicals are toxic to the liver. This toxicity may take the form of overwhelming hepatic necrosis, leading to coma and death, or it may be subclinical and pass entirely unnoticed In small amounts, alcohol may cause mild, inapparent injury. Heavier consumption leads to more serious damage, and prolonged, heavy use may lead to cirrhosis (exact amount is unknown) Certain drugs, including tranquilizers (phenothiazines), certain antibiotics, antineoplastic agents, and anti-inflammatory drugs, may cause liver injury Usually this is mild and manifested only by elevation of liver function tests, which return to normal when the drug is discontinued. This may lead to massive hepatic failure or cirrhosis The most common drug associated with serious hepatic injury is acetaminophen. When taken in massive overdose, it produces fatal hepatic necrosis unless rapid treatment is initiated Assessment Of Liver Function \*Analysis of Bilirubin (Method Selection) Unfortunately, no single method for the determination of bilirubin will meet all the requirements of the clinical laboratory

- For the evaluation of jaundice in newborns (no lipochromes), the direct spectrophotometric method is satisfactory)
- The sources of error in this technique are turbidity, hemolysis, and yellow lipochrome pigments
- Hemolysis and turbidity can be blanked out by measuring a second wavelength, but the yellow lipochromes cannot be blanked out
- In patients older than 1 month, a diazo-colorimetric procedure is necessary

### jendrassik-Grof Method for Total and Conjugated Billirubin Determination

- Serum or plasma is added to a solution of sodium acetate and caffeine-sodium benzoate, which is then added to diazotized sulfanilic acid to form purple azobilirubin
- The sodium acetate buffers the pH of the diazotization reaction, whereas the caffeine-sodium benzoate accelerates the coupling of bilirubin with diazotized sulfanilic acid
- This reaction is terminated by the addition of ascorbic acid which destroys the excess diazo reagent
- A strongly alkaline tartrate solution is then added to convert the purple azobilirubin to blue azo-bilirubin, and the intensity of the color is read at 600 nm
- A fasting serum specimen, which is neither hemolyzed nor lipemic, is preferred.
- The specimen should be kept in dark after collection, analyzed within 3hrs, kept for 1 week in the refrigarator or 3 months in -20

### Reference range

- Normal blood contains no conjugated bilirubin.
- Some conjugated bilirubin is reported as normal because current available methodology picks up some of the total bilirubin as a false positive

العلام Reagent على المساور ال

Conjugated: 0-0.2 mg/dL (0-3 µmol/L) to tal p (owinguted)) clude

Unconjugated: 0.2-0.8 mg/dL (3-14 µmol/L) was well with

■ Total: 0.2-1.0 mg/dL (3-17 µmol/L) كالموسن ( لاعتب كانوافكه Total: 0.2-1.0 mg/dL (3-17 µmol/L)

Reagents Solvents Solvents Solvents FULL TERM, TOTAL
FOR infants

1-6 mg/dL

2-6 mg/dL

24 hours 1-6 mg/dL 2-6 mg/dL

48 hours 6-8 mg/dL 6-7 mg/dL

3-5 days 10-12 mg/dL 4-6 mg/dL

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- The absorbance of bilirubin in serum at 455 nm is proportional to its concentration
- The serum of newborns does not contain lipochromes, such as carotene, that would increase the absorbance at 455 nm. The absorbance of hemoglobin at 455 nm is corrected by subtracting the absorbance at 575 nm.

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Error will be introduced if the buffer is turbid. Because the method depends on the extinction coefficient of bilirubin, all volumes must be accurate and cuvettes must be flat-surfaced, with a path length of exactly 1 cm

- This method is relatively insensitive to hemolysis, which is often present in specimens obtained from infants, due to difficulty in skin puncture technique
- > it is significantly affected by the presence of lipochromes and so cannot be used in infants older than a few months of age

### Urobilingen in Urine and Feces

- Urobilinogin is a colorless end product of bilirubin metabolism that is oxidized by intestinal bacteria to the brown pigment urobilin
- In the normal individual, part of the urobilinogen is excreted in the feces, and the remainder is reabsorbed into the portal blood and returned to the liver. A small portion that is not taken up by the hepatocytes is excreted by the kidney as urobilinogen
- Increased levels of urinary urobilinogen are found in hemolytic disease and in defective liver-cell function, such as hepatitis

Absence of urobilinogen from the urine and stool is most often seen with complete biliary obstruction. Fecal urobilinogen is also decreased in biliary obstruction and in hepatocellular disease

### Urobilinogen in Urine and Feces

Most quantitative methods for urobilinogen are based on the reaction of this substance with p-dimethylaminobenzaldehyde to form a red color.

Major improvements were made by using alkaline ferrous hydroxide to reduce urobilin to urobilinogen and added sodium acetate to eliminate interference from such compounds as indole

The use of petroleum ether rather than diethyl ether for the extraction of urobilinogen was introduced to help in the removal of other interfering substances

### Determination of Urine Urobilinogen (Semiquantitative)

Principle. Into

Urobilinogen reacts with p-dimethyl aminobenzaldehyde (Ehrlich's) (eagent) to form a red color) which is then measured spectrophotometrically.

