Hepatitis A in a Patient with Acute Lymphocytic Leukemia

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Abstract: Precursor B-cell acute lymphoblastic leukemia accounts for 2% of all lymphoid neoplasms in the United States. A five-year-old boy presented with a case of acute lymphocytic leukemia (ALL), which was the first case in literature depicting chemotherapy or ALL causing hepatitis A. As such, we report this case for its rarity. Informed consent was received from the patient before undertaking and reporting this study.

Keywords: ALL, Hepatitis A, Chemotherapy

1. INTRODUCTION

Precursor B-cell acute lymphoblastic leukemia accounts for 2% of all lymphoid neoplasms in the United States and occurs most frequently in childhood. Childhood acute lymphoblastic leukemia is highly responsive to chemotherapy, so it is a standard of care treatment [1-3].

In the literature, there were numerous references of temporal and causal relationships of non-specific hepatitis and hepatitis A to ALL. However, this is the first case depicting chemotherapy or ALL causing hepatitis A.

We report a case of ALL precursor B-cell ALL undergoing chemotherapy that lead to hepatitis A, which is the first reported case in literature. Nonspecific hepatitis was discussed in the literature, but we specifically describe ALL or chemotherapy-induced hepatitis A, which has not previously been reported in literature [4].

Furthermore, chemotherapy also leads to pulmonary complications, as evidenced by the bronchopneumonia in this patient [5].

2. CASE REPORT

A five-year-old boy presented with lower back pain on April 25. The next day, he developed a sudden onset, high grade, documented fever of 103 F without rigors and chills, which was temporarily relieved by medications. On May 3, his CBC showed thrombocytopenia (41000), and the differential leukocyte count indicated 50 percent lymphocytes.

The boy experienced no change in weight for the prior four months. Specifically, his recent measurements were: weight 16 kg (15th percentile), height 109 cm (below mean), and occipitofrontal circumference of 50 cm (below mean). In addition, a gastrointestinal examination showed tender hepatomegaly (liver was 6 cm below the costal margin) and splenomegaly (spleen was 4 cm below the costal margin), with an intact motor and sensory system.

The CBC indicated Hb 6.9 g/dl, WBC 7.1 × 103 (out of which 88% were lymphocytes, i.e. lymphocyte predominant), and platelets 34 × 103. Also, the dengue serology was non-reactive, and the hepatitis B and C screenings by ELISA, as well as the direct Combs’ test were all negative. The bone marrow biopsy performed on May 19 indicated Sudan black b negative acute leukemia FAB type L2, precursor B lymphoblastic leukemia. A whole body scan was performed as well, which turned out to be normal, as shown in Figure 1.

Repeted bone marrow aspirate on July 7 showed morphological remission of the bone marrow. The patient developed acute viral hepatitis (IgM positive) on his 3rd day of consolidation chemotherapy (on July 19), which continued until day 18 of consolidation. Moreover, on July 30, he developed febrile neutropenia and thrombocytopenia, suspecting chemotherapy-induced myelosuppression even after supplementation with filgrastim. The patient’s consolidation phase was completed on August 16, 2018.
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The echocardiography of the patient depicted a structurally and functionally normal heart. Additionally, the ultrasound showed pericholecystic edema, bilateral nephromegaly, bilateral renal parenchymal changes, and hepatomegaly. Also, the Typhidot IgG was positive, and the HCV was not detected by PCR.

The patient then developed bronchopneumonia, and medical staff decided to delay the next phase of chemotherapy for 14 days. Nevertheless, the patient again developed acute viral hepatitis in the next phase of chemotherapy, i.e. intermittent maintenance.

3. DISCUSSION

This five-year-old boy developed hepatitis A, which showed tender hepatomegaly and fever on physical examination. His low hemoglobin and lymphocyte predominant WBCs were thought to be due to acute lymphocytic leukemia and chemotherapy. Hepatitis A and other viral hepatitis have a causal relationship with ALL as mentioned above. In fact, this case depicted that ALL and/or chemotherapy caused the hepatitis A. Nevertheless, hepatitis A can also be caused by poor sanitation, as there are many cases of viral hepatitis due to the fecal-oral route. Thus, infection by contamination should be ruled out first, especially in a country like Pakistan, where hepatitis A is endemic.

Furthermore, the patient had febrile neutropenia and thrombocytopenia, thought to be caused by chemotherapy [6]. The Typhidot test was positive with neutropenia, which was also a complication of chemotherapy [7].

Nephromegaly and parenchymal changes indicated Fanconi syndrome but without the usual clinical presentation. Nonetheless,
Fanconi syndrome can sometimes appear with atypical presentation [8]. Future studies should focus on the role that ALL and/or chemotherapy may play in causing hepatitis A and related complications, as well as how targeted treatment strategies may improve prognosis.

REFERENCES


