Conclusion Our findings are consistent with hypothesis that B cell response in RA synovium is probably antigen driven and oligoclonal.

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SLE – Clinical aspects

AB0071 LYMPHOCYTOTOXIC ANTIBODY (LCA) AMONG IRAQI SLE PATIENTS AND THEIR RELATIVES

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Background

Objectives To study the Lymphocytotoxic antibodies (LCA) in SLE patients and their first degree relatives compared to matching controls.

Methods Complement dependent microcytotoxicity test was used to assess the presence of LCA, their temperature dependence in addition to HLA specificity among 40 SLE patients, 184 of their first degree relatives in comparison to 100 subjects of a blood bank donor as a healthy control.

Results Lymphocytotoxic activity was manifested in 70% of patients, 33.36% of relatives compared to 80% of control group. While cold dependent antibodies were higher among patients group (92.8%) compared to 35% of their relatives. The presence of these antibodies in a control group was less temperature dependent, since 62.5% of them reacted both at 22°C and 15°C and only 12.5% of them exhibited a sole reactivity of 15°C.

No association between LCA and and of HLA antigen of both classes was detected, however, patients had a tendency to react with a large number of lymphocyte samples than did positive sera from other group. Generally, females had a higher percentage of LCA (42.15%). Yet, the highest percentage was reported among sisters, followed by mothers, while brothers exhibited the least frequency (15.15%). In contrast to females, male subjects reported the highest frequency of these antibodies among age group of > 50 years.

Conclusion Patients expressed a high positivity rate of cold LCA. Moreover, female first degree relatives showed higher positivity rates than male relatives or controls.

AB0072 LYMPHOCYTOTOXIC ANTIBODY (LCA) AMONG IRAQI SLE PATIENTS AND THEIR RELATIVES

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Objectives To study the lymphocytotoxic antibodies (LCA) in SLE patients and their first degree relatives compared to matching controls.

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AB0073 A CASE OF FULMINANT LUPUS IN A YOUNG MAN WITH HEREDITARY HAEMOCHROMATOSIS

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Background Mild derangements of liver function tests in systemic lupus erythematosus (SLE) are not uncommon, reflecting either disease activity or treatment. Rare causes of hepatic dysfunction include arteritis and lupoid hepatitis.¹ We report on a patient with SLE and coexisting haemochromatosis.

Objectives

Methods CASE REPORT: A 26 year old man presented with acute onset of jaundice and skin rash. He had a history of intermittent rash and arthralgia with serology positive for ribonuclear protein antibodies characteristic of mixed connective tissue disease. On examination he was icteric with a pyrexia. There was a maculopapular rash typical of subacute cutaneous lupus over his anterior chest wall.

Results Investigations showed a normocytic, normochromic anaemia, a thrombocytopaenia and a lymphopaenia. His prothrombin time and partial thromboplastin time were prolonged. Liver function tests showed an elevated total bilirubin and raised aspartate transferase and alanine transferase. Autoimmune serology showed positive antiRNP/Sm. Complement levels were low. Anticardiolipin antibodies were negative. Viral titres were negative. Serum ferritin level was elevated. Blood cultures grew *Staphylococcus aureus*. He was treated with high dose corticosteroids. He had intravenous antibiotics. The coagulopathy was treated with vitamin K and fresh frozen plasma. The thrombocytopaenia was treated with immunoglobulins. In spite of further management, unfortunately he died. A postmortem showed acute renal tubular necrosis and crescentic glomerulonephritis. Liver histology showed periportal deposition of iron predominantly in zone three. This, together with the presence of iron deposition in the exocrine pancreas gland confirmed the diagnosis of hereditary haemochromatosis.