

Antinuclear Antibodies in Mycobacterium Tuberculosis Infection

Sir,

A 10-year-old girl presented with fever, evanescent rash involving the proximal portion of the arms and legs and arthralgia of both small and large joints for 2 months. There was no history of cough, breathlessness, palpitation, chest pain, abdominal distension, loss of weight or appetite, skin & mucus membrane bleeds. On examination, she was afebrile and weighed 28 Kg (88% of expected) with height of 136 cm. She had pallor, cervical lymphadenopathy that was neither matted nor tender and maculopapular rash on the proximal portion of the arms and legs. BCG scar was seen. Systemic examination including eye examination was normal.

Her WBC count was 8700-cells/mm³ with normal differential count; Hb was 8.3 g/dl and peripheral smear showed microcytic hypochromic red cells with no blasts and normal platelets. ESR at 1 hr was 110 mm. Mantoux done with 1 TU of PPD was positive (12 mm). Chest X-ray revealed calcified mediastinal and perihilar nodes. Blood and urine cultures were sterile. Sputum for tubercle bacilli was negative and ultrasonogram abdomen was normal. HIV II and I were negative. In view of prolonged fever with rash and joint pains ANA and anti Ds DNA were done and were strongly positive 1: 40 and 1: 10 dilution respectively done by indirect fluorescent antibody technique. Anti antibodies against U1 Ribonucleoprotein (anti U 1 RNP), anti neutrophil cytoplasmic antibody (ANCA) and anti smooth muscle antibody (ASMA) were negative. Rheumatoid factor was negative. Serum complement and ferritin levels were normal. Bone marrow examination revealed erythroid hyperplasia. Excision lymph node biopsy from the cervical lymph node revealed areas of central necrosis surrounded by multinucleated histiocytes and langhans giant cells consistent with tuberculosis and smear was positive for tubercle bacilli. She was started on treatment with antitubercular drugs: isoniazid(H), rifampicin [R] and pyrazinamide(Z) for 2 months followed with HR for four more months (2HRZ + 4 HR) as per category III of revised national tuberculosis program of India (RNTCP) following which she improved gradually.

Mycobacterial infections are known to induce the development of autoantibodies¹ and a few of these are also diagnostic markers for other diseases. The presence of auto-antibodies like antinuclear antibody (ANA) and ANCA in patients with tuberculosis is an interesting

finding which makes us wonder whether mycobacterial infection acts as a trigger or it is due to the drug regimen.² Mycobacterium tuberculosis stimulates the release of oxygen metabolites from neutrophils that are activated by interaction with phenol glycolipids of the cell wall of M. tuberculosis, leading to the release of lysosomal enzymes from the neutrophils and thereby leading to the development of autoantibodies against the granular component of those cells.³ Vandana *et al*² had reported ANCA and ANA positivity in 34% and 24.3% of patients with tuberculosis respectively. Win *et al*⁴ had reported ANA positivity from pleural fluid in a girl with tuberculous pleural effusion and Elkavam *et al*⁵ had reported a higher proportion of children with tuberculosis with anti ds DNA positivity (32%). This 10-year-old girl with tuberculosis had positive antinuclear antibody and anti ds DNA and negative ANCA, though she did not have other features suggestive of connective tissue disorders. She responded well to antituberculous therapy that was initiated and on follow up after 2 months her ESR level had returned to normal (1 hour ESR-22 mm) though her serum ANA and anti Ds DNA remained positive. This case is reported here to sensitize the presence of autoantibodies in patients with tuberculosis and the need for follow up.

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