# Acute Hepatitis in Children: First Approach

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Condition: **Acute Hepatitis in Children** · Based on: *Nelson's Textbook of Pediatrics & UpToDate*

# Pathophysiology

Acute hepatitis in children results from hepatocellular injury triggered by infectious, toxic, metabolic, or immune-mediated mechanisms. The common final pathway involves:

### Viral Cytopathic Effect

Direct injury from HAV, HBV leading to hepatocyte necrosis and inflammation

### Immune-Mediated Destruction

CD8+ T-cell mediated destruction of infected hepatocytes — predominant in HBV

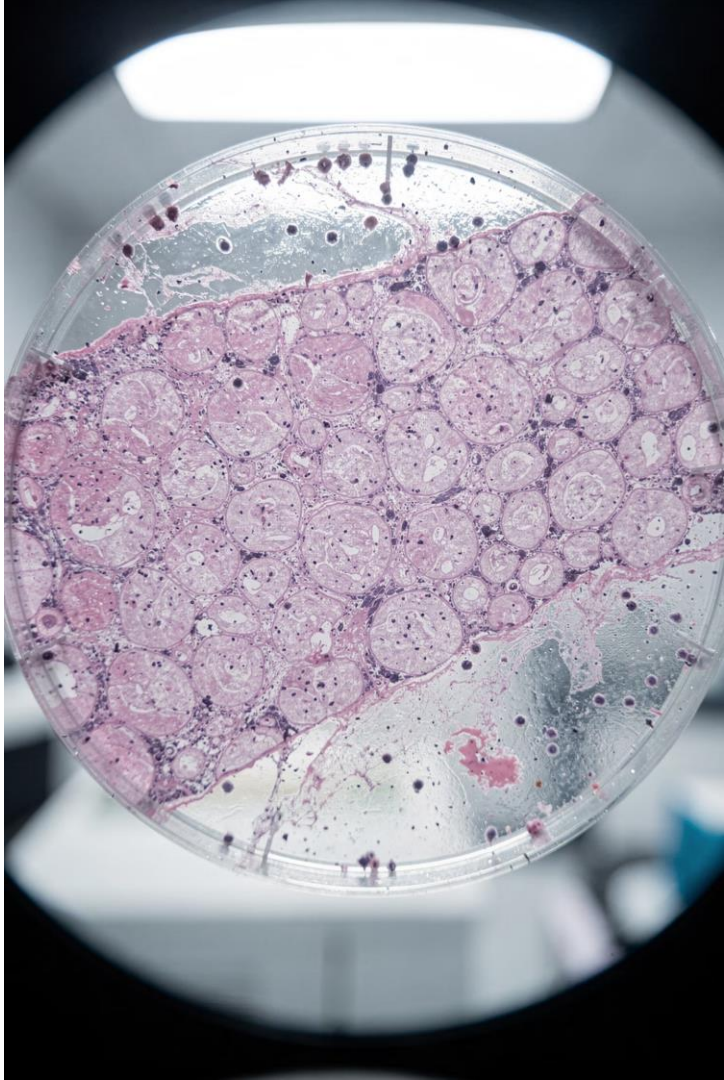
### Mitochondrial Dysfunction

Oxidative stress from toxins or metabolic disease disrupting cellular energy production

### Cytokine Storm

TNF-α and IL-6 mediated inflammation amplifying hepatocellular injury cascade

⚠ This results in elevated transaminases, impaired synthetic function (coagulopathy, hypoalbuminemia), and potentially **fulminant hepatic failure** if injury is severe or prolonged.



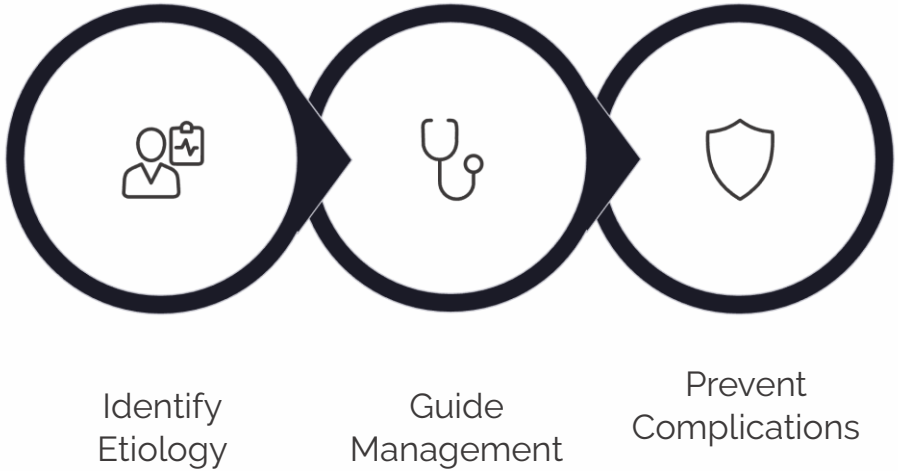
# Brief Report

## Definition

Acute hepatitis in children is defined as inflammation of the liver lasting **less than 6 months**, characterized by elevated serum aminotransferases (ALT, AST).

