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Case Report on Hepatitis-A

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ABSTRACT

Introduction: *Hepatitis is the inflammation of the liver which occurs from a variety of causes i.e. infectious and non-infectious, hepatitis A is caused due to picornavirus usually transmitted by the feco-oral route. It is a highly contagious liver infection that affects the individual's liver ability to function.*

Clinical findings: *Mild grade, intermittent fever with chills, yellowish discolouration of skin, Clay coloured stools, Vomiting, Reduced Oral intake.*

Diagnostic evaluation: *CBC: TLC- 17500/cu.mm, Hb- 8.5gm% , RBC- 3.74 millions /cu.mm LFT: Total Bilirubin- 8.1mg/dl, Bilirubin Conjugated- 6.7 mg/dl, Bilirubin Unconjugated- 1.4 mg/dl, SGOT-248 IU/L, SGPT-419 IU/L, Alkaline phosphatase 316 IU/L, Total protein – 9.3gram/dl; Ultrasonography: Hepatospleenomegaly with thickened edematous gallbladder wall. Anti HAV: 0.30 positive.*

Therapeutic intervention: *Medical Management: Inj. Cefotaxime 650mg BD IV, Inj. Ciprofloxacin 130mg TDS IV, Tab. Udiliv 150mg BD Orally, Inj. Piptaz 1.3gram in 300 ml NS IV, syp. Meftal- p 6ml SOS Orally, Syp. Becasul 5ml BD orally, Syp. Duphalac 10 ml HS.*

Outcomes: *The medication has started for hepatitis A . The fever was reduced, Yellowish discolouration of skin was diminished, the oral intake was increased and the level of Bilirubin was Normal. The child showed improvement.*

Conclusion: *The patient was admitted to paediatric ward, AVBRH with known case of hepatitis A and he had a complaint of mild grade, intermittent fever with chills, yellowish discolouration of skin, clay coloured stools, vomiting, reduced oral intake. After getting proper treatment his condition was improved.*

Key words: Hepatitis A, Feco-oral route, Hepatosplenomegaly, Picornavirus, Yellowish discoloration

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INTRODUCTION

The most frequent type of acute viral hepatitis is hepatitis A. Hippocrates is credited with the first description of hepatitis (epidemic jaundice), While hepatitis A epidemics have been observed for a long time, both army and urban communities are affected. HAV is a single-stranded RNA virus that causes hepatitis, which is tiny and non-enveloped. It's acid-resistant and thermostable. HAV was assumed to be an enterovirus for a long time after its discovery; it was discovered in 1991, as a member of the Picornaviridae family's Hepatovirus genus. HAV replicates in hepatocytes, disrupting liver function and triggering an immunological response those results in liver inflammation. The fecal-oral pathway is used to acquire HAV. Person-to-person transmission is widespread, though it is usually restricted to close relationships [1].

The hepatitis A virus is found all throughout the world. The highest, though, rates of hepatitis A are found in poorer nations and territories those due to ill health. Traveler-acquired disease is responsible for about half all instances that have been reported in developed nations.

Hepatitis A endemicity is high in Brazil, according to Luxemburger and Dutta, which means that more than 90% of children aged 5 to 14 were seropositive for the virus. There's been a change in the disease's epidemiology since that time. In 2018, they checked the Epidemiological Bulletin of the Brazilian Ministry of Health, It is easy to see that the incidence rate of hepatitis A has been steadily declining since 2007,in 2017; the number of cases fell from 7.1 to 1.0 per 100,000 people. Between 1997 and

2017, a lot happened. The majority of hepatitis A cases were in youngsters under 10 years old (53.8 percent). There has been a rise in the number of people who have been diagnosed with the condition those aged 20 to 39 years old in the last two years, as well as a shift in the contamination path. There had been a considerable decrease in instances involving contamination of food and a considerable the rise in instances involving transmission through sexual contact. The decline in occurrence happened before the introduction of the universal vaccine.

The Brazilian vaccination schedule did not include universal immunization for children aged 12 months and up until 2014. In terms of hepatitis A mortality in Brazil, There were 1125 people that died linked to Hepatitis A virus between 2000 and 2016. There is currently no data on mortality among Brazilian youngsters following the introduction of the universal vaccine.

Person-to-person HAV outbreaks involving drug users or persons who are homeless have recently been reported in the United States, which could indicate there has been a change in the epidemiology of HAV infection [2].

