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# Autoimmune hepatitis in an eight-month-old child

**Arz Muhammad, Ghous Bux Soomro, Muhammad Adeel, Raja Taha Yaseen, Zain Majid*, Nasir Hassan Luck**

**Abstract**

Autoimmune hepatitis (AIH) is considered a rare entity in hepatitis B infection in endemic regions being even fewer in the pediatric population (2). AIH is categorized into types 1 and 2, which are differentiated by their autoantibody profiles. Here we consider a case of an eight-month-old female child who was referred to our hospital with hepatocellular type of jaundice. On further workup, she was diagnosed to have AIH and was later on started on prednisolone. This case report highlight the importance of considering AIH in any pediatrics patient presenting with features of chronic liver disease.

**Keywords:** Autoimmune hepatitis, Autoantibodies, Chronic hepatitis

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Autoimmune hepatitis (AIH) is a chronic inflammatory disorder of the liver, which is characterized by liver function test abnormalities shown by raised levels of serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), raised serological markers (IgG), the presence of autoimmune serology and characteristic findings on liver biopsy namely interphase hepatitis (1). AIH is rarely found in the pediatric population. Approximately 0.1 to 0.23 per 100 000 cases of AIH in the pediatric population are reported (2). AIH has two types main; AIH type-I (AIH-I) and AIH type-II (AIH-II). The former is manifested by patient positive for serum ANA and/or anti-smooth muscle antibody (anti-SMA) while the latter is manifested by patient positive for anti-liver kidney microsomal type 1 antibody (anti-LKM-1) and/or for anti-liver cytosol type 1 antibody (anti-LC-1) (3). AIH has a rapid progression in its course in pediatric population. Hence, a critical response of early treatment is essential to halt any progress of cirrhosis (4). This study presents an example of AIH in eight months old child; significantly aiming to highlight the fact that AIH can be present in any age group.

An eight-month-old child with a history of yellow discoloration of skin and sclera for four months, presented to our pediatric gastrointestinal (GI) outpatients' department. The child was healthy four months back with no complaint. There was no history of abdominal distention, vomiting or diarrhea or change in stool color. Along with that no associated account of skin rash, bruises or pruritus. Further, no history of contact with any jaundiced patient, or raw milk ingestion. Moreover, her parents denied any history of blood transfusion, nor was she was not on any medication.

Regarding her neonatal history, she was born at full-term and had an uneventful pregnancy, with normal delivery requiring no neonatal intensive care unit (NICU) admission. Her developmental history was appropriate for her age. Family history revealed consanguinity and she had four siblings, all were healthy with no similar ailment in her family. Social history revealed that the patient belonged to a low-socioeconomic strata. Her vaccination history was up-to-date, while nutritionally she was dependent on mother milk and dietary supplements as advised by dietitian.

On examination, she was active and alert, looked pale and jaundiced. Vital signs showed that heart rate was 120 beats/minute, temperature was 37°C, blood pressure was 110/60 mm Hg, and respiratory rate was 26/minute. Abdomen was soft and not distended. The liver was 2cm below the costal margin with a liver span of 9 cm. The spleen was palpable about two cm below the costal margin. Chest, cardiovascular examination and nervous system examination were unremarkable. Eye exam, musculoskeletal and skin were normal with no lymphadenopathy.

Laboratory work was carried out. Table 1 shows the complete blood picture of the patient. Table 2 shows her coagulation profile while renal and liver functional tests

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Department of Gastroenterology, Sindh Institute of Urology and Transplantation, SIUT, Karachi, Pakistan

***Corresponding Author:** Zain Majid, Email: Zain88@hotmail.com

**Implication for health policy/practice/research/ medical education**

This study presents an example of AIH in an eight-month old child; significantly aiming to highlight the fact that AIH can be observed in any age group.

are summarized in Table 3. Erythrocyte sedimentation rate (ESR) was 28 mm/h. Blood culture was negative. Urine analysis, culture and TORCH profile was negative. Other pediatric liver diseases like progressive familial intrahepatic cholestasis and tyrosinemia were also ruled out

Therefore, we sent her hepatitis serology tests hepatitis A (HAV IgM), hepatitis B (HBsAg and HBc IgM) hepatitis C (anti-HCV Ab), hepatitis E (HEV IgM). Her autoimmune panel showed that antinuclear antibodies (ANA) was positive while anti-smooth muscle antibodies (ASMA), anti-mitochondrial antibodies (AMA,) liver kidney microsomal antibodies (anti-LKM) and anti-SLA were negative. Serum immunoglobulin g (IgG) was 24 mg/dL, while serum ceruloplasmin level was within normal amount.

