



Internal Medicine



Subject: Hepatitis

Group no: C8

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هَذَا خَلَقَ اللَّهُ فَأَرْوِي مَاذَا خَلَقَ الَّذِينَ مِنْ دُونِهِ جَ بَلِ الظَّالِمُونَ فِي ضَلَالٍ مُبِينٍ

* **Hepatitis**: inflammation of hepatocytes caused by → viral, drug induced, ischemic...etc.
↳ leading to damage.

Viral Hepatitis

History Taking

When taking a history, always ask about:

- Risk factors (e.g., IV drug use, sexual exposure, blood transfusions, travel history)
- Maternal and family history (genetic predisposition, perinatal transmission)

General Features of Hepatitis Viruses

Feature	Description
Type of Virus	All hepatitis viruses are RNA viruses , except Hepatitis B (DNA virus) .
Other Causes	CMV, EBV, and Herpes can cause hepatitis, especially in immunocompromised patients.
Common Symptoms	Fatigue, jaundice, itching, elevated transaminases (ALT, AST), ALP, GGT, and bilirubin.

Direct cytopathic effect → Hep A, C, D, E
Immune-mediated injury → Hep B

Albumin vs PT/INR

acute loss of function/الكبد في وظيفة الكبد "حاد" في وظيفة الكبد

PT/INR الإجابة: ⚡

★ Why PT/INR reflects acute loss of liver function?

Because:

- Clotting factors (II, VII, IX, X) are synthesized in the liver.
- Half-life of Factor VII is ~6 hours → very short.
- So if the liver suddenly fails → PT/INR becomes prolonged within hours.

✓ PT/INR = best indicator of acute liver synthetic failure

✓ Used in acute liver failure criteria (King's College)

★ Why Albumin does NOT reflect acute loss?

- Albumin half-life = about 20 days
- Takes weeks to drop
- So albumin reflects chronic liver disease, not acute.

✓ Chronic liver disease → low albumin

✓ Acute liver failure → albumin still normal early on

ALD > AST But in Alcoholic hepatitis AST > ALT

Hepatitis A (HAV)

Overview

- Causative Agent: Hepatitis A virus (HAV) – a non-enveloped, **RNA virus** (genus Hepatovirus, family Picornaviridae).
- Transmission: Fecal-oral route (e.g., contaminated food, water).
- Incubation Period: 15-50 days (average 30 days).
- Infectivity: Patients are infectious up to 2 weeks after the onset of jaundice.
- Reservoir: **Humans** are the only known reservoir.

B is a dna virus

Clinical Course

*Stage	Key Events
Prodromal Phase	① Fatigue, loss of appetite, decreased smoking habits
Early Infection	Virus present in feces , ALT & AST begin to rise
Acute Phase	Jaundice appears, IgM anti-HAV develops
Resolution	Self-limiting in most cases; provides lifelong immunity

- Chronicity: **HAV does not cause chronic hepatitis.**
- Severe Cases: **Fulminant hepatic failure occurs in <1% of cases.**
- Immunity: Infection confers lifelong immunity and is **preventable by (va)**

vaccination (2 doses)
للمعانة منه

Diagnosis

Test	Result
* IgM anti-HAV antibody	Positive (indicates acute infection)

Extrahepatic Manifestations

System	Manifestations
Renal	Nephritis, glomerulonephritis (GN)
Skin	Rash
Joints	Arthralgia
Cardiac	Carditis

Management

Treatment	Details
* Supportive Care	Rest, hydration, symptom management (no specific antiviral treatment)

★ 1) GGT – Gamma-Glutamyl Transferase

GGT = موجود غالباً في إنزيم كبدى (Liver enzyme)

- Liver (hepatocytes)
- Bile ducts (canaliculi)
- Pancreas
- Kidney

🔥 استخدام مهم جداً / Very important clinical use

GGT + ALP together

	الحالة	ALP	GGT	التفسير
Liver source		↑	↑	من الكبد ALP
Bone disease		↑	Normal	من العظم ALP

يعني إذا ALP مرتفع و GGT طبيعي → المشكلة بالعظم مش بالكبد.

وظيفته / Function 🔥

• يساعد في نقل الـ glutathione خلال أغشية الخلايا.

