# Paediatric Gastroenterology

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# ACUTE HEPATITIS

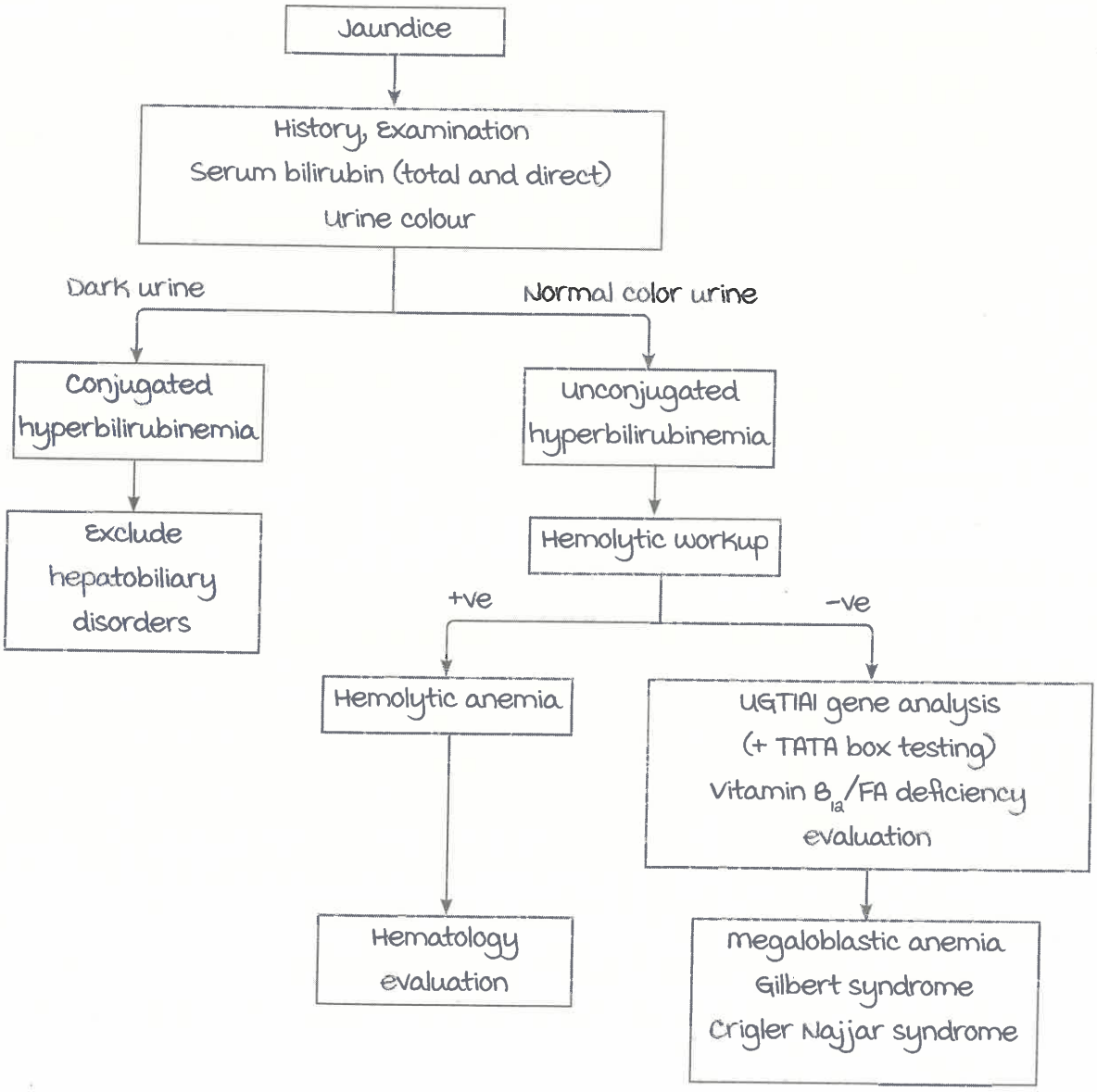
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## Introduction

00:00:21

### Clinical features :

- Jaundice (Icterus).
- Yellow discoloration of eyes, skin & mucous membranes.
- Urine, stool colour :
  - Dark urine + pale stools : Hepatic origin.
  - Normal urine colour : Unconjugated jaundice.



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Unconjugated hyperbilirubinemia with negative hemolytic features :

- Gilbert syndrome :
  - Presents in older child.
  - Normal LFT.
  - Exacerbated in events of stress.
- Crigler Najjar syndrome :
  - Younger age group.
  - Type I and type 2.
  - may respond transiently to phenobarbitol.
  - Definitive mx : Liver transplant.

## Jaundice due to liver dysfunction :

Acute jaundice :

- Viral hepatitis.
- Drug induced.
- Systemic infections.
- Biliary sepsis (Cholangitis).

Chronic liver disease :

- Wilson disease.
- Autoimmune liver disease.
- Viral hepatitis (B > C).
- Cholestatic disorders.
- Budd-Chiari syndrome.

Acute infective hepatitis :

| Hepatotropic | Non Hepatotropic |
|---|---|
| • Hepatitis A<br>• Hepatitis B<br>• Hepatitis C<br>• Hepatitis D<br>• Hepatitis E | • Dengue<br>• Parvovirus B19<br>• Cytomegalovirus, Epstein Barr virus<br>• Herpes viruses (HSV 1 & 2)<br>• Others : VZV, Adeno, HHV etc |

Note :

- Hepatitic C : Allmost never presents like acute hepatitis.
- Hepatitis D : Rare in children and occurs only if there is an underlying Hep B infection.

