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Case Report

A Complex Infectious Case of Acute Viral Hepatitis A, Hepatic Dysfunction, Severe Dehydration and Jaundice: Diagnostic Way of Approach Towards Clinical Outcome

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I N F O

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A B S T R A C T

With an emphasis on acute viral hepatitis A and related complications, the study intends to examine the clinical presentation, diagnostic way and management approaches of infectious diseases. Through extensive laboratory testing, imaging and clinical surveillance, a case report of a 21year old male with acute viral hepatitis A, hepatic dysfunction, severe dehydration and jaundice was reviewed. the results showed positive serological testing indicating acute viral hepatitis A, abnormalities consistent with substantial rise of liver enzymes. The instance underlines the value of coordinated diagnostic and treatment techniques as well as the complexity of illness. The findings highlight the viral diseases with vigilance, particularly in environments with low resources. All things considered, the study emphasises on how important comprehensive healthcare methods are for enhancing patient outcomes and tackling complex illness relationships.

Keywords: Acute viral hepatitis- A, Infection, Hepatic Dysfunction, Severe Dehydration, Jaundice

Introduction

One of the most prevalent infectious diseases is acute viral hepatitis, with hepatitis A being the most prevalent type in much of the world. Hepatitis A outbreaks have been known to occur concurrently with combat for ages, impacting civilians as well as soldiers. There is no particular medication treatment available. Hepatitis A typically causes no symptoms in infants, but it can cause a clinically noticeable illness in adults, frequently accompanied by jaundice. Severe hepatitis A has been known to cause acute liver failure in young infants, although it is more common in middle-aged and older adults as well as those with existing chronic liver conditions. It is also an uncommon consequence of hepatitis A during pregnancy. Hepatitis A has a greater economic impact on adults due to its increased severity with age; nonetheless, there is currently limited

research on the expenses of sickness. The virus that causes hepatitis A does not cause persistent viral infection or an intestine carrier status, nor is it associated with chronic liver disease. In nature, infection is sustained through serial transmission between acutely contaminated individuals to vulnerable others. There is no proof that the virus responsible for hepatitis A is an infectious species or that there is an infection reservoir outside of humans, despite reports of transmission from newly infected primates who are not human to humans.[1,4,8]

Every year, tens of millions of people worldwide get the Hepatitis A virus (HAV), mostly through contact with infected people and fecal-oral transmission from tainted food or drink. Young infants frequently don't have any symptoms, but older kids and adults may have jaundice, take longer to heal, and run the danger of dying from acute liver failure. People who are infected usually miss several weeks of work or school, which results in high medical expenses. There are differences between nations according to socioeconomic status, despite the fact that the incidence of HAV is declining globally due to an aged population at infection. The median age at infection is rising while the incidence of HAV is declining in middle-income nations with growing earnings and urbanization. A number of regions, including East and Southeast Asia, Europe, Latin America, and portions of the Middle East and North Africa, saw significant rises in AMPI between 1990 and 2005. It's interesting to note that since older patients are typically more severely afflicted than younger ones; a decreased prevalence of HAV may result in increased hospitalizations and mortality.[2,4]

HAV is a single strain positive sense RNA virus with six genotypes that is a member of the Hepato virus genus. Humans are infected by genotypes I through III. There are two types of HAV in the host: quasi-enveloped virions that circulate in the blood and bare virions that are lost in the feces. The synthesized genome-length RNA is contagious in both forms. The majority of liver damage is caused by the host immunological response, and HAV has no appreciable direct cytopathic effect. Humoral response directly contributes to infection prevention, but cellular immunity seems to be in charge of viral clearance following initial infection. HIV infection and other cellular immune response deficiencies can result in prolonged shedding (and infectivity) without a noticeable increase in symptoms HIV infection and other cellular immune response deficiencies can result in prolonged shedding (and infectivity) without a noticeable rise in symptom severity.[3,4,6]

Hepatocytes are reached via portal circulation once HAV enters the bloodstream through the digestive system. There are two infectious forms of it: quasi-enveloped virions (eHAV), which are present in blood and released from infected cells, and naked virions (nHAV), which are lost in feces. Its etiology and transmission are aided by this duality. The HAV cellular receptor-1 is the site of infection, while new research indicates there could may be additional entrance points. Although type I interferons are still produced by plasmacytoid dendritic cells in response to the infection, the host's immune response requires intricate interactions with innate and adaptive systems. HAV uses ways to avoid innate immunity.[4,5]