- Sensitive indicator of cholestasis (انسداد أو مشكلة في القنوات الصفراوية).

متى يرتفع؟ / When does it rise 🔥

Very sensitive but not specific.

- ✓ Cholestasis – انسداد صفراوي
- ✓ Biliary obstruction
- ✓ Alcohol use (GGT لأنه يُحفَّز بالـ microsomal induction يرتفع مع شرب الكحول)
- ✓ Drugs liver enzyme induction (phenytoin, carbamazepine) تسبب
- ✓ Fatty liver
- ✓ Hepatitis

2) Bilirubin

هو ناتج تكسير الهيم (heme) من RBCs.

يوجد نوعين:

🟡 Unconjugated bilirubin (غير مباشر - غير مذاب)

(Indirect)

• غير مذاب في الماء

• مرتبط بالـ ألبومين

• قبل دخول الكبد

• يرتفع في:

- Hemolysis (تكسير دم)
- Gilbert syndrome
- Crigler–Najjar
- Liver uptake problem

🟡 Conjugated bilirubin (مباشر – مذاب)

(Direct)

• مذاب بالماء (water-soluble)

• بعد معالجة الكبد

• يرتفع في:

- Obstruction / Cholestasis
- Hepatitis
- Biliary atresia
- Dubin-Johnson / Rotor

★ مخطط سريع (Summary Table)

Test	High Means	Notes
GGT ↑	Liver disease, cholestasis, alcohol	Helps confirm ALP source
Bilirubin ↑ indirect	Hemolysis, Gilbert	Not water-soluble
Bilirubin ↑ direct	Obstruction, hepatitis	Water-soluble
ALP ↑ + GGT ↑	Liver/bile problem	Important combo
ALP ↑ + GGT normal	Bone problem	Very high-yield

Prevention



Method	Details
Vaccination	2 doses of HAV vaccine
Post-Exposure Prophylaxis	Depends on patient's age & risk factors:
1-40 years (healthy)	HAV vaccine within 2 weeks
>40 years, immunocompromised, or chronic liver disease	HAV vaccine + HAV immunoglobulin

Complications

Type	Description
Prolonged Cholestatic Hepatitis	Persistent cholestasis lasting >3 months after acute hepatitis. (jaundice)
Relapsing Hepatitis	Recurrent hepatitis episodes after complete resolution.

Hepatitis B (HBV) (important)

Overview

Feature	Description
Causative Agent	Hepatitis B virus (HBV)
Viral Type	Double-stranded DNA virus , belongs to the <i>Hepadnaviridae</i> family

Mode of Transmission

- Parenteral: Blood transfusion, needle sharing, IV drug use
- Sexual: Unprotected intercourse with an infected individual
- Perinatal: Mother-to-child transmission (most common in endemic areas)
- Others: Healthcare-related exposure, contaminated instruments

Acute Hepatitis B.

Incubation Period: 1-4 months.

Clinical Presentations

Presentation	Percentage (%)	Characteristics
Mild Subclinical (Anicteric)	70%	ALT, AST >1000 IU/L, asymptomatic or mild symptoms
Acute Icteric Hepatitis	30%	Jaundice, fatigue, nausea, vomiting, elevated LFTs
Acute Fulminant Hepatitis	<1%	Hepatic encephalopathy, coagulopathy, liver failure

Serologic Markers in Acute Infection

- HBsAg (Hepatitis B surface antigen) → First serologic

marker to appear

Recovery Rates:

- >95% recover completely
- <5% progress to chronic infection (>90% in children)

لأن كل ما انتابوا به أصبح كل ما زاد فرصة يصاب Chronic

Treatment of Acute Hepatitis B.

