

Trauma and seronegative spondyloarthritis: report of two more cases of peripheral arthritis precipitated by physical injury

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SUMMARY Two more cases of B27 associated peripheral arthritis triggered by physical injury are reported. One patient developed arthritis after a minor insult and in the other Reiter's syndrome occurred after the injury. Possibly, trauma causes release of self antigens from the injured joints.

In a recent issue of the *Annals* our group reported the cases of two HLA-B27 positive patients who developed peripheral arthritis immediately after a significant traumatic episode.¹ Unlike previously reported cases of HLA-B27 associated peripheral arthritis triggered by trauma²⁻⁴ they showed a rapid, severe destructive process. In the past few months both patients have had total hip replacement.

We have recently observed two more cases of B27 associated peripheral arthritis triggered by physical injury which are reported here.

Case reports

PATIENT NO 1

A 41 year old man was run over by a car and taken to hospital on 22 May 1987. He had multiple contusions and his right scaphoid was fractured. Four days later a surgical operation was performed on his right hand. On the fifth day he had diarrhoea and fever, and in the following days urethritis with mucoid discharge and arthritis with effusion of both knees.

Arthrocentesis yielded a yellow, turbid fluid with reduced viscosity. Gram stain showed no organism, and culture was negative. Urethral and stool cultures were also negative. Blood examination showed the following significant values: erythrocyte sedimentation rate (ESR) (Westergren) 97 mm/1st h, seromuroids 72 mg/l (normal <50), raised C reactive protein (CRP), α_2 globulin 14.9% (72 g/l total

protein), and white cell count $14.9 \times 10^9/l$ with 89% polymorphonuclear leucocytes.

The urethritis and diarrhoea subsided in a few days without any treatment, but arthritis persisted. The patient was given diclofenac 150 mg/day.

On 9 July 1988 the patient was referred to us because of the persistence of arthritis. His past history disclosed numerous episodes of inflammatory low back pain and stiffness and alternate sciatic pain without radiation below the knee since 1970, together with three other traumatic episodes with fracture of the bones of the hand or foot in the last 13 years. He denied having extramarital sexual intercourse or having ever had urethritis, diarrhoea, conjunctivitis, uveitis, psoriasis, peripheral arthritis, or peripheral enthesopathy in the past.

His family history disclosed that his mother had presented numerous episodes of unilateral acute anterior non-granulomatous uveitis and had been suffering from inflammatory low back pain and stiffness.

Physical examination showed slight swelling without any obvious effusion, together with warmth, and tenderness of both knees. Lumbar and cervical spine movement and chest expansion were normal.

Investigations showed ESR (Westergren) 107 mm/1st h, CRP 37 mg/l (normal <50), α_1 acid glycoprotein 2.39 g/l (normal 0.55-1.40), haptoglobin 11.4 g/l (normal 0.4-1.8), α_2 globulin 11% (72 g/l total protein), IgA 4.45 g/l (normal 0.5-3.25), IgG 19 g/l (normal 8-15), and a white cell count of $10 \times 10^9/l$ with 79% polymorphonuclear leucocytes. Tests for antinuclear antibodies and the rheumatoid factor were negative.

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Cervical, dorsal, and lumbar spine radiographs and sacroiliac joint plain films were normal.

The arthritis subsided two months after it had begun. In September 1987 the acute phase reactants were normal. In the past 15 months the patient has had other episodes of inflammatory low back pain and stiffness.

PATIENT NO 2

A 25 year old man effected a parachute jump on 8 May 1987 and landed with his knees extended instead of flexed, as is usual. Four hours later his knee pain became more acute, and on the following day he developed arthritis with effusion of both knees. Arthrocentesis yielded a yellow, turbid fluid with reduced viscosity. Gram stain and culture were negative. Blood examination gave the following significant values: ESR (Westergren) 30 mm/1st h and CRP 25 mg/l. Tests for the rheumatoid factor and antinuclear antibodies were negative. The patient was treated with local injections of steroids and referred to us.

His past history was negative for inflammatory low back pain and stiffness, peripheral arthritis, peripheral enthesopathy, urethritis, diarrhoea, uveitis, conjunctivitis, or psoriasis. The family history was negative for seronegative spondyloarthropathy and the other HLA-B27 associated syndromes.

