

* hepatitis → inflammation of liver.

↳ presence of inflammatory cells in liver.

Causative → viruses, bacteria, protozoa, + non-infectious agents ↳ smoking toxins -
↳ drugs

* viral cause *

↳ Major cause to infectious liver disease (+) additional to hepatitis viruses causing sporadic cases of hepatitis.

① yellow fever virus

② Cytomegalovirus.

③ Hepatitis simplex ④ Rubella

⑤ enteroviruses

↳ all infect liver.

acute infections by viruses can't be distinguished from each other clinically, (hepatitis) usually hepatic enzymes in serum increase (AST) aspartate aminotransferase, as well as alanine amino transferase. to allow diff from other liver diseases.

general rule. Ratio ALT/AST > 1 indicates cytoplasmic damage in other words = hepatitis.

although all of these viruses directed towards liver. and basic hepatitis symptoms are similar. it differs ① structure. ② mode of replication. ③ mode of transmission. ④ time course.

* Most causes of acute viral hepatitis in children and adults are caused by one of the 5 following agents.

① H. virus A previously known as infectious hepatitis + ss RNA, non enveloped

② H. virus B Serum hepatitis Ds DNA, enveloped.

" " C common cause for post transfusion hepatitis + ss RNA, enveloped.

" " D Deltavirus, dependent in co infection with H. B - ss RNA

" E enterically transmitted hepatitis - like hepatitis A virus Regardless

* regardless of virus type, identical histopathological lesions are observed in liver during acute disease. These viruses aren't cytopathic but immunological responses that lead to these lesions mainly.

* Hepatitis A virus *

distinct member of pico family, another name for it is enterovirus 72, RNA virus non enveloped has only one serotype. and there is 7 genotypes that are known for it.

Reproduction: liver excreted: bile then excreted in feces
of infected persons two weeks before onset of symptoms - up to one week post symptoms

established for causative agent of more than > 40% to 50% of acute hepatitis cases. The most common viral hepatitis virus.

Spread by  feco-oral route. outbreaks associated with contaminated food and water. human appears to be major natural hosts. of hepatitis A

* enterically transmitted virus → feco oral route.

They can be demonstrated in feces by electron microscope in feces and no chronic infection for H.A. Same for E initially only IgM antibody response then followed by IgG antibody response and give life long immunity post infection, so there is no chronic infection and no carrier state

* epid. Microbiology of Hepatitis A

global epid. Microbiology / Most common type of viral hepatitis.

Most common: ① outbreaks ② epidemics.

Commonly seen ① children ② young adults

90% of infected children and up to (25-50)% of infected adults have inapparent but productive infection

(Pico oral route) arise from infections of contaminated food/water

① overcrowding ② poor sanitation → triggers.

Hepatitis A → infectious hepatitis. *causative disease. Clinically milder or asymptomatic in young children and no carrier state.*

More than 90% of adults pop in many developing countries show serological previous hepatitis A infection. and travelers from developed country who enter these endemic areas are particularly susceptible.

patients are more contagious in 2 weeks prior to onset of clinical disease characterized by jaundice mainly

high prevalence of anti-hepatitis A antibodies usually in jaundice & prevalence of hepatitis A and antibodies against it.

* Clinically

infectious dose for hepatitis A is less than 100 particles

enough to establish infection contagiousness / communicability

usually 2 wks before onset of symptoms usually characterized by onset of jaundice

Jaundice usually is observed in 70% to 80% of adults

but 10% in children, mainly less than 6 yrs

The most common presentation of hepatitis virus infection is

asymptomatic → most children

(3)

usually symptomatic → adults.

nausea
vomiting
anorexia

usually 2 phases → pre-icteric phase > flu like illness
→ icteric phase → jaundice, abdominal pain.
→ hepatosplenomegaly
→ pale stool / dark colored urine

Sometimes sudden onset of fever, anorexia, pain: right upper quad
within several days → classical history in sym patients
dark urine / pale colored stool (clay)

(1-5) days later clinical jaundice. liver is enlarged and tender

Recovery takes first days to weeks

99% of hepatitis A are self limiting (2-4) wks no carrier

no chronic case → لا مزمنة

* Some patients might suffer from full permanent hepatitis < 1% of infected patients.

