Acute poststreptococcal polymyalgia

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SUMMARY Three patients developed severe incapacitating diffuse pain and tenderness of the skeletal muscles after acute streptococcal infection. There was no evidence of concomitant arthritis, glomerulonephritis, or inflammatory muscle disease in any of the cases. All patients responded promptly to anti-inflammatory therapy. Severe myalgia should be considered an additional complication of immunologically mediated poststreptococcal response.

Rheumatic fever, acute poststreptococcal glomerulonephritis, Schönlein-Henoch purpura, and erythema nodosum are well recognised, immunologically mediated complications of streptococcal disease.¹⁻⁴ On the other hand, severe diffuse pain and tenderness of skeletal muscles without concomitant arthritis is not a recognised feature of poststreptococcal disease. Recently, we encountered three patients who developed incapacitating acute myalgia after streptococcal infection.

Case histories

CASE 1

A 13-year-old girl was admitted to our department because of severe myalgia involving the upper and lower limbs of three days' duration. Two weeks before admission she had had an upper respiratory infection. She also had fever, arthralgia of the small joints of the hands, and was bedridden because of pain. She had not received any medication. On admission her temperature was 39°C and she had a diffuse giant urticarial rash. The throat, lungs, and abdomen showed no abnormal findings. The heart sounds were normal, and no murmurs were heard. There was severe bilateral tenderness and weakness of the deltoid, biceps, and quadriceps muscles, but there were no signs of arthritis.

Laboratory tests showed an erythrocyte sedimentation rate (ESR) of 80 mm after one hour (Westergen), haemoglobin 11.1 g/dl (111 g/l) with normocytic normochromic indexes, leucocytes 8100/mm³ ($8.1 \times 10^9/l$), and thrombocytes 515 000/mm³

($515 \times 10^9/l$). Urinalysis was normal, as were the serum electrolytes, glucose, urea, creatinine levels, and liver function tests. The serum albumin concentration was 32 g/l. Muscle enzymes, including creatine phosphokinase, aldolase, transaminase, and lactic dehydrogenase, were all within normal levels. Many cultures obtained from the throat, sputum, urine, and blood were negative. The serological tests for hepatitis B antigen, Epstein-Barr virus, cytomegalovirus, and cold agglutinins, the Weil-Felix reaction, Widal test, and test for antinuclear antibodies were also negative. The antistreptolysin O titre was 1/400 on admission and 1/800 two weeks later. The C-reactive protein was 5+. An electrocardiogram showed T wave inversion in leads V₁-V₃ which reverted to normal during the hospitalisation. A chest radiogram was normal. The patient was treated with aspirin 2.7 g daily and responded dramatically. By the following morning the muscle pain had resolved, and a scheduled electromyography was cancelled. She remained symptomless thereafter on maintenance aspirin therapy.

CASE 2

A 24-year-old man was admitted to our department because of severe myalgia. Ten days previously he had a sore throat associated with fever and was treated with ampicillin and aspirin for three days. A week later he developed severe proximal and distal muscle pain and weakness. On admission he appeared ill and his temperature was 38.1°C. The throat, lungs, abdomen, and skin were normal. Heart sounds were normal, and no murmurs were heard. The proximal and distal muscles were so tender that he was unable to move because of the incapacitating pain. There was marked weakness of the proximal and distal muscles of the upper

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extremities and moderate weakness of the proximal and distal muscles of the lower limbs.

Laboratory examinations showed: ESR 85 mm after one hour, haemoglobin 16.4 g/dl (164 g/l), white blood cell count $15\,600/\text{mm}^3$ ($15.6 \times 10^9/\text{l}$) with 70% polymorphonuclears, and thrombocytes $336\,000/\text{mm}^3$ ($336 \times 10^9/\text{l}$). Urinalysis was normal. Serum electrolytes, glucose, liver and kidney function tests were normal. Repeated determinations of muscle enzymes (creatine phosphokinase, lactic dehydrogenase, oxaloacetic transaminase, and aldolase) were all normal. Serological tests for hepatitis B antigen, Epstein-Barr virus, cytomegalovirus, cold agglutinins, antinuclear antibodies, and rheumatoid factor were negative. The antistreptolysin O titre increased over three weeks from 1/160 to 1/333, and C-reactive protein was 4+. Serum protein electrophoresis showed increased α_2 globulin (19.5% of total protein) and a diffuse hypergammaglobulinaemia. An electrocardiogram and chest roentgenogram were normal. Electromyography of the biceps, deltoid, flexor digitalis, quadriceps, and gastrocnemius showed mild myopathic changes without spontaneous activity. In addition, a mild reduction of recruitment and excess of polyphasic activity were noted. A muscle biopsy specimen obtained from a tender biceps muscle was normal. Aspirin in a dose of 4.8 g/day resulted in a prompt relief of pain, and the tenderness disappeared within a week.