Children who have hepatitis A before the age of six usually have no or moderate symptoms (70 percent), and if they do become unwell, they usually recover in less than two decades. Adults and children in their teens and twenties who contract hepatitis A maybe there are no signs or symptoms or only a weak disease (30%), but the majority develop so the signs and symptoms of jaundice (70 percent). A weak disease may last 1-2 weeks, whereas a more serious condition may last months. Jaundice, dark-coloured urine, light-coloured stool, Tiredness, Belly discomfort, Decreased appetite, Nausea, Diarrhoea, and Fever are some of the most frequent symptoms of HAV infection. The “Hepatitis A IgM Antibody test” is a test of the blood for the treatment of hepatitis A. This test will indicate IgM antibodies that are positive in people who are currently infected with hepatitis A. While the majority of people recover entirely from hepatitis A, a tiny percentage of people, those with a history of liver illness are disproportionately affected experience severe injury to the liver that can lead to demise. HAV is well-protected with the immunization against hepatitis A. The vaccination is both useful and safe. The vaccination is given in two doses (shots) separated by 6-12 months. After two dosages, Protection is guaranteed for a period of 20 years to life. Starting at the age of one year, The American Academy of Paediatrics is a group of doctors that specialize in children's health recommends that all children get vaccinated against hepatitis A. All travellers to countries where the HAV virus is prevalent should get a hepatitis A vaccine, according to the CDC. At the age of 6-11 months, a prophylactic dosage of hepatitis A vaccination can be administered to infants who would be traveling internationally [3].

Patient identification

A male child of 4 years old from Digras, Yawatmal was admitted to Pediatric WARD No 22, AVBRH on 25 may 2021 Diagnosed as the case of Hepatitis A. He weighs 13 kgs with height of 105.5 cm. Head circumference is 46 cm.

Present medical history

A male child aged 4 years old was brought to AVBRH on 25 MAY 2021 by his parents with complaints of Mild grade, intermittent fever with chills since 20.

Days, yellowish discolouration of skin since 10 days, Clay coloured stools, Vomiting 1 episode per day since 8 days, Reduced Oral intake for which he was admitted to paediatric ward no 22. He is a case of Hepatitis A and his bilirubin level was 8.1 mg/dl.

Past medical history

No any past medical history.

Family History

Not significant.

Past interventions and outcome

No any past interventions.

Clinical findings

Mild grade, intermittent fever with chills, yellowish discolouration of skin, clay coloured stools, vomiting, reduced oral intake.

Etiology

The faecal-oral pathway is how HAV spreads from person to person.

The virus has been discovered in the blood after hepatic replication and is inevitably eliminated into the feces through the biliary system by day’s10–12 minutes after exposure

The major method of transmission is excrement from a sick person that is transmitted to water and food by insufficient hand hygiene.

The virus can also be transmitted by food from a contaminated source that is undercooked (cooked at less than 185°F) or through a food worker who has been infected. Additionally, international travel to HAV endemic regions is possible. People who engage in high-risk activities (such as the use of illicit substances or intercourse, which necessitates oral-anal contact such as MSM) are more likely to develop the virus. Individuals suffering from blood clotting diseases such as haemophilia, may contract the virus by administering blood products such as factor VIII and IX concentrates that have been solvent/detergent-treated, albeit this is a rare occurrence. Animal handlers who deal with nonhuman primates are also at danger since they come into touch with the animal's saliva of contracting the

illness. HAV has not been found to be transmitted by human saliva. In pregnant women, there has been no evidence of HAV transmission from mother to foetus [4].

Physical examination

It was found that the patient had Hepatosplenomegaly from ultrasonography, on through Examination from head to foot, yellowish discoloration of skin and sclera was noted. The child is weak, thin and has dull look as he has Grade I malnutrition.

Diagnostic assessment

CBC: TLC- 17500/cu.mm, Hb- 8.5gm% , RBC- 3.74 millions /cu mm.

LFT: Total Bilirubin- 8.1mg/dl, Bilirubin Conjugated- 6.7 mg/dl, Bilirubin Unconjugated- 1.4 mg/dl, SGOT-248 IU/L, SGPT-419 IU/L, Alkaline phosphatase 316 IU/L, Total protein–9.3gram/dl

Ultrasonography: Hepatosplenomegaly with thickened oedematous gallbladder wall.

