



Journal Homepage: -www.journalijar.com
**INTERNATIONAL JOURNAL OF
 ADVANCED RESEARCH (IJAR)**

Article DOI:10.21474/IJAR01/19217
 DOI URL: <http://dx.doi.org/10.21474/IJAR01/19217>



RESEARCH ARTICLE

**RARE HEMATOLOGICAL COMPLICATION OF HEPATITIS A IN CHILDREN:
 BONE MARROW APLASIA IN A CASE REPORT**

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Manuscript Info

Manuscript History

Received: 31 May 2024
 Final Accepted: 30 June 2024
 Published: July 2024

Abstract

Introduction: Complicated forms of hepatitis A, particularly hematological complications, are rare and are described as severe conditions in children.

Case Report: This case involves a 4-year-and-4-month-old girl who initially presented with a cholestatic jaundice flare due to viral hepatitis A. The symptomatology became complicated a month later by a hemorrhagic syndrome consisting of recurrent epistaxis, pallor, and asthenia, evolving in a context of prolonged fever and deterioration of the general condition. Clinical examination found a febrile child at 39°C, generalized cutaneous-mucosal pallor, petechial purpuric spots on the trunk, and no hepatosplenomegaly. The hematological workup showed aregenerative pancytopenia with normal transaminases. The myelogram revealed a poor bone marrow. Bone marrow biopsy confirmed bone marrow aplasia. Vitamin B12 and B9 levels were normal. Serologies for HBV, HCV, EBV, and CMV were negative. HVA IgG serology was positive with borderline HVA IgM. The patient was put on transfusion support with third-generation cephalosporin and aminoglycoside antibiotics. The diagnosis of post-hepatitis acquired bone marrow aplasia was confirmed. Treatment with cyclosporine was started, leading to good clinical evolution and improvement of the hemogram after two months of follow-up.

Conclusion: Although hematological complications of hepatitis A are still rare, they remain fatal in the majority of cases. This underscores the need for biological and clinical monitoring in the management of hepatitis A in children.

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Introduction:-

Hepatitis A is a common viral infection in children, primarily linked to hygiene conditions. It often presents with mild symptoms and generally has a good prognosis. However, complicated forms can develop, including hematological complications, whose prognosis can vary [1]. We report a case of viral hepatitis A complicated by bone marrow aplasia. The aim of this work is to highlight the existence of rare hematological manifestations complicating viral hepatitis A in children.

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Case Report:

This case concerns a 4-year-and-4-month-old girl who initially presented with a cholestatic jaundice flare due to viral hepatitis A. The symptomatology became complicated a month later by a hemorrhagic syndrome consisting of recurrent epistaxis, with pallor and asthenia evolving in a context of prolonged fever and deterioration of the general condition. Clinical examination found a febrile child at 39°C, generalized cutaneous-mucosal pallor, petechial purpuric spots on the trunk, and no hepatosplenomegaly. The hematological workup showed aregenerative pancytopenia with normal transaminases (Table 1). The myelogram revealed a poor bone marrow. Bone marrow biopsy confirmed bone marrow aplasia. Vitamin B12 and B9 levels were normal.

Table 1: - Biological results of our patient at admission.

Laboratory parameter	Values	Reference Ranges
White blood cell (/ μ L)	2010	5200-11000
Neutrophil (/ μ L)	810	1500-7000
Hemoglobin (g/dL)	6,8	10,5-13,5
Mean corpuscular volume (fL)	86	80-98
Mean corpuscular hemoglobin concentration (%)	29	27-32
Platelets (/ μ L)	10000	150000-400000
Reticulocyte (/ μ L)	12000	20000-80000
SGPT(U/l)	28	5-34
SGOT(U/l)	23	5-55

SGOT:serum glutamic-oxaloacetic transaminase

SGPT: serum glutamic-pyruvic transaminase

Serologies for HBV, HCV, EBV, and CMV were negative. HVA IgG serology was positive at 9.43 (VN < 1.10) with borderline HVA IgM at 1.25 (VN < 1.10). The patient was put on transfusion support with third- generation cephalosporin and aminoglycoside antibiotics. The diagnosis of post- hepatitis acquired bone marrow aplasia was confirmed. Treatment with cyclosporine was started, leading to good clinical evolution and improvement of the hemogram after two months of follow-up.

Discussion:-

Among the extrahepatic complications of viral hepatitis A, hematological manifestations are the most frequent, occurring regardless of the clinical or biological intensity of the liver disease [1]. Post-hepatitis bone marrow aplasia is severe, occurring within six months following at least one episode of rapidly onset clinical hepatitis, usually seronegative for known hepatitis viruses [2]. Our patient presented with bone marrow failure syndrome two months after hepatitis A. Diagnosis is based on the notion of viral hepatitis preceding the aplasia. However, asymptomatic forms of viral hepatitis can go unnoticed, leading to a mistaken diagnosis of idiopathic aplasia. Clinical signs often include pallor with multiple skin hemorrhages [3], lymphopenia, hypogammaglobulinemia [4], and febrile neutropenia [5]. Bacterial and fungal infections may appear secondarily in the presentation of the disease [6]. Further complications, including myelodysplasia, can develop [7]. Our patient also presented with the same clinical and biological signs described in the literature, with febrile neutropenia resolving quickly after ten days of probabilistic antibiotic therapy without identified germs in the blood culture.

The prognosis for post-hepatitis bone marrow aplasia is fatal in the majority of cases, with mortality ranging from 78% to 88% [8,9]. In this presented case, the hepatitis had a favorable outcome, as did the pancytopenia after two months of cyclosporine treatment. A similar published case [10] also showed good progress under immunosuppressive therapy with gradual improvement of the hemogram. However, there is no etiological treatment, and the severity of the spontaneous evolution justifies attempts at bone marrow transplantation. Unfortunately, in our country, bone marrow transplantation, especially allografts, lags significantly behind other countries in the region.

Conclusion:-

Although hematological complications of hepatitis A are rare, they remain severe and fatal in the majority of cases

described in the literature. This implies the need for biological and clinical monitoring in the management of hepatitis A in children.

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