Although it can cause liver damage, the adaptive immune response—particularly T-cell-mediated immunity—is crucial for the removal of viruses. While research on chimpanzees highlights the function of CD4 lymphocyte helper T cell responses for infection resolution, human studies identify virus-specific cytotoxic T cells known as CD8. Acute hepatitis diagnosis A entails finding serum IgM antibodies or, in situations with symptoms, seroconversion. Lifelong protection is provided by a robust IgG response against viral capsid proteins, and liver damage can be avoided by immunization within two weeks. People are most contagious two weeks before to the onset of jaundice because HAV is expelled in bile and stool after liver replication. One week following the onset of jaundice, infectivity declines.[4,5]

Rarely is a liver biopsy done for simple acute hepatitis A infections. Histopathological analysis of a biopsy shows varying degrees of apoptosis along with inflammation and necrosis in the periportal areas. A final diagnosis requires laboratory confirmation, even though cholestasis combined with an increased presence of plasma cells in the portal and periportal areas may be more suggestive of hepatitis A. Necrotic bridges may develop between portal sites in cases of severe hepatitis.[4]

Hepatitis A virus (HAV) infection can show clinically as either asymptomatic or symptomatic, with the latter being characterized by clay-colored feces, dark urine, and jaundice. Cholestasis hepatitis, recurrent infections, and, in extreme situations, fulminant hepatitis are further symptoms. After an incubation period of roughly 28 days, HAV infections typically have an acute start with prodromal symptoms such as malaise, vomiting, anorexia, fever, and abdominal pain, followed by a more severe phase lasting about 8 weeks.[7] Jaundice (40%–80%), black urine (68%–94%), and weariness (52%–91%) are the main signs of an acute hepatitis A virus (HAV) infection. One-third of patient's report having stomach pain, and appetite loss is prevalent (42%–90%). Alcoholic stools and other cholestatic symptoms are reported by half. While older children and adults exhibit more symptoms, particularly jaundice in more than 70% of cases, children under the age of six may be asymptomatic.[4] The ailment usually goes away in two months, with an average length of two weeks.

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Peak infectivity occurs two weeks prior to or one week following the onset of symptoms. A clinical examination may show painful hepatomegaly, icterus, and occasionally a minor pleural effusion. In addition to uncommon extra hepatic symptoms such splenomegaly, lymphadenopathy, or arthritis, scratch marks and shining nails may be seen during the cholestatic phase. Although these are rare consequences, hepatic encephalopathy and coagulopathy should be taken into consideration.[4,7]

Laboratory testing is essential for the diagnosis of acute hepatitis suspected of being caused by HAV infection. Elevated bilirubin, serum aminotransferases, and alkaline phosphatase are important biochemical markers; alanine aminotransferase (ALT) levels may rise noticeably more than in other viral hepatitis types. While blood testing may reveal minor lymphocytosis and normal prothrombin time, elevated bilirubin typically follows ALT rises; abnormal prothrombin time indicates a serious risk of liver injury. Although the results are frequently nonspecific, an abdominal ultrasound can help rule out causes of liver problems, distinguish between different types of cholestasis, and assess fever with abdominal pain. Prolonged prothrombin time should raise the possibility of acute liver failure (ALF), which calls for intracranial pressure and liver function monitoring. Pruritus and high bile acid levels are prominent symptoms of the cholestatic type of hepatitis A, which is indicated by raised ALP and gamma-glutamyl transferase. Severe fat-soluble vitamin deficits are usually not caused by cholestasis.[4]