Management	Indications
Supportive care	Most cases resolve spontaneously
Antiviral Therapy Indications	Acute fulminant hepatitis (HE, INR >1.6), immunocompromised patients, coexisting HCV or other chronic liver diseases, protracted course (bilirubin >10 mg/dL for >4 weeks)
Interferon (INF)	Should be avoided

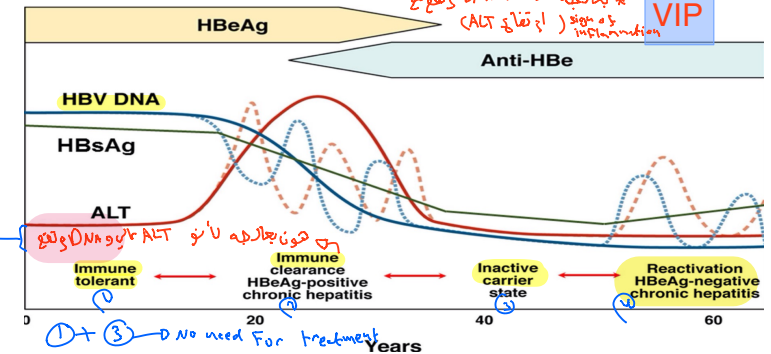
HBsAg (+) → means that the virus is in active state
e of Coantigen
لأنه في حالة العدوى النشطة

Chronic Hepatitis B

Diagnosis Criteria : HBsAg positive for >6 months

Stages of Chronic Hepatitis B

Stage	Characteristics	Treatment Recommendation
Immune Tolerant	High HBV DNA, normal ALT, no inflammation	No treatment
Immune Active (HBeAg Positive/Negative)	High HBV DNA, ALT >2x ULN, inflammation	Treat if evidence of inflammation
Inactive Carrier	Low/undetectable HBV DNA, normal ALT	No treatment
Cirrhosis (Compensated/Decompensated)	Fibrosis/cirrhosis, risk of HCC hepatocellular cancer	Treat regardless of HBV DNA level



Screening & Prevention

Test	Indication
HBsAg, HBsAb, HBeAb ↳ surface	Screening for suspected HBV infection
HBsAb-negative individuals	Vaccinate if no chronic infection

HBeAg Significance

- HBeAg Positive → Active viral replication (high infectivity)
- HBeAg Negative → Low replication or mutation

Hepatocellular Carcinoma (HCC) Risk

Can develop HCC even without cirrhosis in chronic HBV pts

Treatment of Chronic Hepatitis B

Indication for Treatment	Criteria
Immune Tolerant / Inactive Carrier	No treatment recommended only follow up
Active Infection	HBV DNA high + ALT ⁽¹²⁾ elevation + inflammation ↳ hepatocyte destruction → risk for biopsy
Cirrhosis	Treat regardless of HBV DNA level ↳ HBsAg > 20000 ↳ Anti-HBe > 2000

First-Line Therapy for Chronic HBV

Drug	Class
Entecavir	Nucleoside analogue
Tenofovir	Nucleotide analogue

VIP

- Goal of Therapy → HBV DNA undetectable by week 48

لأنه إذا كان HBV DNA غير قابل للكشف بعد 48 أسبوعاً

وجدوا إنه المرض الذي عدم Cirrhosis وال DNA عالي حينئذٍ عدم decompensation

↑ % complications

VIP

HBsAg Anti-HBs Anti-HBc

يُنصَحهم يأخذوا vaccine
Susceptible (Never exposed)

Vaccinated	Negative	Positive	Negative
Past Infection	Negative	Positive	Positive
Acute Infection	Positive	Negative	IgM Positive
Chronic Infection	Positive	Negative	IgG Positive

Chronic Hepatitis B & Pregnancy

Management Approach

Step	Action
Measure HBV DNA at weeks 26-28 (2nd trimester)	
If HBV DNA >200,000 IU/mL or previous child was HBV-positive	Start Tenofovir treatment
If HBV DNA is low and no previous HBV-positive child	Observe without treatment

Indications for HBV Treatment in Pregnancy

- Advanced fibrosis & cirrhosis
- Active HBV infection with high viral load & elevated ALT

HBsAg (+)	Infection is present (acute or chronic).
Anti-HB_s (+)	Immunity from past infection or vaccine.
Anti-HBc IgM (+)	Recent/acute infection.
Anti-HBc IgG (+)	Past infection (not vaccine-induced). / <i>chronic</i>
HBeAg (+)	High infectivity , active replication.
Anti-HBe (+)	Lower infectivity, immune control.

