cerebrovascular insult (1 pt) and idiopathic intracranial hypertension (1 pt). Only two patients of 50 with primary Sjogren syndrome expressed CNS manifestations as cranial nerve paralysis or disseminated demyelination disease. We also analysed our 20 patients with primary antiphospholipid syndrome, that is not connective tissue disease, but its immunological-mediated pathogenesis may affect functions of CNS. Among them two patients had multiinfarct dementia.

Conclusion We concluded that CNS involvement is not frequent manifestation in our patients with connective tissue diseases.

FRI0092 TWO CASES OF ETANERCEPT-INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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10.1136/annrheumdis-2001.127

Background Anti-TNF alpha therapies in human rheumatoid arthritis, appear to be very safe and efficient. Most frequently reported adverse events are infectious complications and local manifestations at site of injection. Auto-antibodies have been reported in some cases.

Objectives We report 2 cases of etanercept-induced SLE in patients with seronegative RA and previous isolated positive ANA.

Methods

Results A 38-year-old Caucasian woman was started on etanercept on February 2000, after a 16-year history of severe seropositive RA unsuccessfully treated with hydroxychloroquine, D-Penicillamine, gold salts, and methotrexate plus prednisone. When aged 32, positive ANA (1/400°) and transient low C4 were noticed with weakly positive Anti-DNA (ELISA, 40 IU/ml, positivity > 40); she had no anti-ENA, anti-keratin, APF or anti-cardiolipin antibodies. RA remained very active [morning stiffness > 3 h, CRP 60, 28 swollen joints]. MTX was stopped and the patient started on etanercept. RA was remarkably controlled within one month, and prednisone tapered. 3 months later, she presented scattered lesions located on the face and the scalp (no sun exposure), fatigue and diffuse pain. Skin analysis demonstrated dermal atrophy with keratinocytes necrosis, perifollicular and perivascular lymphocytic infiltration, with no vasculitis. She had positive ANA, anti-DNA, anticardiolipin Ab and low C4 with negative anti ENA. She also had elevated muscle enzymes, normal blood count, normal liver and renal functions (no proteinuria). Etanercept was stopped; she was started on topical steroids. One month later skin lesions had cleared, very active polyarthritis was again present. At that time ANA remained positive, anti-DNA decreased as well as muscle enzymes and ACL antibodies. Biological abnormalities had returned to normal values in September 2000. The patient has not been rechallenged.

A 50-year-old woman with a familial history of RA, had active arthritis despite steroids and sulphasalazine. Methotrexate and ciclosporin were excluded (chronic alcoholic hepatitis and HTA). Etanercept was introduced in October 1999. RA was remarkably controlled, but she developed after 4 months a diffuse erythematous and purpuric skin eruption with fine scaling (face, dorsa of hands and fingers), erosions and crusts, not involving the injection sites. Histological analysis showed hyperkeratosis, keratinocyte necrosis and perivascular lymphocytic infiltration without vasculitis. She had lymphopenia,

thrombocytopenia, elevated ESR, abnormal liver function test, and normal renal function, positive ANA (1/640), negative anti-double-stranded-DNA and anti-ENA Ab. After etanercept stoppage, skin manifestations spontaneously resolved but synovitis reappeared. The patient has not been rechallenged.

Conclusion Imputability of etanercept the disease is very likely, given the temporal correlation between the onset of signs with the initiation of injections, and their resolution following withdrawal of the drug. Further evaluation is needed to know whether presence of positive ANA or association with other SLE drug inducer are risk factors for development of these TNF inhibitors-induced manifestations.

FRI0093 TWO CASES OF ETANERCEPT-INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background Anti-TNF alpha therapies in RA appear to be very safe and efficient. Most frequently reported adverse events are infections and local manifestations. Auto-antibodies have been reported in some cases.

Objectives We report 2 cases of etanercept-induced SLE in patients with RA and previous isolated ANA.

Methods

Results A 38-year-old Caucasian woman was started on etanercept on February 2000, after a 16-year history of severe seropositive RA unsuccessfully treated with hydroxychloroquine, D-Penicillamine, gold salts, and methotrexate plus prednisone. When aged 32, positive ANA (1/400°) and transient low C4 were noticed with weakly positive Anti-DNA; she had no anti-ENA, anti-keratin, antiperinuclear factor or anti-cardiolipin antibodies. RA was very active [morning stiffness > 3 h, CRP 60, 28 swollen joints]. MTX was stopped and the patient started on etanercept. RA was remarkably controlled, and prednisone tapered. 3 months later, she presented scattered lesions on the face and the scalp (no sun exposure), fatigue and diffuse pain. Skin analysis demonstrated dermal atrophy with keratinocytes necrosis, perifollicular and perivascular lymphocytic infiltration, with no vasculitis. Biological tests revealed positive ANA, anti-DNA, anticardiolipin antibodies, low C4, negative anti ENA. She also had elevated muscle enzymes, normal blood count, liver and renal functions and no proteinuria. Etanercept was stopped; she was started on topical steroids. One month later skin lesions had cleared, very active RA was again present. At that time ANA remained positive, anti-DNA decreased as well as muscle enzymes and ACL antibodies. Biological abnormalities had returned to normal values in September 2000. The patient has not been rechallenged.

A 50-year-old woman had active RA despite low dose steroids and sulphasalazine. MTX and ciclosporin were excluded because of alcoholic hepatitis and hypertension. Etanercept was introduced in October 1999. RA was remarkably controlled; she developed 4 months later a diffuse erythematous and purpuric skin eruption with fine scaling (face, dorsa of hands and fingers) erosions and crusts, not involving the injection sites. Histological analysis demonstrated hyperkeratosis, keratinocyte necrosis and perivascular lymphocytic infiltration without vasculitis. Biological tests showed lymphopenia, thrombocytopenia, elevated ESR, abnormal liver function test, and normal renal function tests,