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| Study | HAV | HEV | HBV | Co-infection | Others |
|---|---|---|---|---|---|
| Panda et al Delhi, 1989 (n=456) | 55.8 % | - | 20.2 % | - | 23.2 % (NANB) |
| Malathi et al Chennai, 1998 (n=127) | 38.6 % | 15.7% | 13.4 % | 21.3 % | - |
| Poddar et al, Chandigarh, 2002 (n=172) | 64.5 % | 16.3% | 7.6 % | 8 % | - |
| Srivastava et al, Lucknow, 2012 (n=130) | 53.1 % | 6.9% | 13.8 % | 26.2 % | - |

Summary :

- Hepatitis A : 38-65 %.
- Hepatitis E : 7-16 %.
- Hepatitis B : 8-20 %.
- Co-infection : 8-26 % (Also in enteric fever).
- Unknown : 23 % → 11 % → 3.5 % → 0 %.

Note :

- Always exclude HAV, HEV, HBV in all AVH cases.
- Anti HCV : Silent/chronic cases.
- Complete evaluation in all : Co-infection is possible.
- Test for NHVs (Serology/PCR) :
  - If acute liver injury + negative classical serology.
  - Choose as per clinical picture.
- in case of acute hepatitis B, ask for anti HBc IgM.

## Viral hepatitis

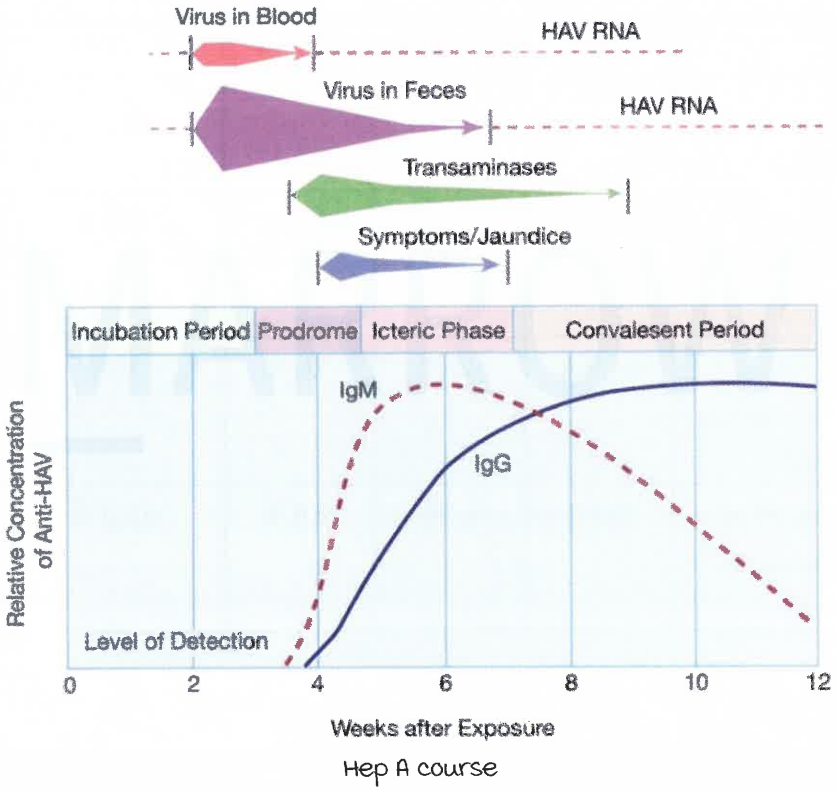
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### Typical hepatitis A :

Work up :

- LFT (Transaminases ↑ upto 10 times the normal).
- PT-INR.
- USG abdomen.

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Hep A course

## Etiological work up :

All patients :

- IgM HAV.
- IgM HEV.
- HbsAg, IgM Anti Hbc.
- Anti HCV : Only in chronic cases.

Selected patients/if above negative :

- IgM CMV/EBV/VZV/HSV/dengue/parvo/adeno virus.

## Differential diagnosis of acute jaundice :

| With persistent/prolonged fever (After Jaundice appears) | Without fever |
|---|---|
| • Malaria/typhoid/leptospirosis<br>• Biliary sepsis (Cholangitis)<br>• Liver abscess (Rarely) | • Wilson disease<br>• Autoimmune Liver Disease<br>• Drugs/toxins :<br>– ATT drugs<br>– Herbal medicines<br>– Paracetamol<br>– Valproate/other Antiepileptics<br>– Amox-Clavulanic Acid |

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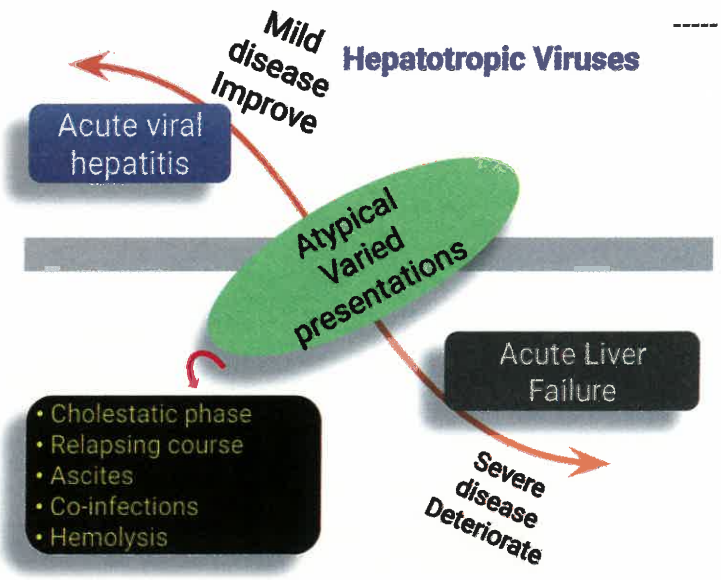
## Spectrum of hepatitis A :