Ultrasound-abdomen showed a liver span of 9.0 cm, heterogeneous in texture and with irregular margins. No intrahepatic ductal dilatation was seen. Portal vein of 0.6 cm, gallbladder was normal. Pancreas appeared normal. Spleen was enlarged (12 cm). Both kidneys were normal. No ascites were seen.

CT scan abdomen showed heterogeneous echotexture of the liver with patchy ill-defined hyperechoic area might represent focal fatty infiltration. Therefore, we proceeded with her liver biopsy that revealed marked expansion of portal tracts with mixed inflammation cell infiltrate

**Table 1.** The complete blood count data

| Hemoglobin | PCV | MCV | TLC | Platelets |
|---|---|---|---|---|
| 9.2 g/dL | 28.2% | 89.5 fl | 14.4/µL | 267/µL |

PCV, packed cell volume; MCV, mean corpuscular volume; TLC, total leukocyte count

**Table 2.** Coagulation profile

| PT | | APTT | | INR |
|---|---|---|---|---|
| Control | | Control | | 2.03 |
| Patient | 21.2 | Patient | 37.3 | |

PT, prothrombin time; APTT, activated partial thromboplastin time; INR, international normalized ratio.

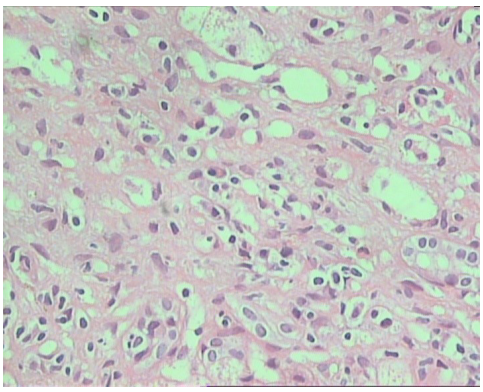

**Figure 1.** Photomicrograph showing expanded portal tract with plasma cell rich infiltration and rosette formation (H&E stain ×400)

including neutrophils, lymphocytes, eosinophils and occasional plasma cells and fibrosis. Periportal and intra-parenchymal fibrosis was also observed (Figure 1). Florid piecemeal necrosis and foci of lobular inflammation were seen. Bile ducts were ductular proliferation and prominent resetting of hepatocytes was observed.

Features were suggestive of AIH. The patient was treated with syrup prednisolone along with multivitamins and minerals and was later discharged.

## Discussion

AIH is usually diagnosed in children before they reach the age of years or mainly before the onset of puberty. Almost 75% of the affected pediatric population are females (5). Thus, AIH affecting male children is less likely.

AIH is very less common in Asian children. If not treated properly, AIH over years develops into cirrhosis and can cause frequent fatalities. Hence, a proper and early diagnosis is essential. However, mortality rate decreases with treatment (6).

This patient revealed only high IgG level after the child was ill for four months. Other possible etiologic factors were excluded. In order to reach clear diagnosis of AIH, these are essential criteria (7).

Liver biopsy is prerequisite to diagnose and evaluate the infection status. It also helps to determine the need for therapy.

Research reports have revealed 50% of pediatric population infected by AIH have developed cirrhosis at presentation (8). The same was true in our case. Thus, pediatricians should consider AIH in children that are presented with chronic liver disease. Conclusively, though less common among children, the AIH should be kept under differential diagnosis of both acute and chronic liver

**Table 3.** Renal and liver profile

| Creatinine | Urea | TB | Direct bilirubin | AST | ALT | GGT | ALP | Albumin |
|---|---|---|---|---|---|---|---|---|
| 0.42 mg/dL | 11 mg/dL | 15.87 mg/dL | 8.91 mg/dL | 201 IU/L | 284 IU/L | 74 mg/dL | 683 IU/L | 2.8 g/dL |

AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; ALP, alkaline phosphatase; TB, total bilirubin

disease. Simultaneously excluding the relative commonly occurring viral and metabolic infections. Only untreated AIH progresses to cirrhosis and early treatment of AIH improves the rate of survival.

### Authors' contribution

AM, RTY and GBS managed the patient. MA wrote the initial draft. ZM wrote the final draft. NHL left critical comments on the final draft.

### Conflicts of interest

The authors declare that they have no competing interests.

### Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors. The patient's parents gave the consent to publish it as a case report.

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