AN UNUSUAL CLINICAL PRESENTATION OF GROUP A STREPTOCOCCAL INFECTION

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Abstract

After two decades of decline of Group A streptococcal infections, the recent years are witnessing a resurgence in the incidence and severity of infections caused by Group A Streptococcus including necrotizing fasciitis and streptococcal toxic shock syndrome sometimes with fatal outcomes.

We present an unusual case of Group A streptococcal infection in a 4-year-old boy who did not have any predisposing factors for Group A streptococcal infection.

Keywords: Group A Streptococcus; Streptococcal toxic shock syndrome; Bacteraemia

1. Introduction

Group A Streptococcus (GAS) causes common childhood diseases such as streptococcal pharyngitis and impetigo and can cause severe life-threatening invasive disease including streptococcal toxic-shock syndrome (STSS) and necrotizing fasciitis1. Invasive GAS infection was defined as a disease associated with the isolation of Streptococcus pyogenes from a normally sterile body site. A wide variety of clinical syndromes were associated with invasive GAS disease. Cellulitis, upper respiratory tract infection and bacteraemia without focus were the most common syndromes.2 Childhood invasive GAS disease occurs at an incidence similar to the adult population but has a lower rate of streptococcal toxic shock syndrome and case fatality. Chickenpox dramatically increases the risk for acquiring invasive GAS disease. Besides this, children with malignancies, underlying chronic illness, trauma to the skin and soft tissue will predispose to GAS invasive disease.2 We report an unusual case of GAS invasive disease in the absence of any such predisposing factors.

2. Case report:

A previously healthy 4-year-old boy was admitted to our teaching hospital with a history of fever, chills, headache and difficulty in swallowing since 5 days. On examination, he had a high grade temperature (102°F) which was continuous and associated with chills. A throat examination revealed congestion and inflammation with enlarged tonsils and pus points. Lymph node examination revealed palpable cervical lymph nodes. Vital signs like heart rate were 100/minute, respiratory rate was 30/minute and blood pressure was 90/60 mm Hg. There were no cutaneous lesions.

A complete blood examination showed haemoglobin of 12.6g%, a W.B.C count of 11,500/mm³ with 68% neutrophils, 29% lymphocytes, 3% eosinophils and 1% monocytes. There were no abnormal cells in the peripheral smear and no parasites were seen. There were no abnormal findings in the urine and culture was sterile. A gram stain of the throat swab showed 2+ gram positive cocci with numerous polymorphonuclear leucocytes; culture yielded heavy growth of Group A Streptococcus which was sensitive to penicillin, ampicillin, ciprofloxacin, cephalaxin and resistant to erythromycin.
Initially, the patient was treated with ciprofloxacin 250 mg twice daily and antipyretics for 3 days. The fever continued for 3 days and the patient became more toxic and dehydrated. He was then started on intravenous fluids and blood for culture was sent. Blood culture yielded heavy growth of *Group A Streptococcus* after 24 hours incubation with similar antibiotic susceptibility. Intravenous cefotaxime was administered for 5 days. Patient became afebrile after 2 days and was discharged after 5 days of treatment. The patient recovered completely.

3. **Discussion:**

*Streptococcus pyogenes* (Group A in the Lancefield classification) is one of the most important human pathogens and the most frequent cause of acute pharyngitis and produces a variety of skin infections including impetigo, cellulitis, erysipelas, wound infections and gangrene. The recent resurgence of invasive GAS infections is a reminder of the pathogen’s ability to cause severe and even life-threatening infections like bacteraemia, toxic shock syndrome and necrotizing fasciitis. The very young, the elderly and those with underlying conditions are at particularly high risk for invasive disease. Bacteraemia occurs in most cases. Mortality rates can be up to 80%. A study by Laupland et al found the incidence of invasive GAS disease to be 1.9 cases per 100000 per year. Chickenpox infection is the most important risk factor identified for the acquisition of invasive GAS infection in children. Varicella zoster virus infection is associated with a 58-fold increased risk of acquiring invasive GAS disease in children.

Invasive GAS disease can be caused by a number of distinct serotypes producing different superantigens and it has been shown that the response of an individual to different serotypes can be very different and follows the host specific immune response to the infecting strain. Besides, low level of isolate specific neutralizing antibodies may contribute to the risk of invasive GAS disease. But the mechanism by which lack of these antibodies may contribute to increased invasiveness of the organisms is not clear. However, the superantigens are known to cause tissue damage and are capable of activating resident macrophages to produce inflammatory mediators and chemotactic factors. In the absence of neutralizing antibodies, the superantigen-mediated inflammatory reactions may facilitate bacterial invasion of host tissue.

Our case is unusual in the sense that our patient did not have any predisposing factors like Varicella zoster infection, malignancy, immunosuppression, underlying chronic illness, steroid therapy or trauma to skin and soft tissue.

**Conclusions**

Streptococcal sore throat may lead to life-threatening complications like bacteraemia even in the absence of predisposing factors depending upon the pathogenic potential of the infecting strain and the host response to the infecting strain. Considering its recent resurgence, physicians must recognize the early signs and symptoms of invasive GAS infections because of the rapidity with which they progress and their potential for a fatal outcome, therefore the adequate treatment of streptococcal sore throat and early detection of complications and treatment will reduce the mortality in invasive disease.

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