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Peritonsillar abscess – an unusual presentation of Kawasaki disease

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Abstract
Kawasaki disease (KD) is a paediatric illness characterised by prolonged high fever, mucocutaneous lesions and lymphadenopathy. It is potentially fatal as coronary arteritis occurs in up to a third of affected children. We present a seven-year-old child who was admitted to hospital with neck pain and fever. Despite intravenous antibiotic therapy and a quinsy right tonsillectomy on the sixth day after admission, the patient's symptoms persisted. With the appearance of further signs and symptoms the diagnosis of KD was made two days after operation. The patient's symptoms resolved with aspirin and intravenous gammaglobulin therapy. A literature review of the various aspects of KD is presented.

Key words: Peritonsillar abscess; Mucocutaneous lymph node syndrome (Kawasaki disease)

Introduction

Kawasaki described an acute febrile mucocutaneous syndrome with lymphoid involvement and specific desquamation of the fingers and toes in children in 1967 (Kawasaki, 1969). It was initially thought to be benign and self limiting. However, it was soon noted to be associated with potentially fatal cardiac complications. Perionsillar abscess occurs infrequently in KD and clinicians should be aware of this unusual presentation (Rothfield *et al.*, 1990).

Case report

A previously healthy seven-year-old white male presented with a two-day history of right-sided neck pain and fever. On admission the patient appeared ill and irritable. Physical examination revealed a temperature of 38.8°C, congested throat, hyperaemic tympanic membranes and a tender bilateral cervical lymphadenopathy more marked on the right side. Investigations showed a raised white cell count with neutrophilia, a raised c-reactive protein and a normal platelet count. A throat swab was taken and the patient was started on intravenous penicillin.

He continued to have spiking temperatures to 40°C with enlarging cervical nodes and torticollis. An ultrasound scan showed nodes up to 2 cm in diameter but excluded an abscess.

Titres to Epstein-Barr virus, cytomegalovirus, toxoplasma and mycoplasma were not raised. Antistreptolysin O titres were not raised. Culture of the throat swab was negative.

On the fourth day of admission he was noted to have trismus and early signs of right quinsy. Intravenous metronidazole was added to the antibiotic regimen. In the next two days he developed a diffuse macular rash, bilateral conjunctivitis, a strawberry tongue and cracked lips. He continued to spike temperatures to 39.6°C. On the sixth day of admission he underwent a right tonsillectomy under general anaesthesia with release of about 1 ml of pus. This pus did not grow any organisms on culture.

During the next two days the patient continued to spike temperatures and developed arthritis of his knees, shoulders and wrists. He also had bilateral inguinal lymphadenopathy. At this stage he had a platelet count of 493×10^9 (day 9 of his illness). The platelet count reached a high of 746×10^9 by the 15th day of his illness.

The diagnosis of KD was considered and he was treated with aspirin in a dose of 30 mg/kg/day and intravenous gammaglobulin in a dose of 2 gm/kg given over 12 hours. He had a normal electrocardiogram and echocardiogram at diagnosis. On the eleventh day of his illness examination showed desquamation of his fingers and toes. All his systemic symptoms resolved after starting the treatment and he was discharged five days later (two weeks after admission).

He took aspirin in a dose of 150 mg a day for the next eight weeks. Blood tests, electrocardiogram and echocardiogram were normal at eight weeks.

Discussion

This case demonstrates many of the features of KD, a number of which may lead to presentation to an otolaryngologist. The aetiology of KD is still unknown. At present there is no diagnostic test for KD. The diagnosis is established by the recognition of five out of six characteristic clinical criteria and the exclusion of alternative diagnoses which may present similar manifestations (Table I).

Cervical lymphadenopathy is seen in only 50–75 per cent of patients with KD, whereas the other criteria are seen in over 90 per cent of patients. Other manifestations of the disease include polyarticular arthralgia and arthritis, diarrhoea, pneumonia, aseptic meningitis, mild hepatitis with a degree of obstructive jaundice and extreme irritability (Tizard *et al.*, 1991).