Complicated :

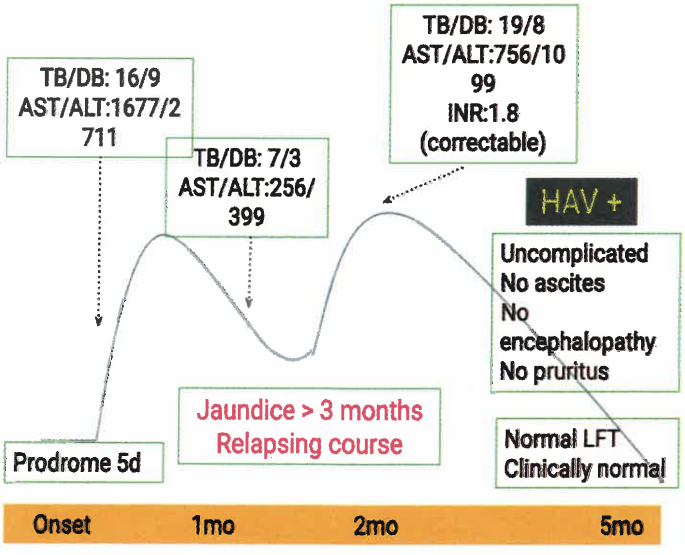
- Hyperacute liver failure.
- Acute liver failure.
- Late onset hepatic failure.
- Acute on chronic liver failure.

Atypical (30%) :

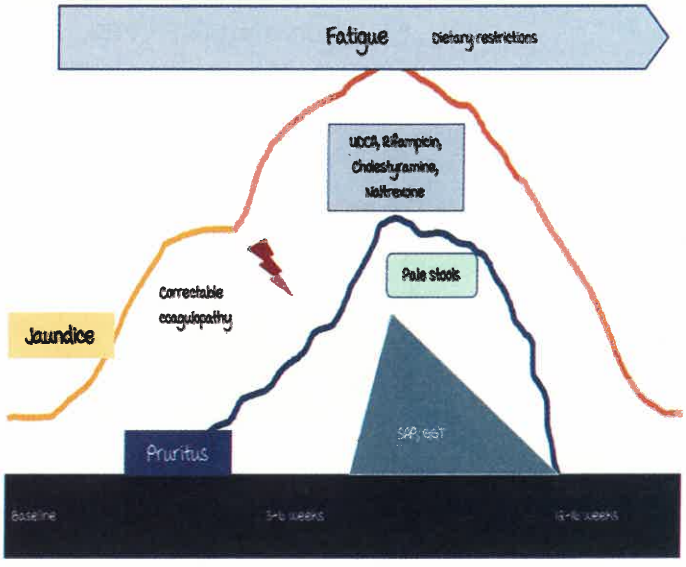
- Hepatic :
  - Prolonged cholestasis.
  - Relapsing hepatitis.
  - Ascitic form.
  - Autoimmune trigger.
- Extra hepatic :
  - intravascular hemolysis, aplastic anemia.
  - Immune thrombocytopenia.
  - HLH, Guillian-Barre syndrome.
  - Transverse myelitis.
  - Glomerulonephritis.
  - myocarditis, myositis.