Hepatitis A virus (HAV) lacks specific therapy, focusing instead on symptom relief and improved hygiene to prevent transmission. The use of immunoglobulins for prophylaxis has declined since a vaccine was introduced, with the hepatitis A vaccine authorized for post-exposure prophylaxis in immunocompetent patients aged 12 months to 40 years since 2007. Europe has used an inactivated HAV vaccine since 1991, while China adopted a live attenuated version in 1992. The inactivated vaccine is given in two to three doses and has been available in the U.S. since 1995. Routine vaccination is now standard for high-risk groups, including all children aged 12 to 23 months. Vaccination rates are 87% for the first dose and 57% for the second, significantly reducing HAV infections. More than 90% of those completing the two-dose series develop a lasting immune response. Acute liver failure occurs in less than 1% of acute HAV cases, and 31% of these require liver transplants. Post-transplant patients with HAV have poorer survival rates compared to those with hepatitis B, with HAV recurrence and severe pancreatitis as additional risk factors, making post-transplant monitoring critical.[9,11]

HAV complications include: Fulminant hepatitis and acute liver failure, Acute damage to the liver, Extended cholestasis, Acute-on-chronic liver failure in individuals with metabolic dysfunction linked to steatotic liver disease or a history of hepatitis C virus infection, Pericarditis, renal failure, thrombocytopenia, acute pancreatitis, aplastic anemia, autoimmune hemolytic anemia, Guillain-Barré syndrome, vasculitis, and arthritis are among the severe side effects linked to HAV infection.[4,10,14]

Case Report

A 21year old male was admitted to a multispecialty hospital in western India with complaints of persistent vomiting, abdominal pain, fever (high grade), decrease in oral intake, dizziness, anorexia, headache, burning micturition, dark urine, fatigue, yellowing of skin and eye. sleep disturbance with no past medical history and patient was having allergy from tab perinorm (rashes and vertigo). No social history and family history was observed in patient.

On arrival at the hospital, he was conscious; he had a high-grade fever (101.2 °F) with a normal pulse rate, blood pressure, respiratory rate and urine output.

As shown in Table 1, laboratory investigations suggest increase in GGT (333u/l) shows liver dysfunction also increase in total bilirubin (6.9mg/dl), direct bilirubin (6.4mg/dl) and indirect bilirubin 96.3mg/dl) as it indicates liver disease.

High total bilirubin means liver, red blood cells or gall bladder/ bile ducts might have a problem causing a yellow pigment buildup leading to jaundice (yellow skin eyes), dark urine, fatigue

It also shows increase in SGOT (ALT) 9519u/l) indicates liver stress or damage due to hepatitis A in this condition.

Decrease neutrophils levels (31%) indicates due to infection which can cause neutropenia due to hepatitis A virus (57%)

Increase in lymphocyte count (lymphocytosis) is common in hepatitis A due to lymphocytes especially natural killer mk cells and T cells are crucial WBC that activate to fight the viral infection and clear infected liver cells, loading to their temporary rise in the blood as the immune system responds. Increase in CRP (17.5) level indicates inflammation and signaling that immune system may response to infection.

Increase in S. alkaline phosphatase (176u/l) indicates liver dysfunction due to hepatitis A virus.

Specific test like hepatitis A virus (HAV) IGM antibody test (6.98) is high indicates hepatitis A infection positive or reactive or very recent hepatitis A infection.

Urine analysis – protein, sugar and ketone bodies, bile salt and bile pigment present in urine

USG abdomen with pelvis -:

Gall bladder: gall bladder is partially distended and shows peri-cholecystic edema noted at wall thickness measure approx. 6.3 mm no e/o calculus is seen.

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Liver: mild periportal scarring noted. visualized small bowel loops appear fluid filled

Above abdominal findings are likely due to infective etiology due to hepatitis.

The patient was admitted to ICU and treated carefully with constant monitoring. Antibiotics like injection ceftriaxone 1 gm BD given, tab pantoprazole 40 mg OD was given for acidity to sustain acid reflux. tab xetox (l glutathione) BD to help strengthen immune system, promote tissue development and repair. tab heptagon OD was given, tab udiliv 300mg TDS to manage liver damage and treat liver issues by improving bile flow and protecting liver cells from toxic bile acids, reducing cholesterol and preventing liver damage. Tab levocetirizine was given and tab domperidone (sos). Injection ondansetron IV (sos) was given for nausea and vomiting if required. Injection vitamin K (stat), Injection paracetamol IV 100ml (sos) for fever. tab heptagon OD and udiliv 300mg BD was given for nutritional supplement and to prevent vitamin and mineral deficiencies and to support liver health as laboratory investigations suggest hepatic abnormalities. Tab levocetirizine (sos) as required was given due to allergic reaction due to tab perinorm (rashes, vertigo) given due to heartburn, vomiting but stopped due to reaction and change for new medication respectively with other medication.

