

Does Streptococcus-A cause hepatosplenomegaly and atypical lymphocytosis?: a case report

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Atypical lymphocytosis and hepatosplenomegaly are seen in many viral or bacterial infections such as infectious mononucleosis, cytomegalovirus (CMV), toxoplasmosis, rubella, pertussis, tuberculosis, brucellosis, typhoid fever, malaria, syphilis (1). Local infections caused by group A hemolytic streptococci are usually confined to tonsils, upper respiratory tract and middle ear. Lymphocytosis and hepatosplenomegaly are not expected to be associated with this type of disorder (2,3). However in recent years, there have been many reports that invasive group A streptococci may be associated with unusual clinical manifestations such as toxic shock syndrome, hepatosplenomegaly, and necrotizing fasciitis (4,5). This report presents the case of an infant with fever, lymphadenopathy, atypical lymphocytosis, hepatosplenomegaly and positive throat culture for group A hemolytic Streptococcus, who exhibited dramatic improvement in clinical and laboratory findings by penicillin treatment.

Case report

A one year old female infant was referred to our hospital in March 1996 for evaluation of hepatosplenomegaly, cervical lymphadenopathy, and fever. Her history revealed that she had been well until 4 weeks previously, when a regular visit to her local physician revealed hepatosplenomegaly. During the 4 weeks of follow up the physician noted that, in addition to continuous fever, there was a gradual increase in the size of the spleen, liver and cervical lymph nodes. Her physical examination at admission showed a fever of 38.5°C and bilateral mobile submandibular lymph nodes 3x3 cm in size. The liver and spleen both extended 5 cm below the respective costal margins.

The hemoglobin was 9 g/dl, WBC count 19.7X10⁹/L with 12% polymorphonuclear leukocytes, 2% monocytes, 24% lymphocytes and 62% atypical lymphocytes containing large cytoplasm. Some serologic tests (HbsAg, Anti-HBsAg, IgG-IgM of Epstein barr virus (EBV), CMV and Toxoplasmosis) which were taken at admission and one week later gave negative results. Bone marrow aspiration smear revealed normocellular marrow with normal myeloid/ erythroid ratio (3/1). Lymphocytes were 20% and, there was no increase in

blasts. The serum iron level was 29 µg/dl, total iron binding capacity 396 µg/dl, and transferrin saturation 7.3%. Group A-hemolytic Streptococci grew in throat culture. The patient was treated by procaine penicillin 400.000 U for ten days. Three days after the initiation of penicillin therapy, there was no fever and the physical examination revealed considerable improvement in hepatosplenomegaly and lymphadenopathy. A week later, the spleen was palpable 1 cm below the costal margin, and the liver at 2 cm and, there was no lymphadenopathy. In the laboratory studies, hemoglobin was 9 g/dl and WBC 8.7X10⁹/L with 60% lymphocytes. There was no atypical lymphocyte (Table I). Three weeks later there were no abnormalities in the physical and laboratory examinations.

Table I. Changes in Clinical and Laboratory Findings by Penicillin Therapy.

Findings	Before Therapy	3rd day
Fever (°C)	38.5	36.5
Liver (ECM)	5cm	2cm
Spleen (ECM)	5	1
Lymphadenopathy	3x3cm	2x2cm
WBC count (X10 ⁹ /L)	19.7	8.7
Lymphocytes %	24	50
Atypical lymphocytes %	62	10

Discussion

Generalized lymphadenopathy, hepatosplenomegaly and fever accompanied by atypical lymphocytosis in the peripheral smear suggested that the child might be infected affected by EBV. A positive throat culture for group A hemolytic streptococci is not an unusual finding in infectious mononucleosis. However, prompt resolution in abnormal clinical and hematological findings by penicillin therapy without any serological evidence for EBV and CMV infections suggested that group A hemolytic streptococci was the responsible etiologic agent in the patient.

In recent years, there have been several reports dealing with invasive Streptococci infections. According to these publications the clinical spectrum of the disease varies from organomegaly to epidermal necrolysis and toxic shock syndrome (4,5). However, lymphocytosis with atypical lymphocytes has not been

reported in any of these patients. We believe that our patient was most probably affected with an invasive Streptococcus infection. This observation indicates that patients with clinical and laboratory abnormalities suggesting the presence of a viral disease should also be evaluated for bacterial infection as well.

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References

1. Feigin RD. Streptococcal infections, In; Behrman RE. Nelson Textbook of Pediatrics. Philadelphia: WB Saunders Company, 1992: 698-702.
2. Dobson S. Group A streptococci revisited. Arch Dis Child 64: 977-991, 1989 .
3. Lanzkowsky P. Disorders of white blood cells. In; Lanzkowsky P. Manual of Pediatric Hematology Oncology. (2ndEd). 1995: 180-182.
4. Davies HD, A, Schwartz B. Invasive group A streptococcal infections in Ontario, Canada. N Engl J Med 335: 547-554, 1996.
5. Holm SE. . Invasive group A streptococcal infections. N Engl J Med 335: 590-591, 1996.

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