* Many persons have serological evidence of acute hepatitis A infection
① asymptomatic ② mildly ill without jaundice

unlike hepatitis B virus. h. A can't!!! initiate chronic infection.

and not associated with hepatic cancer

1% that may suffer from full permanent liver disease → < 1%
and Mortality Rate (0.1-0.2)% for those who suffer from full permanent.

* diagnosis according to clinical picture mentioned, history, ph. examination
and you look for liver biochemistry that is elevated liver enzymes
Mainly ALT as well as AST which protect jaundice, icteric state
where serum bilirubin levels high.

Continuation of diagnosis.

Haematological test: leucopenia? with relative lymphocytosis in infected patient and the (ESR) usually elevated

Viral Markers / Serology for hepatitis virus that can be detected on usually anti-hepatitis virus IgM in acute infection. It remains usually high for up to 3 to 6 months followed by IgG antibodies to hepatitis A virus that become predominant after more and common in general population over age of 50 yrs.

There have immune electron Microscope, we can identify virus particles in stool specimens and isolation of virus in cell culture remain the standard

* Typical Serology Course of hepatitis A virus, initially can be identified in the stool & raised liver function test. ALT / AST which are noted earlier in H.A.V followed by anti-hepatitis A virus IgM that start rising usually one month post exposure and peaks on 3rd month post exposure before being replaced with IgG antibodies to hepatitis A virus.

previous infection and reservoir → id. long immunity

Treatment? no specific treatment (supportive measures indicated) adequate nutrition and bed rest avoidance to exposure to contaminated food/water

~~2 weeks treatment~~ → passive immunization → prophylaxis with immune serum globulin before or early in incubation period
→ active immunization
less than 2 wks post exposure on 16-90% if in proximity

2 forms of immunisation

① passive immunization → prophylaxis with immune serum globulin given usually before or early in incubation periods. less than 2 wks post exposure
usually 80% - 90% effective in preventing clinical illness
Prevent H. N. infection

passive?

② active? when immune serum globulins are produced
it given before or during incubation periods of

@ active immunization

There is vaccine against hepatitis A virus with formalin killed vaccine that usually induces antibody with similar to those induced with the wild virus type
100% protective

preventing major spread of hepatitis → interrupting feco-oral route of spread of virus accomplished by avoiding potentially contaminated water or food especially under cooked shellfish and proper hand washing

usually important that chlorine treated in drinking water is generally sufficient to kill virus.

E

* hepatitis E virus * similar to A virus. @ spread by feco-oral route.
in a virus similar but distinct from B virus

Viral particles in stool are spherical usually with 30 nm and unenveloped exhibit spike on their surface like hepatitis A. infection with E is frequently subclinical, when symptomatic it causes only mild disease that may subside. high risk group with hepatitis E pregnant women malnourished people, endemic and developing areas it has highest

highest risk in 16 weeks of life

and infection is associated with contaminated drinking water, it doesn't appear to spread from person to person. Most cases have been identified in developing countries with poor sanitation. recurrent epistaxis have been described in these areas.

incubation period $\rightarrow$ 40 days (2-8) weeks

diagnosis $\rightarrow$ present at 19M
(no treatment)

* Mostly Subclinical specially in children

Clinically similar to hepatitis A infection except bilirubin levels are high and jaundice is deeper and more prolonged.

normal case fatality rate is 1 to 2% but higher reaching 10 to 20 percent in pregnant and malnourished

There is no chronic stage or carriers

diagnosis is by excluding other types as well as molecular PCR

Supportive treatment