Spectrum of Hep A

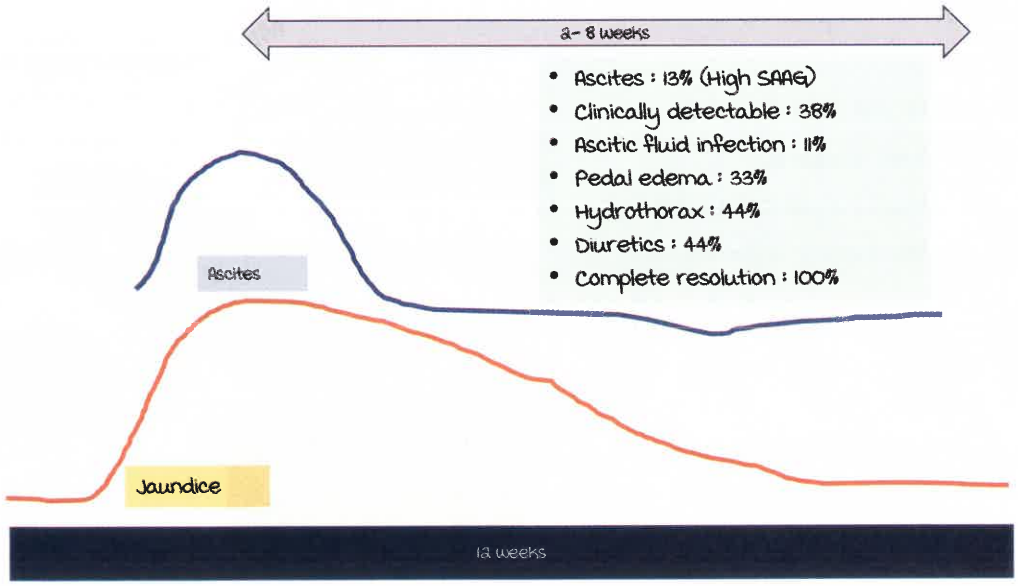



Hep A disease course




Prolonged cholestasis

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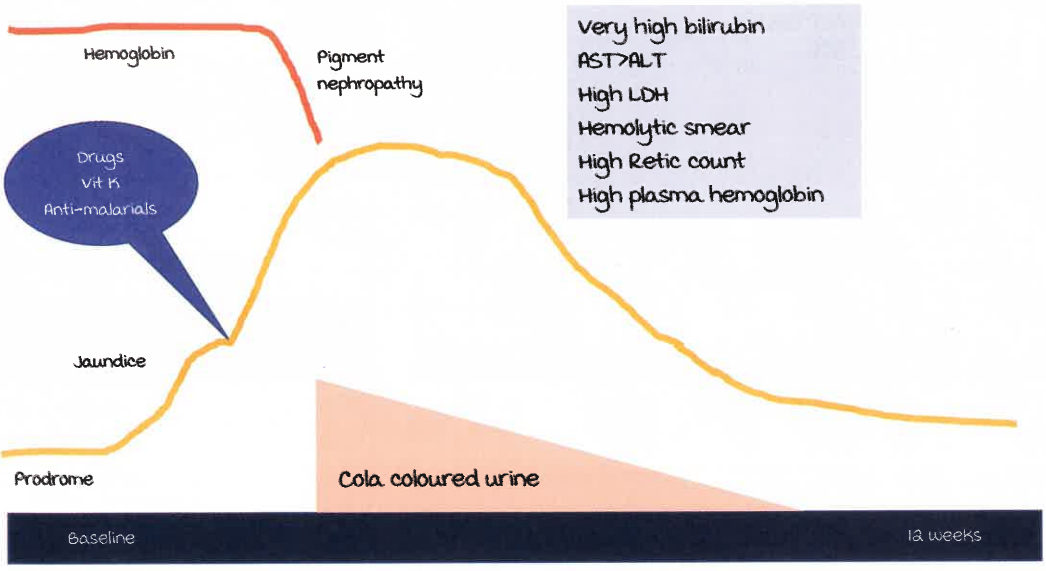

AVH with ascites

Late onset hepatic failure : 5-12 weeks (↑ Chance of ascites & encephalopathy).

## Intravascular hemolysis :

Causes :

- G6PD deficiency : 36%.
- HAV associated autoimmune hemolysis.
- Presenting feature in 6.7% cases of Wilson disease.
- in all Wilson disease : 22%.
- Wilson with ALF : 68%.
- Autoimmune hemolysis in AIH : 35%.




intravascular hemolysis

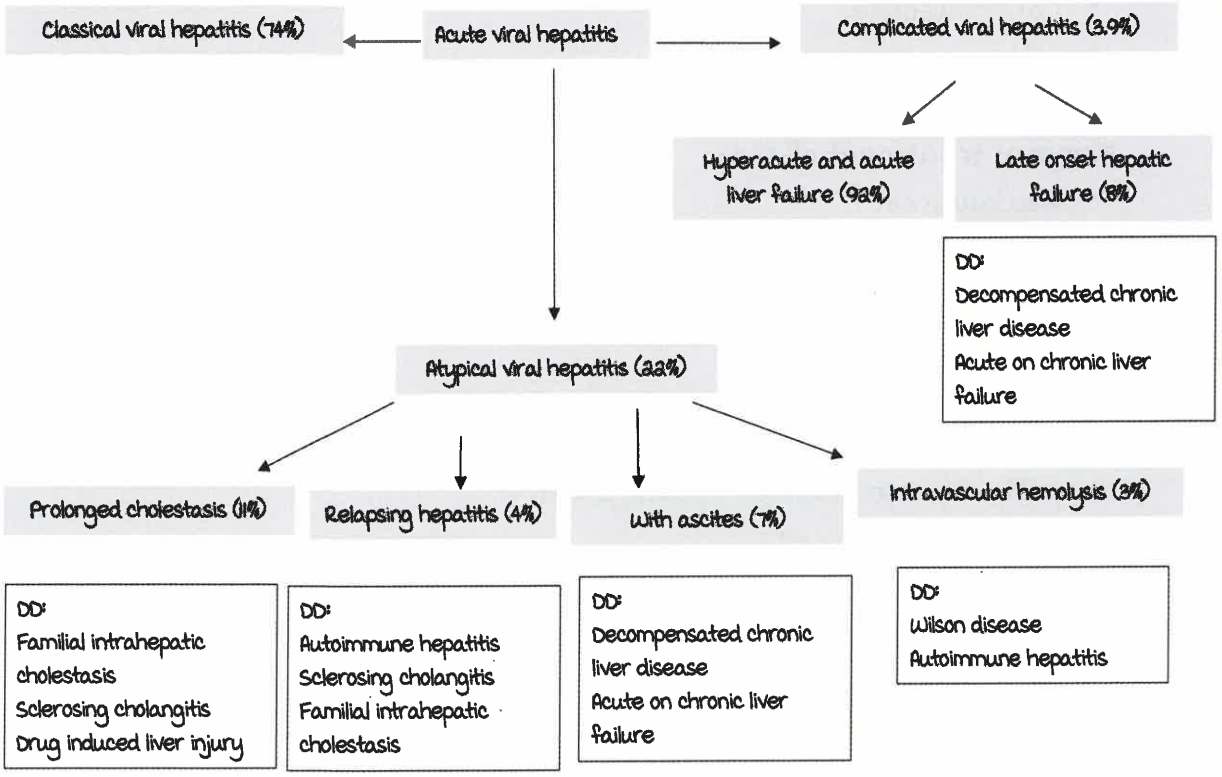
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Hematological manifestations of acute viral hepatitis :

- Aplastic anemia : 5-11% Present as hepatitis associated aplastic anemia.
- HAV : < 3% cases.
- Hepatitis to aplastic anemia : 62 days.
- Adolescent males.
- CD8 T cell mediated BM suppression.
- Good outcome with BM transplantation.
- Anti-thymocyte globulin, cyclosporin.
- HLH.
- Immune thrombocytopenia.
- IgG glycoprotein IIb/IIIa.
- Thrombocytopenia : 2.6%.
- Leukopenia : 16%.