Neonatal HBV Transmission

During Delivery : >95% of cases

Prevention of Neonatal HBV Infection

- Newborn should receive:
- Hepatitis B immunoglobulin (HBIG)**
- HBV vaccination**
- Cesarean Section (CS) is NOT recommended**

Occupational Exposure to Hepatitis B

Status of Healthcare Worker	Management
Unvaccinated	HBIG + HBV vaccine
Vaccinated, but non-responder	HBIG + repeat vaccine series
Vaccinated, responder	No intervention needed

Recommended PEP for Hepatitis B Virus:

Vaccination/Ag response status of exposed patient	Treatment when source patient is:		
	HBsAg positive	HBsAg negative	Source unknown or not available for testing
Unvaccinated/non-immune	HBIG × 1; initiate HB vaccine series	Initiate HB vaccine series	Initiate HB vaccine series
Previously vaccinated, known responder	No treatment	No treatment	No treatment
Previously vaccinated, known non-responder	HBIG × 1 and initiate revaccination or HBIG × 2	No treatment	No treatment unless high-risk source; if high-risk source, treat as if source were HBsAg positive
Previously vaccinated, response unknown	Single vaccine booster dose	No treatment	No treatment unless high-risk source; if high-risk source, treat as if source were HBsAg positive
Still undergoing vaccinated	HBIG × 1; complete series	Complete series	Complete series

HIV / HBV Co-Infection

Patient Status	Recommended Treatment
Not on HAART & not starting soon	Pegylated INF- α or Adefovir
Starting HAART & HBV treatment	Lamivudine + Tenofovir or Emtricitabine + Tenofovir
Already on HAART	Add Pegylated INF-α or Adefovir

Key Takeaways

- HBsAg is the first marker to appear in acute infection.
- >95% of acute infections resolve spontaneously, but <5% progress to chronic disease.
- Children have a higher risk of developing chronic HBV (>90%).
- First-line treatment for chronic HBV: Entecavir or Tenofovir.
- Pregnant women with high viral loads should be treated with Tenofovir.
- Neonates require HBIG & vaccination to prevent transmission.
- HBV can cause HCC even in the absence of cirrhosis.

Hepatitis C (HCV)

Overview

Feature	Description
Causative Agent	Hepatitis C virus (HCV)
Viral Type	RNA virus , belongs to the <i>Flaviviridae</i> family
Most Common Cause (MCC)	Chronic hepatitis & cirrhosis in the USA

Screening & Diagnosis ↳ it also can lead to HCC.

Test	Purpose
HCV-Ab (ELISA)	Screening test
HCV RNA PCR	Confirmatory test if HCV-Ab positive

HCV RNA + / Anti-HCV - → early infection

No RNA / Anti-HCV + → Past infection or resolved

Acute vs Chronic HCV

Stage	Outcome
Acute Infection	Most cases progress to chronic infection
Chronic Infection	Persistent HCV RNA for >6 months

- 15-25% of acute cases resolve spontaneously within 6 months → No need for treatment in this period

Vaccination in HCV Patients

- All HCV patients should receive standard adult vaccinations, including:
- Hepatitis A (HAV) vaccine if not immune
- Hepatitis B (HBV) vaccine if not immune

it has no vaccine

Goal of Therapy

- Sustained Virologic Response (SVR) at 6 months = Undetectable HCV RNA
- SVR is considered a cure

VIP

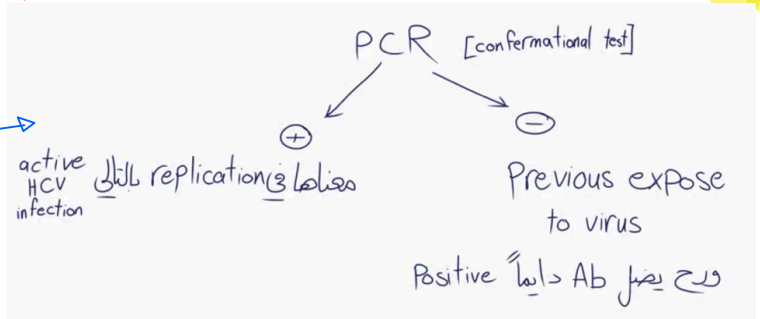
Treatment of HCV

Patient Group	Recommended Treatment
All chronic HCV patients	Direct-acting antivirals (DAAs) (curable)
Common DAA Regimens	Sofosbuvir + Velpatasvir OR Glecaprevir + Pibrentasvir
Treatment Duration	8-12 weeks

