than in those without (85.3% vs. 54.8%; $p = 0.007$). No differences were observed according to the shared epitope and the frequency of HLA-DR4. The different TNF- α polymorphisms were equally distributed in AKA positive and negative populations. No significative radiological differences (presence of erosive disease and Larsen score) were observed at study entry between both populations. A trend to a more progressive radiological damage was observed at the end of follow-up (32 ± 6 mo) in those patients with AKA, although the differences were no significative (Larsen score of 6.1 ± 4.3 in AKA(+) vs. 4.5 ± 5.5 in AKA (-). This trend is also observed when only RF positive patients were evaluated.

Conclusion These results indicate that AKA cannot identify a distinct immunogenetic subgroup in Spanish reumatoid population. The role of AKA as a possible marker of a more aggressive joint disease merits further investigation.

THU0162 BONE INVOLVEMENT IN GAUCHER DISEASE

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Background

Objectives

Methods Gaucher disease (GD) is an autosomal disorder of glycosphingolipid metabolism due to deficiency of the lysosomal acid glucocerebrosidase. Although prevalent among Ashkenazi Jews, it has been described in all other ethnic groups. Bone lesions are associated with type 1 and type 3 GD. They are secondary to the progressive accumulation of histiocytes and macrophages laden with glucosylceramide in bone marrow. Expression of skeletal involvement in Gaucher patients ranges from asymptomatic disease, with or without radiologic signs, to symptomatic disease, which can be severe and engender considerable pain and disability. MRI appears to be the most valuable imaging technique for evaluating bone marrow involvement. Splenectomy is suggested to accelerate skeletal deterioration. Our patient was a 23-year old man type 1 GD who was admitted for evaluation of hepatosplenomegaly and thrombocytopenia. He also complained of episodic joint involving both knees, shoulders, wrists and elbows lasting 2 – 5 days that started a year before admission. The pain was defined as non specific pain. Clinical examination yielded local tenderness. Hepatosplenomegaly was confirmed by abdominal CT scan. Biology revealed an hypersplenism (anaemia, thrombocytopenia) and polyclonal hypergammaglobulinemia. X ray of femurs and humerus showed a metaphysal multicystic osseous changes with endosteal cortical erosions. X ray of pelvis showed an osteosclerosis of iliac bones, lytic process of pubis symphysis and pathological fracture of both ischiopubic bones. MRI showed medullar bone infiltration in femurs, pelvis and humerus. ^{99m}Tc bone scan was normal. On liver as well as on bone marrow biopsy examination, Gaucher's cell infiltrates were detected. The level of leukocyte acid beta-glucosidase activity was very low. Hypersplenism was the indication for splenectomy. The weight of the resected spleen was 1950 gr; Following surgery, the patient noted an exacerbation of major joint pain and bone pain involving both femurs and humerus which improve after one week of non steroidal anti-inflammatory drugs. Hypersplenism (thrombocytopenia in particular) was improved. Hepatomegaly without evidence of

hepatic dysfunction persisted. Enzyme replacement therapy wasn't available.

Results

Conclusion

THU0163 CORTISOL AND PROLACTIN LEVELS IN RHEUMATOID ARTHRITIS AND SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Accumulated evidence suggests the existence of bidirectional communication between the neuroendocrine and immune systems. The pituitary gland can regulate the immune response secreting both immunostimulatory [e.g. prolactin (PRL)] and immunosuppressive (cortisol via ACTH) hormones. A disorder of the immunoregulatory circuit between these systems could potentially result in the development of immune mediated diseases. Hiperprolactinemia has been implicated in the pathogenesis of systemic lupus erythematosus (SLE), Reiter's Syndrome and psoriatic arthritis. Elevated PRL levels have been described in patients with rheumatoid arthritis (RA). However, clinical studies regarding the PRL level and disease activity have yielded contradictory results.

Objectives In this study our aim was to assess prolactin and cortisol plasma levels in patients with RA and SLE and to evaluate their correlation with disease activity.

Methods PRL and cortisol plasma levels were measured in 40 female patients aged (24 RA patients and 16 SLE patients) 38.88 ± 14.44 and 40 age-matched healthy controls. Pregnant patients, patients taking drugs that could increase PRL or cortisol levels and those with renal and/or hepatic failure, were excluded from the study. All subjects were premenopausal women with regular menstrual cycles. Plasma concentrations of prolactin and cortisol were determined by radioimmunoassay under standardised conditions in venous blood drawn through an indwelling cannula. Disease activity was assessed using the Disease Activity Score (DAS) for the RA patients SLEDAI for SLE patients.

Results The mean PRL plasma level was higher in RA and SLE than in the control group (13.8 ± 6.1 ; 15.6 ± 4.2 vs. 8.78 ± 2.43 , respectively, $p < 0.05$). Cortisol plasma levels were found to be higher in the RA and SLE patients than in controls, but there were not significantly different (14.2 ± 3.6 ; 16.3 ± 3.3 vs. 13.1 ± 2.8 respectively, $p > 0.05$).

Conclusion No difference was found in prolactin and cortisol levels between RA and SLE patients.

A correlation between prolactin and cortisol levels was found only in SLE patients ($p < 0.05$).

No correlation was found between PRL or cortisol levels and disease activity in RA and SLE subjects.

THU0164 IS SOLUBLE CD44 ISOFORM VARIANT 5 USEFUL AS PREDICTING FACTOR AND AS PARAMETER FOR LONG-TERM OBSERVATION IN RHEUMATOID ARTHRITIS?

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