CASE 3

Two weeks after an untreated upper respiratory tract infection a 21-year-old woman complained of acute pain and marked weakness in both legs and arms and swelling of the left calf. On admission she appeared to be acutely ill and her temperature was 39°C . Rales were heard over both lung bases, but the heart and abdomen were normal. The muscles of all four limbs were very tender. The strength of the proximal muscles of the upper and lower limbs was markedly decreased and that of the distal muscles moderately decreased. There were no signs of arthritis.

Laboratory data included: ESR 90 mm after one hour, white blood cell count $19\,000/\text{mm}^3$ ($19 \times 10^9/\text{l}$), with a marked shift to the left. Haemoglobin was 8.5 g/dl (85 g/l) with mild hypochromia (mean corpuscular haemoglobin 24 pg) and thrombocytes $615\,000/\text{mm}^3$ ($615 \times 10^9/\text{l}$). Urinalysis showed +1 proteinuria with few red and white blood cells per high power fields but with no casts. Serum electrolytes, glucose, and liver function tests were normal. The blood urea nitrogen was 28 mg/dl (10 mmol/l). Muscle enzymes, including transaminase, creatine phosphokinase, lactic dehydrogenase, and aldolase, were normal.

Serum albumin was 24 g/l and globulin 43 g/l; serum protein electrophoresis showed an increase of α_2 globulin (24%). Antinuclear antibodies and rheumatoid factor were not detected, and serological tests for Epstein-Barr virus, cytomegalovirus, and hepatitis B antigen were negative. An antistreptolysin O titre was 1/833 and increased to 1/1600 a month later. C-reactive protein was 3+. Numerous cultures obtained from blood, urine, throat, sputum, and cerebrospinal fluid were negative. A chest radiogram showed increased interstitial markings, and a small pericardial effusion was noted on echocardiography. Electrocardiography and electromyography were normal.

The patient was treated with prednisone 40 mg daily. Although the fever declined and the myalgia improved gradually, a residual mild proximal myopathy persisted. She was discharged after a month on a tapering prednisone regimen. Three weeks after discharge a complete resolution of her myopathy was observed.

Discussion

The present report describes three patients who showed a similar clinical picture of severe myalgia and fever after streptococcal upper respiratory tract infection. The myalgia was characterised by severe incapacitating muscle pain and tenderness affecting the proximal and distal muscles of both upper and lower limbs, without increase of muscle enzymes. There was serological evidence of recent streptococcal infection in all cases and non-specific signs of inflammation, including a high erythrocyte sedimentation rate, positive C-reactive protein, and an increase of α_2 globulin. Complete and rapid resolution was observed on treatment with salicylates in two patients and prednisone in one case.

The clinical picture and the positive serological evidence for recent streptococcal infection suggest a pathogenesis similar to rheumatic fever. Drug allergy is unlikely, since only one patient (case 2) was treated with ampicillin before admission, and the drug was discontinued five days before onset of the disease. The clinical manifestations of rheumatic fever have undergone considerable modification in recent years, and the application of the Jones criteria in the adult population is often difficult.¹⁻⁵⁻⁷ In classical rheumatic fever the presence of arthritis or carditis is necessary in order to make the diagnosis. About 12% of patients with suspected rheumatic fever are described as 'minor streptococcal disease'² which usually includes arthralgia, high sedimentation rate, fever, and sometimes a prolonged P-R interval in the electrocardiogram. None of the three patients described herein fulfilled the

Jones criteria for acute rheumatic fever, and their clinical picture is closer to that described as 'minor streptococcal disease'.

A careful review of the relevant literature showed that despite the large number of target organs affected in patients with rheumatic fever, poststreptococcal glomerulonephritis, Schönlein-Henoch purpura, and erythema nodosum, the skeletal muscles seem to be spared.¹⁻³ It is possible that myalgia is frequently overlooked in patients with rheumatic fever because of the accompanying arthritis; myalgia after a recent streptococcal infection should be considered as an additional complication of immunologically mediated poststreptococcal response.

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