Anti HAV: 0.30 positive.

Therapeutic intervention

Medical Management: Inj. Cefotaxime 650 mg BD IV, Inj. Ciprofloxacin 130mg TDS IV, Tab. Udiliv 150mg BD Orally, Inj. Piptaz 1.3gram in 300 ml NS IV, syp. Meftal- p 6ml SOS Orally, Syp. Becasul 5ml BD orally, Syp. Duphalac 10 ml HS.

DISCUSSION

A male child of 4 years old from Digras, Yawatmal was admitted to paediatric ward no.22 in AVBRH on 25 may 2021 with the complaints of mild grade, intermittent fever with chills since 20 days ,yellowish discolouration of skin since 10 days, clay coloured stools, vomiting 1 episode per day since 8 days, reduced oral intake. He is known case of hepatitis as soon as admitted to hospital investigation was done and appropriate treatments were started. After getting treatment he shows great improvement and the treatment was still going on till my last date of care.

A study was done, a 3-year-old child was admitted to the out-patient section of a Sub-District Hospital in Ballabgarh, Faridabad district, Haryana, with a 10-daya history of on-again, off-again frenzy, as well as seven days of abdominal pain and distension. The youngster was steady in terms of haemodynamic, and exhibited icterus and hepatomegaly with a liver spread of 12 cm on examination.

First testsexposeda haemoglobin level of 11.6 g/dl, 12,400/mm3 total leukocyte count, a platelet count of 216,000/mm3, and no abnormal cells in the peripheral smear. She also developed conjugated hyperbilirubinemia (total bilirubin 3.5 mg/dl, conjugated fraction 1.5 mg/dl), transaminitis (aspartate aminotransferase 680 U/L, alanine aminotransferase 745 U/L, and alkaline phosphatase 2,632 U/L), and transaminitis (aspartate aminotransferase 680 U/L).

Ultrasound revealed a minor pleural effusion on the right side, a 4 mm gall bladder wall thickening indicative of calculus cholecystitis, significant Hepatomegaly and excess fluid in the abdomen. A positive anti-hepatitis a immunoglobulin M serology was found during the etiological investigation. Hepatitis B, C, and enteric fever serologist were all negatives.

According to this article, Serial clinical examinations, biochemical, and radiological exams are the only ways to handle such a situation conservatively [5].

According to another study which was done by Foster a, Hernandez S. in 2019, Patients with HAV can present with a wide range of symptoms, from asymptomatic to fulminant (i.e., severe, abrupt onset) liver failure. Although many of the people who are infected are asymptomatic, malaise, fever, and anorexia are the most common presenting symptoms if they do arise. Nausea, vomiting, diarrhoea, stomach discomfort, and the development of jaundice are usually followed by these vague symptoms. Nausea, vomiting, diarrhoea, abdominal pain, and the development of jaundice are usually followed by these vague symptoms.

In contrast, fulminant HAV is extremely rare, occurring in only 1-2 percent of patients. 4 Hepatic failure and increasing encephalopathy (i.e., brain pathology) over a period of days describe this condition [6]. A number of studies on different types of hepatitis were reported by Kawalkar et al. [7], Balwani et al. [8], Pratapa et al. [9], Zanwar et al. [10], Vaidya et al. [11]. Other studies on liver disorders were reviewed to compare the clinical pictures [12-15].

CONCLUSION

In affluent countries, hepatitis A accounts for 20–25 percent of all instances of clinical hepatitis. This infection is caused by picornavirus, a cubically symmetrical ribonucleic acid (RNA) with a diameter of 27 nanometres.

During the acute phase of an infection, the viral antigen (HAAG) can be identified in serum, feces, and the liver. The IgM antibody emerges at first, but then fades away after a few weeks. It is replaced by the development of IgG, which lasts for the rest of one's life (anti-HAV). For acute hepatitis A infection, the IgM antibody is used as a marker. IgG anti-HAV indicates previous hepatitis A virus exposure and immunity to recurrent infection. There is no carrier status for this type of hepatitis, and it does not contribute to the development of cirrhosis or chronic active hepatitis. Hepatitis A patients have a favorable prognosis and complete recovery. Large epidemics have mortality rates of less than 1 per 1000 people. There are no long-term consequences or progression to a chronic illness condition.

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