Physical examination showed moderate swelling without obvious effusion and tenderness of both knees. Spinal movement and chest expansion were normal.

The only aspects of laboratory evaluation worthy of note were an ESR of 35 mm/1st h and a CRP of 28 mg/l. HLA was positive for the B27 antigen.

Sacroiliac and lumbar spine x rays were normal.

Effusion of both knees again became evident in June 1987 and was again treated with local injections of steroids. In July 1987 the ESR and CRP returned to normal. From that time the patient has been well and the acute phase reactants have remained negative.

Discussion

There is not much doubt about the close relation in both patients between physical injury and the subsequent appearance of arthritis. Both cases present certain prominent characteristics. The second patient, unlike previous cases of B27 associated peripheral arthritis precipitated by trauma,¹⁻⁴ developed arthritis after a minor insult. He did not

suffer any bruise or fracture but only joint pain after landing with his knees extended. The first patient, like some other cases,^{2,3,5-7} developed Reiter's syndrome after the injury; he had sterile urethritis and diarrhoea in addition to arthritis. He firmly denied having extramarital sexual intercourse.

Seronegative spondyloarthritides include a heterogeneous group of disorders occurring in genetically predisposed subjects after exposure to environmental triggers.⁸ The causative role of some microorganisms is universally accepted in Reiter's syndrome and reactive arthritis⁹ and is under consideration in ankylosing spondylitis.^{10,11} The triggering role of physical injury in precipitating a peripheral arthritis has been suggested by several reports published in the past few years.^{1-8,12} The pathogenic mechanisms involved in trauma derived HLA-B27 associated peripheral arthritis remain to be studied. Possibly, trauma causes release of self antigens from the injured joints.²

References

- Olivieri I, Gherardi S, Bini C, Trippi D, Ciompi M L, Pasero G. Trauma and seronegative spondyloarthropathy: rapid joint destruction in peripheral arthritis triggered by physical injury. *Ann Rheum Dis* 1988; **47**: 73-6.
- Wisniewsky J J. Trauma and Reiter syndrome: development of 'reactive arthropathy' in two patients following musculoskeletal injury. *Ann Rheum Dis* 1984; **43**: 829-32.
- Masson G, Thomas P, Bontoux D, Alcalay M. Influence of trauma on initiation of Reiter's syndrome and ankylosing spondylitis. *Ann Rheum Dis* 1985; **44**: 860-1.
- Jacobs J C, Berdon W E, Johnston A D. HLA-B27-associated spondyloarthritides and enthesopathy in childhood: clinical, pathologic, and radiographic observations in 58 patients. *J Pediatr* 1982; **100**: 521-8.
- Doury P, Pattin S, Ille H, Larroque P, Allard Ph. Syndrome de Fiessinger-Leroy-Reiter avec traumatisme apparemment inaugural. *Rev Rhum Mal Osteoartic* 1974; **41**: 55-7.
- Bernard J G, Badrouillard R, Lucaes R, Feline A. Le syndrome de Fiessinger-Leroy-Reiter. 1. Etude clinique. 2. Etude épidémiologique et considérations étiologiques (à propos de 310 obs). *Semaine des Hôpitaux de Paris* 1964; **48**: 2623-9.
- Noer H R. An 'experimental' epidemic of Reiter's syndrome. *JAMA* 1966; **197**: 693-8.
- Moll J M H, Haslock I, Wright V. Seronegative spondyloarthritides. In: Scott J T, ed. *Copeman's textbook of the rheumatic diseases*. Edinburgh, London, Melbourne, New York: Churchill Livingstone, 1986: 723-44.
- Keat A. Reiter's syndrome and reactive arthritis in perspective. *N Engl J Med* 1983; **309**: 1606-15.
- Ebringer A. The cross-tolerance hypothesis, HLA-B27 and ankylosing spondylitis. *Br J Rheumatol* 1983; **22** (suppl 2): 53-66.
- Geczy A F, Alexander K, Bashir H V, Edmonds J P, Upfold L, Sullivan J. HLA-B27, klebsiella and ankylosing spondylitis: biological and chemical studies. *Immunol Rev* 1983; **70**: 23-50.
- Williams K A, Scott J T. Influence of trauma on the development of chronic inflammatory polyarthritis. *Ann Rheum Dis* 1987; **26**: 532-7.