هم في أسئلة البورد

⚠️ وجود HcAb (+) ← صحت سبوتا يكون هشاشا (current infection) ممكن يكون (Previous infection)

*common scenario → واحد راح يشرح بالدم لغزته ورن عليه المختبر حرك له عندك
 الـ HcAb مزينة شوال الجراد؟؟ بشوف الـ PCR + Liver function test



HCV/HBV Co-Infection Management

Patient Status	Management
Already on HBV therapy	Continue treatment
Meets HBV treatment criteria	Start HBV therapy before DAAs and continue indefinitely
Does NOT meet HBV treatment criteria	Start HBV therapy before DAAs and continue 12 weeks after finishing DAAs

Occupational Exposure to HCV

Risk of Transmission	~3% after accidental percutaneous exposure
Baseline testing	LFTs + HCV-Ab <i>Liver Function Test</i>
4-6 weeks post-exposure	HCV RNA
4-6 months post-exposure	LFTs, HCV-Ab, HCV RNA
Acute infection (first 6 months)	No need for immediate treatment (15-25% clear spontaneously)

HCV & Sexual Transmission

Scenario	Risk
Monogamous couples	Very low risk (~1/190,000 per sexual contact)
High-risk groups (MSM, multiple partners, HIV+ individuals)	Higher risk

Recommendation:

- For monogamous couples → No need to change sexual practices (1/190,000)
- For high-risk individuals → Use barrier protection (e.g., condoms) *let the patient take the decision.*

Key Takeaways

- ✓ HCV is the most common cause of chronic hepatitis & cirrhosis in the USA
- ✓ HCV is mainly transmitted through blood exposure (IV drug use, transfusion, healthcare exposure)
- ✓ All patients should be screened with HCV-Ab, and confirmed with HCV RNA if positive
- ✓ 15-25% of acute infections clear spontaneously → Treatment is not needed in the first 6 months
- ✓ Goal of treatment is achieving SVR (undetectable HCV RNA at 6 months)
- ✓ First-line treatment: Direct-acting antivirals (DAAs) for 8-12 weeks
- ✓ All HCV patients should receive HAV & HBV vaccination if not immune
- ✓ Sexual transmission is very low in monogamous couples but higher in high-risk groups

Hepatitis D (HDV)

Overview

Feature	Description
Causative Agent	Hepatitis D virus (HDV)
Viral Type	Defective RNA virus (requires HBV for replication)
Modes of Acquisition	Co-infection with HBV or Superinfection in chronic HBV patients

Clinical Presentation

Type of Infection	Outcome
Co-infection with HBV <i>→ 2% lead to chronic hepatitis</i>	Symptoms similar to acute HBV
Superinfection in chronic HBV patients <i>→ Bad prognosis</i>	>90% develop chronic hepatitis with rapid progression to cirrhosis

Complications

- More severe liver disease than HBV alone
- High risk of cirrhosis & liver failure

Diagnosis

Test	Purpose
IgM anti-HDV antibody	Screening test
HDV RNA	Confirmatory test

Treatment

Indication	Treatment of Choice
HDV RNA positive & elevated ALT	Pegylated Interferon-alpha (Peg-INF α) for 12 months

Hepatitis E (HEV)

Overview

Feature	Description
Causative Agent	Hepatitis E virus (HEV)
Viral Type	Non-enveloped RNA virus (Hepeviridae family)
Host Range	Swine, cats, rats, humans
Transmission	Fecal-oral route
Incubation Period	~60 days

سؤال متوقع



Clinical Course

Patient Group	Course
General Population	Self-limited disease
Pregnant Women	High risk of fulminant hepatic failure (up to 20% mortality)
Immunocompromised Patients	Can lead to chronic infection

Neurological Complications

- 30-40% of cases associated with neurological disorders such as:
- Neuropathy
- Dementia

