

Scarlet Fever and hepatitis: a case report

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Abstract:

Scarlet fever is a streptococcal infection with a good prognosis. Complications are well described. Hepatitis is a rare complication. We describe a 6-year old boy with scarlet fever, jaundice and elevated liver transaminases. Hippokratia 2008; 12 (3): 186-187

Key words: scarlet fever, hepatitis, streptococcus

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Scarlet fever is a common paediatric infection caused by toxin-producing group A beta haemolytic streptococci (GABHS) found in secretions and discharge from the nose, ears, throat and skin. Usually it follows an upper respiratory tract infection; rarely scarlet fever may occur following an infection of the skin and soft tissue, surgical wounds and infections of the uterus. Morbidity and mortality have decreased dramatically over the decades due to improvements in socio-economic conditions and introduction of antibiotics. Complications include otitis media, pneumonia, septicaemia, osteomyelitis, rheumatic fever and glomerulonephritis. Hepatitis in association with scarlet fever has been described in adults¹ and less frequently in children². We report the case of a 6-year old child with hepatitis secondary to scarlet fever.

Description of Case

A 6-year old boy was referred to our department by a private paediatrician for further investigation of jaundice. The patient was diagnosed to have scarlet fever 4 days prior to admission; the diagnosis was based on the following findings: scarlet macules over generalised erythema covering the trunk and extremities, circumoral pallor, tonsillopharyngitis, Forchheimer's spots, white strawberry tongue and Pastia's lines along skin folds over the antecubital fossae. GABHS was isolated from throat swab culture. Jaundice was noted on day 2 of the disease and the patient mentioned dark urine and abdominal discomfort. On admission the patient was already on penicillin for 4 days and the signs of scarlet fever were fading. He was icteric and the liver was tender and palpable 2 cm below the right costal margin.

Laboratory work up revealed haemoglobin 12 mg/dl, leukocytes 11200 /ml with 67% neutrophils, 22% lymphocytes; erythrocyte sedimentation rate of 61 mm/hr; serum C reactive protein was 37 mg/l (normal < 3mg/l). Antistreptolysin O titre was elevated to 481 mg/dl. Bilirubin was 4.4 mg/dl with 3.5 mg/dl of conjugated bilirubin.

Transaminases were considerably high: SGOT 179 U/L, SGPT 199 U/L, γ GT 149 U/L. Amylase, alkaline phosphatase and LDH were normal. Urinalysis revealed bilirubinuria without proteinuria or haematuria. Antibodies for cytomegalovirus, Epstein-Barr virus parvo B19 virus and hepatitis A and C were negative. The patient was immunised against hepatitis B. Abdominal ultrasonography confirmed hepatomegaly but was otherwise unremarkable. The patient was treated with intravenous cefuroxime for 12 days and remained afebrile. Transaminases returned to normal values on day 15 of the disease. Desquamation of the skin at the tips of the fingers and toes was noted on day 11 of the disease. The patient is now in excellent health one-month post initial presentation.

Discussion

Complications of scarlet fever include rheumatic fever, post streptococcal glomerulonephritis and suppurative sequelae (adenitis, sinusitis, cellulitis and abscess formation). Hepatitis is a rare complication of scarlet fever in the paediatric population^{2,3}. In the adult literature it is considered somewhat more frequent^{4,5}, although the exact prevalence is undetermined. The pathophysiologic mechanism is unclear. Direct bacterial injury, toxicity and immunologic mediation have been proposed. Liver biopsies in patients with scarlet fever have shown granulocytic infiltration of the portal areas and hepatocytic degeneration⁶. In most patients the prognosis is excellent and this was confirmed in our case.

We report the case due to the rarity of the association in order to raise the awareness among paediatricians. Scarlet fever is a benign disease but it may rarely have important complications, including hepatitis.

References

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