A case of multiple sclerosis associated with rheumatoid arthritis and positive anticardiolipin antibodies

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We describe the case of a 59 year old man with longstanding multiple sclerosis (MS) since his early fifties, who after many years developed the clinical and serological manifestations of erosive rheumatoid arthritis (RA). This rare association is interesting owing to the overlapping pathophysiological similarities of T cell and tumour necrosis factor α (TNFα) in both diseases.[1 2] The MS musculoskeletal complaints masked proper assessment of his RA, resulting in a delayed diagnosis. As far as we know, this is the first case in which MS, RA, and serum anticardiolipin antibodies (aCL) have been found to be present simultaneously.

CASE REPORT

A 48 year old man presented to the orthopaedic clinic with left knee and foot pain and an associated mild weakness on the left side. Examination confirmed patellofemoral crepitus, and spastic paraparesis. A diagnosis of MS was made, supported by oligoclonal bands in cerebral spinal fluid, delayed visual evoked responses, and magnetic resonance imaging studies which showed high intensity areas in the brainstem tegmentum and periventricular white matter. His main deficit was spasticity of the legs and intermittent pains in hands and feet. He was mobile with crutches. Baclofen or dantrolene in therapeutic doses did not improve his spasticity.

Eight years later, aged 56, he developed a flitting symmetrical polyarthritis that was relieved by ibuprofen. There were no extra-articular manifestations of RA; he had two hours of early morning stiffness. There was no active synovitis on musculoskeletal examination. Acute phase proteins, full blood count, biochemistry, rheumatoid factor, antinuclear antibody, and extranuclear antibodies were all normal. IgG aCL was 30.3 GPLU, and it remained raised on repeated occasions. He continued to have episodic joint swelling and pain in both feet and hands, without synovitis or serological abnormalities at each review for four years. Feet and hands *x* ray examinations carried out at age 57 were normal. At age 59, he presented with swollen metacarpophalangeal (MCP) joints and painful feet, with almost total loss of mobility and independence. He had evident MCP, right wrist and metatarsophalangeal (MTP) synovitis. Feet and hand *x* ray examinations showed well marked periarticular osteoporosis and erosive arthropathy. Investigations showed an erythrocyte sedimentation rate of 50 mm/1st h, C reactive protein 44 mg/l, and rheumatoid factor negative; biochemistry and full blood count were normal, and IgG aCL was 35.6 GPLU. Treatment was started with methotrexate, folic acid, and low dose aspirin, with great improvement in his joint swelling, mobility, and acute phase response.

DISCUSSION

TNFα and T cells drive the inflammatory cytokine cascade that activates metalloproteinases and other degradative enzymes, thus leading to erosive joint destruction in RA and demyelisation in MS.[3] A bibliographic search to date has confirmed that no reports exist which suggest either an increased incidence or prevalence of RA in patients with MS. This case suggests that regardless of the absence of synovitis and an initial raised acute phase response, a history suggestive of inflammatory joint disease should lead to further evaluations and close follow up to avoid delayed diagnosis of RA and treatment. The concomitant occurrence of reduced activities of daily living and the predominance of longstanding foot pain masked proper evaluation of MTP synovitis.

The opposite is also true, that MS diagnosis can be delayed in patients with active RA, further highlighting the possibility that most of the reported cases of demyelinating process in patients with RA treated with TNFα antagonist represent exacerbation of a pre-existing state of early MS.[4 5]

The relevance of persistently raised aCL in our patient is of clinical interest as he developed RA later in life. It is well recognised that aCL in patients with MS indicate an underlying autoimmune disease or an epiphenomenon of a more diffuse immunological process.[6] It is also apparent that aCL have no influence on MS progression. Roussel *et al* found no correlation between aCL and age, sex, duration of MS from diagnosis, category of MS, clinical course, clinical symptoms, serum levels, or atypical lesions by magnetic resonance imaging.[7] Hence, aCL as in this case, did not influence or change the clinical form of MS.

In conclusion, our report highlights the interesting association between MS and RA. It emphasises the need to consider this potential rare association to avoid delayed diagnosis of RA in patients with MS.

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