Immunisation triggering rheumatoid arthritis?

Sir, We read with interest the article by Turner-Stokes and Isenberg on immunisation of patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE).1 Isolated reports of the onset of SLE following immunisation have occurred,2 but we are unaware of similar cases in RA. We have recently observed a patient who developed RA three weeks after a second dose of tetanus toxoid. A 34 year old woman received two doses of tetanus toxoid in November and December 1986. A week after the second dose she developed severe pain with erythema and induration measuring 10 cm in diameter at the site of injection over the left deltoid muscle. This severe local reaction lasted 10 days. A few days after the erythema and induration started to fade the patient developed a symmetrical inflammatory polyarthritis involving the proximal interphalangeal, metacarpophalangeal, wrist, elbow, shoulder, knee, and the metatarsophalangeal joints. The arthritis was severe with early morning stiffness lasting several hours. There was no past history of arthritis, inflammatory bowel disease, iritis, back pain, psoriasis, or recent infection and no family history of inflammatory arthritis.

Investigations in March 1987 showed haemoglobin 149 g/l, white cell count 8 x 10^9/l, platelet count 307 x 10^9/l, erythrocyte sedimentation rate (ESR) 17 mm/h, C reactive protein 120 mg/l (normal range (NR) <60), albumin 45 g/l, globulin 33 g/l (NR 18–32), antinuclear antibodies negative, and IgM rheumatoid factor positive (titre 1/32). An x ray examination of her hands and feet showed periarticular osteoporosis.

She was treated with bed rest, splintage of the inflamed joints, and non-steroidal anti-inflammatory drugs. The response was incomplete and in December 1987 treatment was started with sulphasalazine. Her arthritis responded and by March 1988 her early morning stiffness was minimal and her joints were quiescent. Her haemoglobin was 151 g/l, ESR 7 mm/h, C reactive protein 60 mg/l, and serum albumin and globulin were 49 and 27 g/l respectively. Her IgM rheumatoid factor remained positive (titre 1/64).

Our patient satisfied the American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis.3 She had a severe local reaction after the second dose of the toxoid; a not uncommon side effect, the incidence of which increases with the age of the subject and is more common in women. Those who develop severe reactions frequently have high titres of the circulating antitoxin.4 It could well be that the B cell proliferation that followed the vaccination might have precipitated the onset of RA in our patient, who might have been genetically predisposed. Although one cannot draw too many conclusions from one isolated case, there was a strong temporal relation between the introduction of the vaccine, the development of the severe local reaction, and the onset of RA.

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References


Lymphoma chemotherapy as remission inducing treatment in rheumatoid arthritis

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