Pediatric Hepatology

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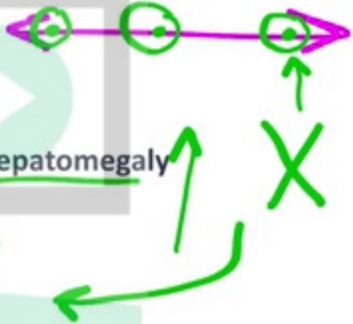
Pathophysiology

- HAV is not cytopathic in itself; research suggests that liver damage is caused by cellular immunity (especially CD8+ T cells).

Clinical features

- **Phases of acute viral hepatitis**
  1. **Prodromal phase:** 1–2 weeks
     - Right upper quadrant pain, **tender hepatomegaly**
     - Fever, malaise
     - Anorexia, nausea, vomiting
  2. **Icteric phase:** ~ 2 weeks
     - **Jaundice**
     - Dark urine and pale stools
     - Pruritus
  - **Resolution of symptoms**
- Most patients achieve full recovery



Diagnostics

- **Confirmatory testing**
  - ↑ **Anti-HAV IgM antibodies**: present in patients with active infection
    - Usually detectable 5–10 days after exposure and 5–10 days before clinical symptoms develop
    - Levels peak commonly ~ 1 month after infection.
    - May persist for up to 6 months after infection
  - ↑ **Anti-HAV IgG antibodies**
    - Develop during active infection and persist indefinitely after infection or vaccination
    - Production begins within 2–3 weeks of infection.
  - HAV RNA can be detected in stool and serum samples using PCR

Hepatitis A vaccine

- Inactivated HAV vaccines for children older than 12 mo.
- Administered IM in a 2-dose schedule, 6-12 month between them
- Protective antibody titer persists for longer than 10 yr in most patients.
- It is not present in the universal immunization of Iraq , In the United States and some other countries, universal vaccination is now recommended for all children older than 12 mo.

IgG

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