## Clinical Spectrum

It spans a broad clinical spectrum — from **asymptomatic transaminase elevation** to **fulminant hepatic failure**. Etiology is diverse; infectious causes dominate in pediatrics (especially viral hepatitis A and B), but toxic, autoimmune, and metabolic causes must also be considered.
Prompt identification of the underlying etiology guides management, prevents complications, and determines need for specialist referral.




A structured first approach ensures no critical diagnosis is missed and that life-threatening presentations are escalated promptly.

# Clinical Features

Presentation varies by age, etiology, and severity. Recognizing the full spectrum — from prodrome to signs of liver failure — is essential for timely management.

## Prodrome

- Low-grade fever, malaise, anorexia
- Nausea, vomiting
- Abdominal pain (RUQ or diffuse)

## Icteric Phase

- Jaundice (scleral icterus first)
- Dark urine (bilirubinuria)
- Pale/acholic stools, pruritus

## Physical Findings

- Hepatomegaly (tender) — most consistent
- Splenomegaly — EBV/CMV or AIH
- Rash, arthralgia — viral or autoimmune

## Liver Failure Signs

- Encephalopathy, coagulopathy
- Ascites, hypoglycemia
- Infants: poor feeding, irritability, cholestatic jaundice

Signs of liver failure — encephalopathy, coagulopathy, ascites, hypoglycemia — indicate **fulminant disease** requiring urgent escalation.

# Etiology: Common Causes in Children

The etiology of acute hepatitis in children is broad. A systematic approach by category ensures no treatable or life-threatening cause is overlooked.

| Category | Examples / Notes | Key Points |
|---|---|---|
| **Viral (most common)** | HAV (fecal-oral, most common in children), HBV (vertical/parenteral), HCV, EBV, CMV, HSV (neonates), Adenovirus, Enterovirus | HAV is self-limiting; HSV in neonates is life-threatening |
| **Drug / Toxin** | Paracetamol overdose (#1 toxic cause), valproate, isoniazid, azithromycin, herbal/complementary medicines | Always take detailed medication and herbal history |
| **Autoimmune** | Autoimmune hepatitis (AIH) — Types 1 & 2; can present acutely with markedly elevated transaminases | Check ANA, anti-SMA, anti-LKM1, IgG |
| **Metabolic** | Wilson's disease (consider in children >3 yrs), Alpha-1 antitrypsin deficiency, fatty liver disease (NAFLD/MAFLD) | Wilson's is fatal if missed — always consider |
| **Systemic / Other** | Sepsis (neonates/infants), ischemic hepatitis, Kawasaki disease, macrophage activation syndrome (MAS) | Liver injury usually reversible with treatment of underlying cause |

# Investigations: Tier 1

ALL PATIENTS — INITIAL WORKUP

The following investigations should be performed in **all children** presenting with suspected acute hepatitis to define pattern, severity, and etiology.

1. **Liver Function Tests**
   ALT, AST, ALP, GGT, total/direct bilirubin — define pattern and severity of injury
2. **Coagulation Studies**
   PT/INR, aPTT — key indicator of synthetic function and liver failure risk
3. **Synthetic Markers**
   Serum albumin, total protein — differentiate chronic vs acute impairment
4. **Blood Glucose**
   Hypoglycemia indicates severe hepatic dysfunction — check urgently

1. **CBC + CRP**
   Infection screen; cytopenias may suggest EBV/CMV hepatitis
2. **Renal Function**
   U&E, creatinine — assess hepatorenal syndrome risk in severe disease
3. **Blood Gas**
   Metabolic acidosis in fulminant failure or paracetamol toxicity
4. **Hepatitis Serology**
   Anti-HAV IgM, HBsAg, anti-HBc IgM, HCV RNA/antibody — first-line viral screen

# Investigations: Tier 2

BASED ON CLINICAL SUSPICION

Second-tier investigations are targeted based on the clinical picture and initial results. Each test is directed at a specific diagnostic category.

## Toxic / Drug Etiology

**Paracetamol level** — if toxic or drug etiology suspected. Use Rumack-Matthew nomogram to guide NAC therapy.

## Autoimmune Hepatitis

**ANA, anti-SMA, anti-LKM1, IgG levels** — autoimmune hepatitis screen in all atypical presentations.

## Imaging

**Abdominal ultrasound** — hepatomegaly, biliary anatomy, vascularity (Doppler). Essential in all cases.

## Viral Infections

**EBV/CMV serology or PCR** — if infectious mononucleosis picture. **HSV PCR** — in neonates with acute liver failure.

## Metabolic Disease

**Ceruloplasmin, 24h urine copper, slit-lamp** — Wilson's disease. **Alpha-1 antitrypsin level** — if metabolic cause considered.

