CASE REPORT

Amebic Liver Abscess and Kawasaki Disease

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Background: Co-occurrence of amebic hepatitis and Kawasaki disease has not been reported previously. **Case characteristics:** We describe two children (aged 4 y and 5 y) with Kawasaki disease and coexisting liver abscess. They were treated with intravenous immunoglobulins with/without percutaneous drainage in combination with amebicidal agents. **Outcome:** Both the children were completely cured of the amebic hepatitis, and had normalization and regression of coronaries at follow-up. **Message:** We report the co-existence of amebic hepatitis with Kawasaki disease.

Keywords: *Fever of unknown origin, Superantigen, Vasculitis.*

Kawasaki disease (KD) results from an interaction of a host of infections (superantigen theory), or a canonical response to a conventional antigen with genetic predisposition [1,2]. The search for the elusive superantigen still continues with streptococcal, staphylococcal, and more recently, candidal elements being thought of as possible triggers [3,4]. Coexisting infections like dengue have been reported [5,6]. We report 2 cases of co-occurrence of amebic hepatitis with KD.

CASE REPORT

Case I

A 4-year-old boy was referred with persistent fever for 18 days and abdominal pain. Ultrasonography abdomen done on day 10 and day 14 outside had revealed an abscess (4 cm × 4.8 cm × 3.8 cm) involving the right hepatic lobe. He had received multiple antibiotics and amebicidal drugs with persistent pyrexia. Examination revealed significant mucositis with erythema and fissuring, pedal edema, and bulbar non-purulent conjunctivitis with perilimbal sparing. No lymphadenopathy or rash was reported. Tender hepatomegaly was present. Laboratory findings on admission (day 18) revealed anemia (Hb 7.6 g/dL), leucocytosis (Total counts 16.8 × 10^9/L, (P44%, L36%), thrombocytosis 1070×10^9/L), elevated acute phase reactants (ESR 112 mm/h and CRP 49.5 mg/L) and hypoalbuminemia (2.5 g/dL). Repeat blood, urine and throat cultures were sterile. Repeat USG was similar to previous scans with no liquefaction. With a suspicion of a coexisting incomplete KD, a transthoracic 2D echocardiogram was done which showed significantly dilated coronary arteries with a proximal right coronary artery saccular aneurysm (RCA Z score 4.31), left main coronary (LMCA Z Score 3.22), left anterior descending artery (LAD-Z score 2.55). Indirect hemagglutination (IHA) test for IgG Ameba antibody was positive. With no response to antibiotics and the positive clinical and cardiac findings, IVIG was administered at 2 g/kg, with complete resolution of pyrexia within 36 hours. Child was discharged on anti amebicidals and low dose aspirin. He returned after a week with desquamation of finger tips confirming our diagnosis. USG improvement and normalization of echo-cardiographic findings were noticed over 10 weeks and 6 months, respectively.

Case II

A 5-year-old girl presented with a history of remittent fever of 7 days, redness of lips and swelling on left side of neck. A history of a macular rash and non-purulent conjunctival congestion on days 3 and 4, respectively was elicited. Clinical examination revealed mucositis with erythema and fissuring, pedal edema, and bulbar non-purulent conjunctivitis with perilimbal sparing. An enlarged left anterior cervical lymph node were also (>1.5 cm), and mild pedal edema were also noted. Her vitals were stable with mild tachycardia (Heart rate 102/min) and respiratory rate of 30/min with normal blood pressure (94/60 mm Hg). Laboratory findings on admission revealed anemia (Hb 7.6 g/dL), leucocytosis (total counts 21.4 × 10^9/L, P 83%, severe thrombocytosis (1071× 10^9/L), elevated acute phase reactants (ESR 90 mm/h and CRP 380 mg/L) and hypoalbuminemia (1.5 g/dL). With negative cultures, a trans-thoracic 2D echocardiography showed LMCA- Z score 0.06, LAD -Z score of 1.91 and RCA -Z score of 5.97.

IVIG was administered at 2 g/kg with a diagnosis of KD. Post IVIG, with persistent but spaced fever spikes, she developed increasing respiratory distress and tender hepatomegaly. Ultrasonography showed a 7.4 × 6.5 × 6.4 cm liquefied abscess with mild pleural effusion. IHA for IgG ameba antibody was positive. Percutaneous drainage revealed 200 mL fluid with an 'anchovy sauce' like appearance, and administration of parenteral amebicidal drugs led to complete resolution of symptoms over the next 3 days. She was discharged on low dose aspirin, and subsequent desquamation at the end of a week confirmed the diagnosis of KD. Subsequently she showed a steady regression of her coronary artery abnormalities over 3 months, and was continued on low dose aspirin with regular echocardiographic monitoring.

DISCUSSION

Multiple infectious agents have been implicated as triggring agents for KD, including viruses, bacteria, rickettsiae and even candidal agents [1]. Adding to the list of coexisting infections with KD, we describe the presence of amebic hepatitis through this report. In the first child, a lack of clinical response along with a sequential evolution of clinical signs suggestive of KD prompted us to confirm the diagnosis with 2 D echocardiogram. The second child had classic features of KD from the beginning, diagnosis was confirmed by echocardiography, but child had only a partial response to IVIG (IVIG-resistant). New onset abdominal pain and respiratory distress prompted a USG revealing the liver abscess. Complete resolution of symptoms was achieved with drainage of the abscess and addition of amebicidals. Both children had evidence of severe inflammation in the form of increased acute phase markers and acute hypoalbuminemia. Serological tests (IHA) confirmed the diagnosis of amebic hepatitis in both of them.

A proven presence of an infection should not dissuade the possibility of a co-existing inflammatory condition like KD, if the response to therapy is inadequate or in the presence of suggestive signs. Whether amebiasis was a coexisting infection with inflammation (KD) causing a subclinical response or the potential organism triggering the development of KD is debatable. However, these cases strengthen the theory regarding the possible role of microorganisms with a disordered innate immune system as a possible etiology. While dealing with a suboptimal response to any infection, possibility of underlying inflammatory conditions like KD needs to be considered in clinically suggestive situations.

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REFERENCES

1. Principi N, Rigante D, Esposito S. The role of infection in Kawasaki syndrome. J Infect. 2013;67:1-10.
2. Kawasaki T. Acute febrile mucocutaneous syndrome with lymphoid involvement with specific desquamation of the fingers and toes in children. Arerugi. 1967;16:178-222.
3. Takahashi K, Oharaseki T, Wakayama M, Yokouchi Y, Naoe S, Murata H. Histopathological features of murine systemic vasculitis caused by Candida albicans extract—an animal model of Kawasaki disease. Inflamm Res. 2004; 53:72-7.
4. Ohno N. Murine model of Kawasaki disease induced by mannoprotein-beta-glucan complex, CAWS, obtained from Candida albicans. Jpn J Infect Dis. 2004;57:S9-10.
5. Agarwal E, Shah D, Gupta P. Dengue fever triggering Kawasaki disease. Indian Pediatr. 2017; 54:51-2.
6. Singh S, Jat KR, Suri D, Rathod RK. Dengue fever and Kawasaki disease: a clinical dilemma. Rheumatol Int. 2009;29:717-9.