Other atypical manifestations :

- Guillian-Barre syndrome.
- Mononeuritis multiplex.
- Transverse myelitis.
- Myasthenia gravis.
- Cardiomyopathy.
- Myositis.
- Acute pancreatitis.
- Glomerulonephritis.
- Cryoglobulinemic vasculitis.
- Antiphospholipid antibody syndrome.
- SLE.




AVH and DDs

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### Tropical infection :

Present like viral hepatitis.

- High grade persistent fever (Look for HLH also).
- A/w :
  - Severe anemia.
  - Conjunctival redness, oliguria.
  - Skin rash, plasma leak.
- Labs :
  - Thrombocytopenia.
  - Minimal to mild icterus.
  - AST > ALT, < 400-500 IU/L.
  - High CPK, LDH.

Note :

Charcot's triad : Jaundice with high colored urine, right hypochondrial pain, fever with chills.

Differential diagnosis of cholangitis :

- Choledochal cyst.
- CBD stones.
- Sclerosing cholangitis.
- Caroli's disease.
- Biliary ascariasis.

### General treatment of AVH :

- Reassurance.
- Normal diet, plenty of fluids.
- No medications needed (in majority).
- No restriction on diet.
- No bed rest needed.

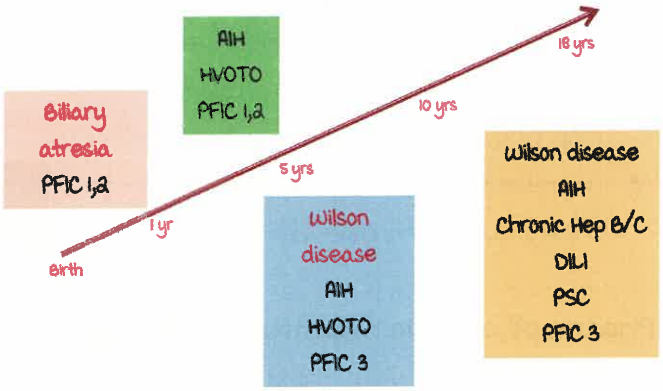
Suspect underlying CLD with acute jaundice :

- Prolonged history, FTT.
- Positive family or past history of liver disease.
- Decompensation (Ascites, encephalopathy).
- Firm organomegaly, CLD stigmata.
- USG : Coarse echotexture, nodularity, collaterals, splenomegaly.

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### CLDs with jaundice :

- Viral hepatitis : HBV.
- Autoimmune liver disease :
  - AIH.
  - Overlap.
  - PSC.
- Vascular disorders : Budd-Chiari syndrome.
- Cholestatic diseases :
  - Biliary atresia.
  - Progressive familial intrahepatic cholestasis (PFICs).
  - Alagille syndrome
  - Fibrocystic diseases.
- Drug induced/toxic liver injury.



Age wise distribution of CLDs

### Etiological pointers :

- Viral Hepatitis : Previous BT/injection use/tattooing/surgical procedures, family history.
- Wilson's disease :
  - Recent change in behaviour/school performance/handwriting.
  - increasing pallor, cola coloured urine (Hemolysis), family history.
- Autoimmune disease.
- Recurrent jaundice, rash, joint pains, redness of eyes, increasing pallor, cola coloured urine.
- Cholestatic liver disease : Pruritus/jaundice.
- Vascular disease : Ascites + jaundice.

## Hepatitis B 00:39:13

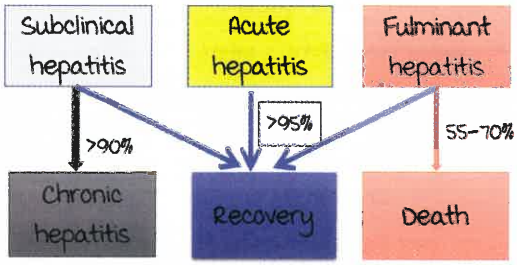
### HBV infection in children :

| Age of infection | Rate of persistent infection |
|---|---|
| Perinatal period(mother HBsAg+)<br>HBeAg +<br>HBeAg - | > 90%<br>20%, (Risk of FHF in precore mutant mother) |
| Preschool age (1–5 yr) | 20–30% |
| School aged (5–15 yr) | 6% |
| Adult | 1 – 5% |

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## Pathogenesis of HBV :

- interaction of HBV with host immune system and causing damage to hepatocytes.
- manifestation varies depending on the maturity of immune cells (Age).




Natural history of HBV infection

## Phases of chronic hepatitis B infection :

Depending on the virus-host interaction.

1. Immune tolerance : HBeAg positive infection.
2. Immune clearance : HBeAg positive hepatitis.
3. Low or non replication phase : HBeAg negative infection.
4. Phase IV : HBeAg negative hepatitis.

### Immune tolerance phase :

- Characteristics :
  - Presence of HBe-Ag antigen.
  - High HBV-DNA.
  - Normal or minimally elevated ALT level.
  - Liver Bx : Normal liver histology or minimal histological activity or scanty fibrosis.
- Persist for 10 to 30 years in perinatally acquired hepatitis B.
- Short-lived or absent in horizontally acquired hepatitis B.

### Immune clearance phase :

- HBe-Ag positive.
- HBV-DNA levels : Fluctuating but progressively decreasing.
- Serum HBV-DNA : > 20,000 IU/ml (105 copies/ml).
- Elevated ALT.
- Liver biopsy : moderate or severe necro-inflammation with variable amounts of fibrosis.
- most of these patients acquire hepatitis B in late childhood, adolescence, or adulthood.