Ascorbic acid is added as a reducing agent to maintain urobilinogen in the reduced state.

The use of saturated sodium acetate stops the reaction and minimizes the combination of other chromogens with the Ehrlich's reagent.

> Specimen unit la mil & main de la color de la color

A fresh 2-hour urine is collected which should be kept cool and protected from light.

### Sources of Error

- The results of this test are reported in Ehrlich units rather than in milligrams of urobilinogen because of interfering substances
- Compounds, other than urobilinogen, that may be present in the urine and react with Ehrlich's reagent include porphobilinogen, sulfonamides, procaine, and 5-hydroxyindoleacetic acid. Bilirubin will form a green color and, therefore, must be removed, as previously described

Fresh urine is necessary and the test must be performed without delay to prevent oxidation of urobilinogen to urobilin. Similarly the spectrophotometric readings should be made within 5 minutes after color production because the urobilinogen-aldehyde color slowly decreases in intensity.

Reference Range: Urine urobilinogen, 0.1-1.0 Ehrlich units/2 hr or 0.5-4.0 Ehrlich units/day (0.8 - 6.8 mmol/day); 1 Ehrlich unit is equivalent to approximately 1 mg of urobilinogen

### Fecal Urobilinogen

- Visual inspection of the feces usually suffices to detect decreased urobilinogen.
- The semiquantitative determination of fecal urobilinogen is available It is carried out in an aqueous extract of fresh feces, and any urobilin present is reduced to urobilinogen by treatment with alkaline ferrous hydroxide before Ehrlich's reagent is added
- A range of 75-275 Ehrlich units/100 g of fresh feces or 75-400 Ehrlich units per 24-hour specimen is considered a normal reference range.

### Measurement of Serum Bile Acids

عاليمة عيهاوا بمناة

- Unfortunately; complex methods are required for the analysis of bile acids in serum
- they involve extraction with organic solvents, partition chromatography; gas chromatography-mass spectroscopy spectrophotometry ultraviolet light absorption, fluorescence, radioimmunoassay and enzyme immunoassay methods
- Although serum bile acid levels are elevated in liver disease, the total concentration is extremely variable and adds no diagnostic value to other tests of liver function.

Enzyme Tests in Liver Disease

Any injury to the liver that results in cytolysis and necrosis causes the liberation of various enzymes.

The most common enzymes assayed in hepatobiliary disease include ALP and the aminotransferases.

Used less often are y-glutamyltransferase, lactate dehydrogenase (LD) and its isoenzymes, 5'-nucleotidase, ornithine carbamoyltransferase, and leucine aminopeptidase

### Enzyme Tests in Liver Disease

الارتفاع لاله حما من عادي إذا عنه ص

Alkaline Phosphatase: in the clinical diagnosis of bone and liver disease.

The most striking elevations occur in extrahepatic biliary obstruction, such as a stone in the common bile duct, or in intrahepatic cholestasis, such as drug cholestasis or primary biliary cirrhosis. This enzyme is almost always increased in metastatic liver disease and may be the only abnormality on routine liver function tests.

The enzyme is found in placenta, and pregnant women also have elevated levels وكعان مكون هرتفع علأ

Aminotransferases (Transaminases)

برتقعوا إدار AST and ALT are two enzymes widely used to assess hepatocellular damage AST is found in all tissue, especially heart, liver, and skeletal muscle.

ALT is more "liver specific"

Enzyme Tests in Liver Disease

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determine whether an ALP elevation is caused by liver or bone disease

This enzyme is much more sensitive to metastatic liver disease than is ALP because, unlike ALP, its level is not significantly elevated in other conditions, such as pregnancy or childhood

Some increase in its activity may occur after abdominal surgery

### Enzyme Tests in Liver Disease اد االديف عند صفيل كلوى مكون ١٥٥٦ محمر مع

serum of almost all patients with hepatobiliary disorders y-Glutamyltransferase (GGT) (high)in kidney and the liver and is elevated in the

consume large amounts of alcohol It is not specific for any type of liver disease but is frequently the first abnormal liver function test demonstrated in the serum of persons who

The highest levels are seen in biliary obstruction

Sensitive test for alcoholic liver disease

confirmation of hepatic neoplasms and to confirm hepatic disease in patients Measurement of this enzyme is also useful if jaundice is absent for the with elevated ALP

and kidney. Leucine Aminopeptidase) widely distributed in human tissue, is found in the pancreas, gastric mucosa, liver, spleen, brain, large and small intestine

differentiate hepatocellular from obstructive jaundice. The serum activity of leucine aminopeptidase cannot be used to

علوله ما اله دا كر نقيسه لأنه ما بغي The measurement of this enzyme does not provide any useful information

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## Enzyme Tests in Liver Disease

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the serum from various tissue injuries helpful diagnostically because LD is present in all organs and released into Lactate Dehydrogenase: Measurement of total serum LD is usually not

information about the site of origin of the LD elevation Fractionation of LD into its five tissue-specific isoenzymes may give useful

in a patient with jaundice D-5 is mostly present in liver and skeletal muscle. elevated LD-5 is noted

slight elevations Moderate elevations of total serum LD levels are common in acute viral hepatitis and in cirrhosis, whereas biliary tract disease may produce only

High serum levels may be found in metastatic carcinoma of the liver.