Table 1.Laboratory Findings

Investigations	*Findings*	*Normal Range*
Haemoglobin	13.5 g/dl	13.8-17.2 g/dl
Red blood cell	4.59 million/cumm	4.20-5.4 million/cumm
Packed cell volume	40.4%	37-47%
RBS (random blood sugar)	92	70-140mg/dl
GGT (gamma glutamyl transferase)	333u/l	15-85u/l
S. alkaline phosphatase	176u/l	46-116u/l
Total bilirubin	6.9 mg/dl	0.1-1.2 mg/dl
Direct bilirubin	6.4 mg/dl	0.1-9.3 mg/dl
SGPT	76 U/L	12-78U/L
SGOT(AST)	519U/L	6-28 U/L
Neutrophils	31%	40-75%
Lymphocytes	57%	20-45%
CRP	17.5mg/l	0.0-5.0 mg/l
Total protein	6.3 gm/dl	6.0-8.3 g/dl
Albumin	3.3gm/dl	3.5- 5.0 g/dl
Hepatitis A virus (HAV) IGM	6.98	> 1.2 reactive

CRP: C REACTIVE PROTEIN
SGOT: SERUM GLUTAMIC OXALOACETIC TRANSAMINASE
SGPT: SERUM GLUTAMIC PYRUVIC TRANSAMINASE

Discussions

Acute hepatitis is frequently caused by the hepatitis A virus (HAV) around the world. Exposure to contaminated food, water, or close physical contact with an infectious individual is the most typical ways that HAV is spread through the fecal-oral route. High-resource nations have low infection rates, according to the World Health Organization. However, individuals who go to endemic areas, injectable drug users, males who have sex with men and secluded tribes are considered high-risk populations.[4] Acute-on-chronic liver failure (ACLF) and non-ACLF are two types of acute-on-chronic liver diseases. ACLF is defined by multiple organ failure after an acute insult, which increases short-term mortality. It affects those with chronic liver disease and has a worse prognosis than acute liver failure. Hepatitis viruses, especially the hepatitis A virus (HAV), which is common in children with ACLF, are frequent triggers in Asia. In India, 42% of children's acute deteriorations are linked to HAV super infection, whereas 61% and 27% of adult cases of ACLF are caused by HAV and hepatitis E virus (HEV), respectively.[13] Effective HAV infection therapies should be the main focus of efforts to improve the prognosis for ACLF Patients with cirrhosis brought on by non alcoholic fatty liver diseases (NAFLD), such as alcoholic liver disorders (ALD) and non -alcoholic steatohepatitis (NASH), may develop ACLF. It is becoming better acknowledged that NASH is a major contributor to ACLF. Reports show instances of ACLF

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in patients with cirrhosis and NASH that are connected to HAV infections. The severity of HAV is influenced by lifestyle variables, concomitant infections, and advanced age. Unsettlingly, cases of severe liver failure and hepatic encephalopathy have been reported in people with acute HAV infection and underlying liver disorders. Excessive alcohol consumption is known to exacerbate ACLF in patients with alcoholic liver disorders, even if the mechanisms connecting alcohol and HAV replication need more investigation.[12]

Strict hygiene procedures, safe food and water, and vaccination are essential for preventing the spread of the Hepatitis A virus (HAV), particularly among high-risk populations and tourists. Improving socioeconomic conditions, immunoglobulin prophylaxis, cleanliness, sanitation, and immunization are important tactics, with a special emphasis on universal pediatric vaccination in intermediate-risk areas. Because HAV is persistent and can spread over long periods of time on a variety of surfaces, foods, and settings, complete cleanliness is required. Immunoglobulin, which is currently mainly recommended for individuals who are hypersensitive to vaccination components, has become less necessary since the development of effective HAV vaccines.[4]

Conclusions

The clinical profile in our intricate case report demonstrated that all of these severities may have uncommon but potential linkages. Specific tests were used to examine and confirm each anomaly. Furthermore, the intricacies of related disorder underscore the significance of all-encompassing healthcare approaches that tackle infectious diseases and their aftereffects.

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