### Low or non-replication phase : inactive carrier.

- HBeAg negativity and anti-HBe positivity.
- HBV-DNA : Undetectable or low (< 2000 IU/ml, 104 copies/ml).
- Persistently normal ALT levels.
- Liver Bx : inactive liver histology with a usually minimal amount of fibrosis.

Phase IV : E negative hepatitis.

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- IVa : Reactivation with the wild-type virus : HBe-Ag +ve state.
- IVb : Replication-competent HBV variants : HBe-Ag -ve disease.
  - HBeAg -ve with anti-HBe.
  - HBV-DNA : 2000-20 mIU/ml (104- 108 copies/ml).
  - ALT elevated.
  - Liver bx : moderate or severe necro-inflammation with variable amounts of fibrosis.
- IVc : Inactive carrier may lose HBs Ag & developed anti-HBs but low titer of HBV DNA persists → Reactivation.

**Occult hepatitis B :**

Presence of HBV DNA in the liver of individuals testing HBSAg-negative with currently available assays & a cutoff value for serum HBV DNA (< 200 IU/mL).

Clinical importance :

- Reactivation.
- Blood transfusion in immune-compromised recipient.
- Organ donation :
  - Liver transplantation.
  - Non liver solid organ transplantation.

**Goals of antiviral therapy :**

1. HBeAg seroconversion : Decrease in viral replication.
2. Normalization of serum aminotransferases and liver histopathology.
3. Prevention of progression of liver disease including cirrhosis and HCC.

Indication of treatment :

- Duration : More than 6 months.
- Biochemical : Raised transaminases >2 times the upper limit.
- Viral replication : HBe-Ag +ve and high DNA.
- Liver histology : Disease activity.

**Acute Hepatitis B :**

- Asymptomatic ← Self limited hepatitis → FHF.
- Incubation period : 45-180 days.
- Children < 5yrs : Anicteric acute hepatitis B.
- Prodrome : Anorexia, fever, nausea, vomiting, myalgia, hepatomegaly.

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- Icteric phase :
  - Jaundice, dark urine hepatomegaly.
  - Self-limited course.
  - Recovery is marked by anti-HBs seroconversion.

## Other causes of hepatitis

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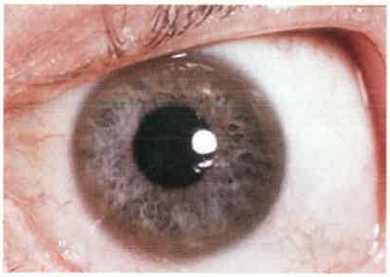
### Wilson disease :

Presentation :

- Persistent hypertransaminasemia.
- Asymptomatic hepatomegaly.
- Fatty liver.
- Acute hepatitis (25 %).
- Chronic Hepatitis (10-30 %).
- Cirrhosis.
- Acute liver failure (10 %).

Features :

- Any suspected liver disease
- Associated neurological illness.
- Positive family history.
- Serum ceruloplasmin is low.
- Eye examination for KF ring.
- 24 hour urinary copper.



KF ring

### Autoimmune hepatitis :

Features :

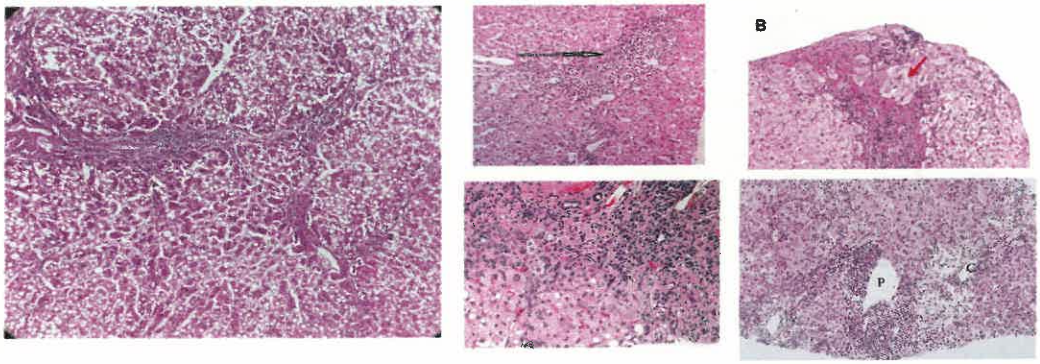
- Recurrent jaundice.
- More incident in female sex.
- Associated thyroid, skin, hemolysis, celiac disease conditions.
- Presence of other autoimmune disorders.

Investigations for diagnosis :

- Total IgG.
- DCT, retic count.
- ANA, ASMA, LKM-1.
- Liver biopsy.

Liver biopsy features :

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Liver biopsy

- Micro and macronodular cirrhosis.
- Lymphoplasmacytic infiltrate.
- Interface activity
- Pseudo rosettes.
- Confluent necrosis.
- Emperipolesis.

## Chronic viral hepatitis :

- Any suspected liver disease.
- Positive family history
- HBsAg, Anti HBc total.
- Anti HCV.