V

# Tests Measuring Hepatic Synthetic Ability

- The measurement of the end products of hepatic synthetic activity can be used to assess liver disease. Although these tests are not sensitive to minimal dystunction liver damage, they are useful in quantitating the severity of hepatic
- correlates well with the severity of functional impairment and is found more antitrypsin) tend to decrease with chronic liver disease often in chronic rather than acute liver disease. The serum a-globulins (almay be a result of decreased liver protein synthesis. The albumin level Most serum proteins are produced by the liver. A decreased serum albumin
- Serum y-globulin levels are transiently increased in acute liver disease and remain elevated in chronic liver disease. The highest elevations are found in chronic active hepatitis and postnecrotic cirrhosis.
- hepatitis, (IgM in primary biliary cirrhosis, and IgA in alcoholic cirrhosis. lgG and IgM levels are more consistently elevated in chronic active



### /Selow Tests Measuring Hepatic Synthetic Ability

they of thick

Prothrombin time is commonly increased in liver disease because the Witamin K from the intestine because the disruption of bile flow results in inadequate absorption of iver is unable to manufacture adequate amounts of clotting factor or

absorption of fat-soluble vitamins. synthesizing capacity from extrahepatic obstruction with decreased some value in differentiating intrahepatic disease with decreased Response of the prothrombin time to the administration of vitamin K is of

liver disease and a poor prognosis A marked prolongation of the prothrombin time indicates severe diffuse

# Tests Measuring Nitrogen Metabolism

The liver plays a major role in removing ammonia from the bloodstream and converting it to urea so in liver failure ammonia will increase leading to coma

In brain, glutamate react with ammonia to give glutamine which increases in CSF to cause encephalopathy

A cerebral spained shind

Hepatitis

inflammation of the liver, may be caused by viruses, bacteria, parasites, radiation, drugs, chemicals, or toxins.

Among the viruses causing hepatitis are hepatitis types[A, B, C, D (or delta),

and E, cytomegalovirus, Epstein-Barr virus, and probably several others.

or inapparent infection with no tendency to chronic disease. Hepatitis A is usually transmitted by the fecal/oral route and causes a mild

causes a serious illness in a minority of patients, however, in many patients, the infection is mild or even inapparent Hepatitis B and Care primarily transmitted parenterally) Hepatitis B

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### Hepatitis

- Acute infection with hepatitis C is usually mild to inapparent
- Hepatitis B has a slight tendency to chronic disease, while most patients with hepatitis C infection develop chronic infection.

\* ماني لقاع لمن ي ( c ) كرالان عنا

- Delta hepatitis is a unique satellite virus that causes a superinfection in patients already infected with hepatitis B.
- Hepatitis E is primarily transmitted by the fecal/oral route and causes serious disease only in pregnant women

ا عراف معلم و Chronic hepatitis is a major cause of morbidity and mortality worldwide

Chronic hepatitis is a major risk factor for the development of hepatocellular carcinoma

### Case study

### CASE STUDY 22-1

The following laboratory test results were obtained in a patient with severe jaundice, right upper quadrant abdominal pain, fever, and chills (Case Study Table 22-1.1).

### Question

1. What is the most likely cause of jaundice in this patient?

### CASE STUDY TABLE 22-1.1. LABORATORY RESULTS

Serum alkaline phosphatase	4 times normal
Serum cholesterol	Increased
AST (SGOT)	Normal or slightly in- creased
5'-Nucleotidase	Increased
Total serum bilirubin	25 mg/dL
Conjugated bilirubin	19 mg/dL
Prothrombin time	Prolonged but improves with a vitamin K injection

### Case study

### CASE STUDY 22-2

The following laboratory test results were found in a patient with mild weight loss and nausea and vomiting, who later developed jaundice and an enlarged liver (Case Study Table 22-2.1).

### Question

What disease process is most likely in this patient?

### CASE STUDY TABLE 22-2.1, LABORATORY RESULTS

Total serum bilirubin	20 mg/dL
Conjugated bilirubin	10 mg/dL
Alkaline phosphatase	Mildly elevated
AST (SGOT)	Significantly elevated
ALT (SGPT)	Moderately elevated
Albumin	Decreased
y-Globulin	Increased

- " سبحان الله ، الحمد تله ، الاولا الله , الله أجر " سبحان الله م المحدود الدولا الله ، الله م المؤمنان المسلمين والعسلمات الأصياء منهم والأموات ، " اللهم اغمز للعدّ منين والمؤمنان المسلمين والعسلمات الأصياء منهم والأموات ، "