Autoimmune Hepatitis (AIH) The pt came with fatigue, joint pain, Amenorrhea

Overview

Feature	Description
Definition	Chronic inflammatory liver disease with autoantibodies & specific histologic findings
Key Histological Feature	Interface hepatitis (inflammatory infiltrate at portal tract)

Types of Autoimmune Hepatitis

Type	Associated Autoantibodies
Type 1 (Classic AIH)	ANA, SMA (Smooth Muscle Antibody)
Type 2 (More Severe, Rare)	LKM-1 (Liver-Kidney Microsomal Antibody)



Diagnosis

Test	Findings
IgG Levels	Elevated
Liver Biopsy	Interface hepatitis (key histological feature)

Treatment

Indication	Treatment
Confirmed AIH	Corticosteroids ± Azathioprine

Key Takeaways

- ✓ HDV requires HBV for replication and causes severe liver disease
- ✓ Superinfection with HDV in chronic HBV patients leads to cirrhosis in >90%
- ✓ HEV is fecal-oral, usually self-limited, but deadly in pregnant women (~20% mortality)
- ✓ 30-40% of HEV cases are associated with neurological disorders
- ✓ Autoimmune hepatitis is diagnosed by elevated IgG, autoantibodies, and liver biopsy
- ✓ Treatment of AIH involves corticosteroids ± azathioprine

* Acetaminophen induced hepatitis (toxic drug) ip # Antidote # → IV N-acetylcysteine (MCQ)

* Fulminant hepatitis

1. Pt with acute hepatitis, encephalopathy, coagulopathy ↑PT/INR.
2. Causes :
 - a- Hep → B, D, E especially in pregnant.
 - b- acetaminophen.
 - c- Wilson disease (young + hemolysis + liver failure)

Hepatitis A

KEY FEATURES

- ✓ Aka, "Infectious"
- ✓ (+) RNA
- ✓ Picornavirus
- ✓ Naked icosahedral capsid

TRANSMISSION ROUTE

- ✓ Fecal-Oral
- Shellfish

ONSET

- ✓ Rapid (~ 4 weeks)

CLINICAL COURSE

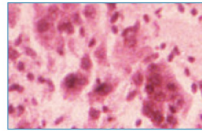
- ✓ Common, usually benign.
- ✓ More severe in adults.
 - Acute **icteric** hepatitis w/ immune-mediated damage.
- ✓ Fulminant is rare.

PREVENTION/TREATMENT

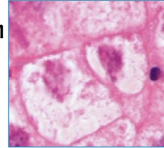
- ✓ Vaccine
- ✓ Self-limiting; Supportive care
- ✓ Antibody (IgG) provides life-long immunity.

HISTOPATHOLOGY

- ✓ Monocytes
- ✓ Councilman bodies (eosinophilic, shrunken)



- ✓ Ballooning degeneration



SEROLOGICAL VALUES

- ✓ Acute = anti-HAV IgM
- ✓ Previous = anti-HAV IgG

Hepatitis B

Virion = "Dane particle"

- Surface antigen HBsAg
- Envelope antigen HBeAg
- Core antigens HBcAg, HBcAg



CLINICAL COURSE(S)

- ✓ Acute w/ Resolution
 - Adults more symptomatic
 - Serum-sickness like
- ✓ Chronic w/ liver destruction
 - **Cirrhosis, liver failure**
 - **Primary hepatocellular cancer**
- ✓ Extrahepatic (uncommon)
 - Glomerulonephritis
 - Polyarthrititis
 - Polyarteritis nodosa
 - Guillain-Barré

SEROLOGICAL VALUES

- ✓ HBV infection = HBsAg
 - Acute = IgM (& HBeAg)
 - Chronic = IgG
- ↑ Infectivity = HBeAg
- ↓ Infectivity = Anti-HBe
- ✓ Replication/Infectious = HBeAg
- ✓ Recovery = Anti-HBs; IgG
- ✓ Immunized = Anti-HBs

KEY FEATURES

- ✓ Aka, "Serum"
- ✓ DNA
- ✓ Hepadnavirus
- ✓ Enveloped icosahedral capsid
- ✓ HBsAg outnumber virions in serum

TRANSMISSION ROUTE

- ✓ Blood, tissue, semen (IVD, Birth, STI)

- ✓ Carrier state.