## Liver Biopsy

If **autoimmune hepatitis** or uncertain etiology requiring histological diagnosis to guide treatment.

# Management: First Approach

Management is etiology-specific but initial stabilization follows a common framework. The table below summarizes specific treatments by cause.

| Etiology | Specific Treatment | Dose / Route | Notes |
|---|---|---|---|
| **HAV** | Supportive only | Hydration, nutrition, rest; avoid hepatotoxic drugs | Notifiable disease; isolation precautions; self-limiting |
| **HBV (acute)** | Supportive; antiviral if fulminant | Tenofovir or entecavir if ALF develops | Most acute HBV in children resolves; monitor for chronicity |
| **HCV** | Specialist referral | Direct-acting antivirals (DAA) after acute phase; defer in children <3 yrs | High chronicity risk; refer to hepatology |
| **Paracetamol toxicity** | N-Acetylcysteine (NAC) | 150 mg/kg IV over 15 min → 50 mg/kg over 4 hrs → 100 mg/kg over 16 hrs | Give within 8 hrs of ingestion for maximum benefit; use Rumack-Matthew nomogram |
| **Autoimmune hepatitis** | Prednisolone + Azathioprine | Prednisolone 2 mg/kg/day (max 60 mg); Azathioprine 1–2 mg/kg/day | Check TPMT level before azathioprine; lifelong therapy often needed |
| **Wilson's disease** | Chelation / Zinc / OLT | D-Penicillamine or Trientine; Zinc 25–50 mg TDS | Orthotopic liver transplant (OLT) for fulminant presentation |
| **HSV (neonates)** | IV Acyclovir | 20 mg/kg IV every 8 hrs for 14–21 days | High mortality if untreated; empiric use in any neonate with ALF |
| **Sepsis-associated** | Antibiotics + supportive | Empiric broad-spectrum IV antibiotics; source control | Liver injury usually reversible with treatment of underlying infection |

# Supportive Measures & Referral Criteria

## Supportive Measures — All Patients

### Hydration

Oral if tolerated; IV if vomiting or dehydrated (NS or D5NS)

### Nutritional Support

High carbohydrate diet, small frequent meals; protein restriction only in encephalopathy

### Avoid Hepatotoxic Drugs

NSAIDs, paracetamol (unless monitoring levels), sedatives, statins

### Monitoring

Daily LFTs, INR, glucose, bilirubin until improving; strict I/O in severe cases

### Vitamin K

1–2 mg IV/IM if coagulopathy — to differentiate from liver synthetic failure

### Treat Pruritus

Cholestyramine or rifampicin if significant; contact/droplet precautions for HAV

## Indications for Urgent Referral / Hospitalization

- **INR >1.5** or coagulopathy unresponsive to Vitamin K — suggests acute liver failure (ALF)
- **Encephalopathy (any grade)** — indicates ALF; urgent transfer to liver unit
- **Hypoglycemia, hyponatremia**, or severe metabolic derangement
- **Bilirubin rapidly rising** or >15 mg/dL
- **Age <3 months** with any liver disease — always refer (biliary atresia must be excluded)
- **Suspected Wilson's disease** with hemolytic anemia (Coombs-negative) — high ALF risk
- **Suspected HSV hepatitis in neonates** — treat empirically without waiting for confirmation

# Key Home Messages

These are the most critical clinical pearls to remember when approaching acute hepatitis in a child:

1

### HAV — Supportive Care

HAV is the most common cause of acute hepatitis in children — self-limiting; supportive care is the mainstay.

2

### INR + Glucose First

Always check INR and blood glucose — the most important markers of liver function and impending failure.

3

### NAC for Paracetamol

Paracetamol toxicity is the leading cause of acute liver failure — treat with N-Acetylcysteine even if late presentation.

4

### Never Miss Wilson's

Never forget Wilson's disease in any child >3 years with unexplained liver disease — treatable but fatal if missed.

5

### Screen for AIH

Autoimmune hepatitis can mimic acute viral hepatitis — check ANA, IgG, and anti-SMA in all atypical cases.

6

### Encephalopathy = ALF

Any child with encephalopathy + liver disease = Acute Liver Failure — refer immediately to a specialist liver centre.

Prognosis is generally **excellent** for viral hepatitis; **poor** for untreated Wilson's disease or HSV hepatitis in neonates. Early recognition saves lives.