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TABLE I
DIAGNOSTIC CRITERIA FOR KAWASAKI DISEASE

(A) Fever of five or more days' duration
(B) Presence of four of the following five conditions:
  (1) Bilateral conjunctival injection
  (2) Change(s)* in the mucous membranes of the upper respiratory tract, such as injected pharynx, dry cracked lips or strawberry tongue.
  (3) Change(s)* in the peripheral extremities, such as oedema, erythema or desquamation (may occur later).
  (4) Polymorphous rash.
  (5) Cervical lymphadenopathy (at least one node greater than 1.5 cm).
(C) Exclusion of: staphyloccocal and streptococcal infection, measles, leptospirosis, rickettsial disease, Stevens Johnson syndrome, drug reaction and juvenile rheumatoid arthritis.

* One of these is sufficient. NB. In the presence of coronary artery aneurysms, (A) plus three of the four criteria in (B) is sufficient.

The most serious consequence of KD is cardiovascular involvement which occurs in 20–30 per cent of patients. The earliest cardiac manifestations occur in the first 10 days of the illness and include myocarditis occasionally complicated by congestive cardiac failure, pericarditis, valvular insufficiency and arrhythmias. Acute coronary arteritis during the first two weeks of illness may lead to coronary artery aneurysm formation. Coronary artery dilatation is first seen at a mean of 10 days after the onset of the illness and the peak frequency of aneurysm formation occurs within four weeks of the start of symptoms. Sudden death occurs in about one to two per cent of all patients with KD and are due to various cardiac complications (Kato *et al.*, 1975). Intravenous gammaglobulin infusions given in high dosage in the first 10 days of the illness considerably reduce the incidence of coronary artery disease. Hence it is essential to make the diagnosis early. Suspected cases should therefore be discussed with or referred to a paediatrician with some experience of the disorder as soon as the possibility of the diagnosis is entertained.

Some children with a prolonged febrile illness and echocardiographically proven coronary artery disease do not fulfil the criteria for KD. It is therefore necessary to be aware of these atypical cases of KD even if only two or three other features of the disease are present (Rowley *et al.*, 1987).

Three phases can be recognised in the evolution of KD. The acute phase lasting seven to 10 days is associated with fever, mucositis, rash and vasculitis. In the subacute phase lasting 10 to 25 days there is resolution of fever, rash and lymphadenopathy. Desquamation of fingers and toes, arthritis, myocardial dysfunction and thrombocytosis become evident during this phase. The convalescent phase begins when all clinical signs resolve and continues for two to three months until all laboratory markers of inflammation have returned to normal levels. In patients with cardiac involvement, a fourth chronic phase may be seen where angina pectoris and myocardial insufficiency due to coronary stenosis may develop (Schaller, 1996). The pathogenesis of KD is unclear but seems likely that activation of inflammatory pathways contributes to vascular damage.

Laboratory findings in KD are not diagnostic but are those of an intense acute inflammatory response. The platelet count is normal in the first week but begins to rise in the second week and peaks at around three weeks from disease onset.

Definitive therapy for KD is possible only when its aetiology is established. Drugs currently used are directed at diminishing the vascular inflammation and risk of thrombosis (Kato *et al.*, 1979). Aspirin should be started as soon as the diagnosis is considered. Recommended doses vary from 30–100 mg/kg/day in the acute stage reducing to an antiplatelet dose of 2–5 mg/kg/day once the fever has declined. Dipyridamole, prostacycline, streptokinase and other drugs have been used successfully in some centres. A single dose of 2 gm/kg intravenous gammaglobulin given as a slow infusion over eight to 12 hours is of proven benefit in reducing the risks of coronary artery abnormalities (Levin *at al*, 1991).

Conlusion

Early recognition and treatment of KD will help to lessen its morbidity and mortality.

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