ONSET

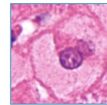
- ✓ Slow (months)

PREVENTION/TREATMENT

- ✓ Vaccine
- ✓ Hepatitis B immune globulin post-exposure prophylaxis
- ✓ Chronic: entecavir, tenofovir, others.

HISTOPATHOLOGY

- ✓ Chronic: Ground glass hepatocyte



Hepatitis C

KEY FEATURES

- ✓ Aka, "Non-A, Non-B"
- ✓ (+) RNA
- ✓ Flaviviridae
- ✓ Enveloped icosahedral capsid

TRANSMISSION ROUTE

- ✓ Blood*, tissue, semen (Contaminated needles)
- ✓ Carrier state.

ONSET

- ✓ Slow (months/years)

PREVENTION/TREATMENT

- ✓ No vaccine.
- ✓ Glecaprevir, Paritaprevir, others.

HISTOPATHOLOGY

- ✓ Cholestasis
- ✓ Necroinflammation
- ✓ Lymphocyte aggregation
- ✓ Fibrosis

CLINICAL COURSE(S)

- ✓ Acute w/ Resolution
- ✓ Rapid onset cirrhosis
- ✓ Chronic - Majority
 - Initially asymptomatic
 - **Cirrhosis, liver failure**
 - **Primary hepatocellular cancer**
- ✓ Alcohol exacerbates damage
- ✓ Extrahepatic (Common)
 - Mixed cryoglobulinemia vasculitis (Cryovas)
 - B-cell non-Hodgkin's lymphoma
 - Membrano-proliferative glomerulonephritis
 - Diabetes mellitus
 - Atherosclerosis
 - Thyroid disease

SEROLOGICAL VALUES

- ✓ anti-HCV, HCV RNA

Hepatitis D

KEY FEATURES

- ✓ Aka, "Delta Agent"
- ✓ (-) RNA
- ✓ Deltavirus
- ✓ Enveloped icosahedral capsid
- ✓ "Defective" - relies on HBV
- ✓ Increases severity of HBV infection.

TRANSMISSION ROUTE

- ✓ Blood, tissue, semen (IVD, STI)

PREVENTION/TREATMENT

- ✓ HBV Vaccine
- ✓ Interferon

SEROLOGICAL VALUES

- ✓ HDV RNA

Envelope from HBV surface proteins (HBsAg)

HBV Co-Infection

- ONSET
- ✓ Slow
 - HDV can't replicate until HBV establishes infection.

CLINICAL COURSE(S)

- ✓ Mild -> Severe
- Usually Acute
- Self-limiting
- ✓ Can be fulminant
 - Encephalopathy, hepatic necrosis. **Fatal.**

HBV Super-Infection

- ONSET
- ✓ Rapid
 - HBV has already established infection, so HDV replicates immediately.
- CLINICAL COURSE(S)
 - ✓ Exacerbation of HBV symptoms.
 - ✓ Can be fulminant

Hepatitis E

KEY FEATURES

- ✓ Aka, "Non-A, Non-B Enteric, Epidemic"
- ✓ (+) RNA
- ✓ Hepevirus
- ✓ Enveloped icosahedral capsid

TRANSMISSION ROUTE

- ✓ Fecal-oral
- Water

ONSET

- ✓ Rapid

PREVENTION/TREATMENT

- ✓ Sanitation
- ✓ Supportive care

SEROLOGICAL VALUES

- ✓ HEV RNA, anti-HEV

CLINICAL COURSE(S)

- ✓ Acute hepatitis w/ Resolution
- ✓ High mortality in pregnancy (esp. 3rd trimester)
 - Fulminant Encephalopathy, hepatic necrosis. **Fatal.**
- ✓ Genotype 3 Chronic?
 - Immunosuppressed

Young + traveller + fever + jaundice + dark urine → Hep A

IV drug user + no vaccination + ↑ALT + Anti HCV + → Hep C

Pregnat + 3rd tri + Acute Liver Failure → Hep E

HbsAg +
 HbeAg +
 ALT normal

→ Chronic Hep B, high infectivity

HbsAg -
 Anti-Hbs +
 Anti-Hbc +

→ Recovered hep B infection