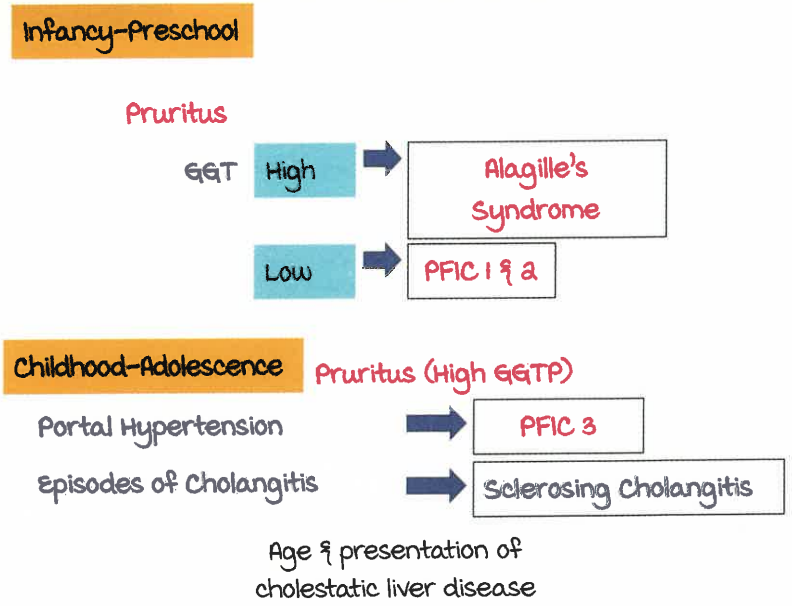
## Cholestatic liver disease :

Features :

- Itching, pale stools.
- Associated vitamin deficiency.
- Shiny nails, lichenoid skin.
- Positive family history.

Investigations :

- Serum GGTP levels.
- USG abdomen.
- Liver biopsy.
- Genetic testing.




Age & presentation of cholestatic liver disease

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Shiny nails



Lichenoid skin

## Budd Chiari syndrome :

- Massive ascites + jaundice.
- Dilated abdominal veins.
- investigation : USG abdomen + doppler.
- Acute : more of jaundice and hepatic encephalopathy.
- Chronic : more of ascites (massive).



Dilated abdominal veins
and massive ascites

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# DIAGNOSIS AND MANAGEMENT OF AUTOIMMUNE HEPATITIS

## Diagnosis of AIH

00:00:19

### Clues to AIH :

- Adolescent girl, relapsing jaundice, vitiligo, high globulin fraction.
- Other features to look for :
  - Vitiligo, scleritis, acne, alopecia, hypothyroidism.
  - Family h/o autoimmune disorders.

### Types of AIH in children :

1. Type 1 AIH (AIH-1) : ANA and/or ASMA positive.
2. Type 2 AIH (AIH-2) : Anti LKM-1 and/or anti-liver cytosol type 1 antibody (Anti-LC-1) positive.
3. Autoimmune sclerosing cholangitis (ASC) : Sclerosing cholangitis (Large/small duct) with AIH, cholestatic pattern of jaundice.

### Clinical presentation :

| Features | AIH-1 | AIH-2 |
|---|---|---|
| Female : male | 3 : 1 | 3 : 1 |
| Frequency | 2/3rd | 1/3rd |
| Age | Adolescents | Younger, infancy |
| Acute hepatitis | 40-50% | 40-50% |
| Fulminant hepatic failure | 3% | 25% |
| Chronic liver disease | 40% | 25% |
| Complications of cirrhosis & PHTN | 10% | 10% |
| Incidental finding of raised hepatic aminotransferases | Rare | Rare |

- Positive family h/o autoimmune disease : 40%.
- Associated autoimmune disorders (20%) :
  - Thyroiditis, coeliac disease, insulin-dependent diabetes, IBD, haemolytic anemia, vitiligo, Behçet disease, Sjögren syndrome, glomerulonephritis, idiopathic thrombocytopenia, urticaria pigmentosa, hypoparathyroidism.

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## Diagnosis :

- Clinical evidence : Recurrent hepatitis, acute liver failure, portal HTN/cirrhosis.
- Biochemical evidence : AST, ALT elevation.
- Immunological evidence : ↑ IgG levels.
- Histologic evidence : Liver biopsy (Transjugular) to confirm the diagnosis and to evaluate the severity of liver damage.
- Exclusion of other etiologies.

## Simplified diagnostic criteria :

| Variable | Cutoff | Points |
|---|---|---|
| ANA or SMA | ≥ 1 : 40 | 1 |
| ANA or SMA | ≥ 1 : 80 | 2 |
| or LKM | ≥ 1 : 40 | 2 |
| or SLA | Positive | |
| IgG | > Upper normal limit<br>> 1.10 times upper normal limit | 1<br>2 |
| Liver histology (Evidence of hepatitis is a necessary condition) | Compatible with AIH<br>Typical AIH | 1<br>2 |
| Absence of viral hepatitis | Yes | 2 |

Scoring :

- ≥ 6 → Probable AIH, ≥ 7 → Definite AIH.
- Addition of points achieved for all autoantibodies (maximum, 2 points).

Problems with simplified diagnostic criteria :

- Not suitable to the juvenile form of the disease :
  - Lower titres of autoantibodies.
  - Antibodies may be negative in fulminant hepatic failure.
  - Does not allow distinction b/w AIH and ASC.

Proposed scoring criteria (ESPGHAN) :

| Variable | Cut-off | Points | |
|---|---|---|---|
| | | AIH | ASC |
| ANA and/or SMA | ≥ 1 : 20# | 1 | 1 |
| | ≥ 1 : 80 | 2 | 2 |
| Anti-LKM-1 or anti-LC-1 | ≥ 1:10# | 1 | 1 |
| | ≥ 1:80 | 2 | 1 |
| | Positive# | 2 | 1 |
| Anti-SLA | Positive# | 2 | 2 |

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| | | | |
|---|---|---|---|
| pANNA | Positive# | 1 | 2 |
| IgG | > ULN<br>> 1.20 ULN | 1<br>2 | 1<br>2 |
| Liver histology | Compatible with AIH<br>Typical of AIH | 1<br>2 | 1<br>2 |
| Absence of viral hepatitis (A, B, C, EBV), NASH, Wilson disease and drug exposure | Yes | 2 | 2 |
| Presence of extrahepatic autoimmunity | Yes | 1 | 1 |
| Family h/o autoimmune disease | Yes | 1 | 1 |
| Cholangioraphy | Normal<br>Abnormal | 2<br>-2 | -2<br>2 |

Score ≥ 7 : Probable AIH, ≥ 8 : Definite AIH.

Score ≥ 7 : Probable ASC, ≥ 8 : Definite AIH.

Typical histological features :

- Lymphoplasmacytic infiltrates.
- Interface hepatitis : Inflammatory spillover beyond the limiting plate.
- Emperipolesis : Hepatocytes engulfing hepatocytes/macrophages.
- Rosette formation.

## Management of AIH

00:17:05

### Complete remission :

- Complete clinical recovery, normalisation of transaminases, normal IgG levels, negative or low titre of autoantibodies (ANA & SMA < 1 : 20, anti-LKM1 & anti-LC1 < 1 : 10) and histological resolution.

### Relapse :

- ↑ Serum aminotransferase levels after remission has been achieved.
- Incidence : 40%. (Need temporary ↑ in steroids).
- Treat all children.
- Cirrhosis at diagnosis : 44% to 80%.
- Most children remain clinically stable and well on long term treatment.
- Upto 90% remission following immunosuppressive treatment.
- Low mortality.

### Standard treatment :

Prednisolone :

- 2 mg/kg/day (maximum 60 mg/day), tapered over a period of 4–8 weeks to a maintenance dose.
- In 1st 2 months : 80% decrease of aminotransferase levels.

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- Remission : 60-80% by 6-12 months.
- Side effects :
  - Mild : Cosmetic (Weight gain, hirsutism).
  - Serious : Osteoporosis, pathological fractures, raised IOP, glaucoma, HTN, mellitus.

Azathioprine :

Eventually needed in 85% of cases.

Protocol :

- Vary from centre to centre.
- If the transaminase levels stop decreasing on steroid treatment alone, azathioprine is added at a dose of 0.5-2 mg/kg/day after a few weeks (usually two) of steroid treatment.
- A combination of steroids and azathioprine from the beginning.
- Side effects :
  - Bone marrow suppression : Check for TPMT, NUDT15 status to assess risk.
  - Hepatotoxicity.
  - Infection.
  - Pancreatitis, GI disturbances.

**AIH with sclerosing cholangitis in children (ASC) :**

- Prospective study b/w 1984 & 1997.
- N=55, Median age : 11 years (2.2 years to 16 years)
- 54 underwent proctosigmoidoscopy and rectal biopsy

Cholangiography :

- Sclerosing cholangitis (ASC) in 49% of AIH.
- 67% : Intra and extrahepatic bile duct abnormalities.
- 33% : Only intrahepatic bile ducts.

**Treatment of autoimmune fulminant hepatic failure :**

Limitations :

- Diagnostic difficulties :
  - Negative autoantibodies.
  - Normal IgG (15-20%).
  - Histology : Risky to perform liver biopsy.
- Adults : Poor outcome (70-90%) with increased risk of sepsis.

Fulminant hepatic failure of autoimmune etiology :

----- Active space -----

Study of 46 children managed in last 15 years.

- 10/46 (22%) had AI-FHF.
- mean follow up : 4.6 years.
- Type 2 AIH : 6, type 2 AIH : 4.
- Liver histology : 9.
- Treatment : 9 with steroids.

Study :

| Patient no. | Age (yrs) | Sex | Diagnosis | HE, degree | Steroids | LTX | Follow up, (yrs) | Outcome |
|---|---|---|---|---|---|---|---|---|
| 1 | 13.5 | m | AIH-1 | 2 | Yes | No | 3 | Well |
| 2 | 6.1 | F | AIH-2 | 4 | Yes | Yes | 3.3 | Recurrence |
| 3 | 5.9 | m | AIH-1 | 2 | No | Yes | 6.4 | Well |
| 4 | 7.6 | F | AIH-2 | 3 | Yes (C) | Yes | 4.5 | Recurrence |
| 5 | 15.1 | m | AIH-1 | 4 | Yes | No | 0.2 | Lost to follow up |
| 6 | 3.1 | F | AIH-2 | 4 | Yes | No | 0.1 | Death because of encephalopathy |
| 7 | 6.9 | m | AIH-2 | 3 | Yes (C) | Yes | 7.4 | Recurrence |
| 8 | 3.6 | m | AIH-1 | 3 | Yes | No | 1.5 | Well |
| 9 | 13.3 | m | AIH-2 | 3 | Yes (C) | No | 4.6 | Well |
| 10 | 1.3 | m | AIH-2 | 4 | Yes | Yes | 2.7 | Well |

When to stop treatment :

- At least 2-3 years of treatment.
- Normalization transaminase & IgG levels, negative autoantibody (Or at max titre of 1 : 20) for at least a year f/b liver biopsy.
- Successful long term complete withdrawal of treatment was possible in 20% of patients with AIH-1, but not in AIH-2.

**Liver transplantation :**

- AIH accounts for 2%-5% of paediatric LTs performed in Europe & US.
- Transplant rate for AIH is variable, ranging from 9% to 55%.
- The reported 5-year survival rate after LT for AIH is excellent (80%-90%).

Recurrence of AIH after LT :

- Steroid treatment (In small doses) is continued post transplantation to prevent recurrence.
- Mean time from LT to recurrence : 5 years.