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Case Report

Leprosy : A Great Mimicking Disease

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Leprosy may mask a variety of diseases. One such disease is systemic lupus erythematosus. The early differentiation between the two diseases is of utmost importance to institute appropriate treatment and reduce patient morbidity and mortality. Leprosy is a communicable, chronic granulomatous disease caused by *Mycobacterium leprae.* This clinically manifests predominantly with neurological and cutaneous features. However, it may also manifest with a variety of autoimmune phenomena indicative of autoimmune diseases, such as Systemic Lupus Erythematosus (SLE) or Rheumatoid Arthritis. Infection with *Mycobacterium leprae* not only mimics lupus flares, but possibly may also act as a trigger for lupus reactivation; however, its relationship is still not fully understood and explored. We report a case that was diagnosed as leprosy but retrospective analysis revealed that it was probably the initial manifestations of Lupus. During hospitalization the patient suddenly developed hypoxia and was found to have pulmonary haemorrhage. He was successfully managed with steroids, Mycophenolatemofetil along with other supportive treatment. Our case highlights the rare presentation of pulmonary haemorrhage in a male lupus patient and focuses on leprosy mimicking lupus.

Key words : Ulnar neuropathy, Systemic Lupus Erythematosus, pulmonary haemorrhage, Leprosy.

Introduction

Leprosy is chronic granulomatous, multi-system infectious disease caused by *Mycobacterium leprae*. It can mimic various collagen vascular diseases, including Systemic Lupus Erythematous, because of immunological alterations common in the disease, which affect the nerve and musculoskeletal system (Hsieh and Wu 2014). Leprosy, not only mimics lupus flares, but possibly the causative organism also acts as atrigger for lupus reactivation. However, its relationship with the auto immune phenomenonis still not fully understood & explored. We present the findings of a patient who initially was treated as a case of leprosy but later diagnosed to have Systemic Lupus Erythematosus and had a stormy course in the hospital due to pulmonary haemorrhage.

Case Report

A 28 years old male presented with complaints of fever and generalized weakness for 2 months. Fever was intermittent in nature, not associated

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with chills. Patient also noticed generalized weakness more in proximal muscles of lower limbs so much so that he had difficulty in climbing stairs. He had received multiple courses of antibiotics and anti-malarial therapy for fever.

One year back, the patient had complained of arthralgia involving small joints along with weakness in left hand. He was unable to perform fine movements with left hand. He developed clawing of left hand; there was no history of skin lesions. He was diagnosed as a case of Borderline Leprosy on clinical grounds and given MDT. There was no skin/nerve biopsy done earlier. He had received multi drug therapy in the form of Dapsone 100 mg/day, Rifamipicin 600 mg once a/ month and Clofazimine 50 mg daily. In addition he gave history of taking variable doses of dexamethasone for one year for treatment of his ulnar neuritis.

On general examination, patient was thin built, had significant pallor, bilateral pitting oedema, dry and ichthyotic skin with left side claw hand

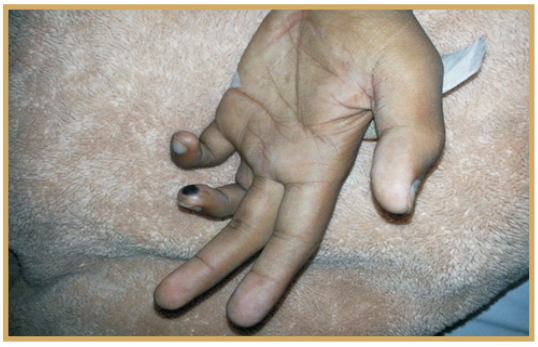

Fig 1 : Left hand showing flexion at proximal and distal interphalangeal joints along with hyperextension at metacarpophalangeal joint

(Fig 1). There was no thickened nerve or hypopigmented patch on his body. Musculoskeletal examination revealed mild proximal weakness of lower limbs power 4/5 according to MRC (Medical Research Council scale 1943). Rest of systemic examination was unremarkable.

The patient was investigated as a follow-up case of leprosy with inflammatory myopathy.

Table 1 : Investigations during hospital stay of patient

Investigations	At admission	7th day	Normal range
Haemoglobin (g/dl)	6	4	11 - 15
Total leucocyte count (thousand/ul)	4.1	8.6	4 - 10
Platelet counts (thousand/ul)	44	64	150 - 450
ESR (mm 1st hour)	72	82	< 13
Serum Albumin (g/dl)	1.3	2.2	3.5 - 5
AST/ ALT (IU/l)	20/34	69/32	< 42/60
Creatinine kinase (IU/l)	132	-	22 - 195
Serum Creatinine (mg/dl)	0.87	0.96	0.6 - 1.3
Serum CRP	29	36	< 6
Anti dsDNA antibodies (IU/ml)	>150	-	< 20
Serum C3 (mg/ l)	272	-	1032-1495
Serum C4 (mg/l)	86	-	167-385
24 hour proteinuria (mg/day)	1500	-	< 150
Serum LDH (IU/l)	467	450	91 - 180
Chest radiograph	Normal	Bilateral infiltrates	

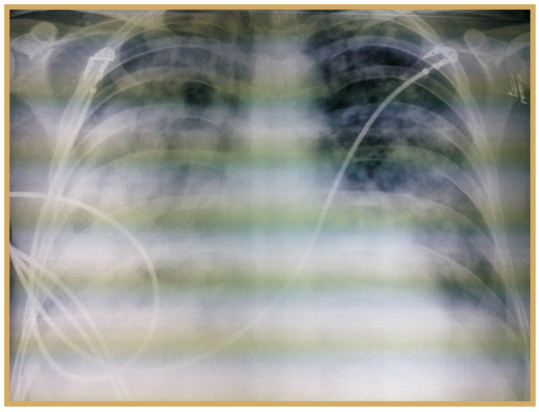

Fig 2 : Chest radiograph showing bilateral diffuse haziness in middle and lower lung fields

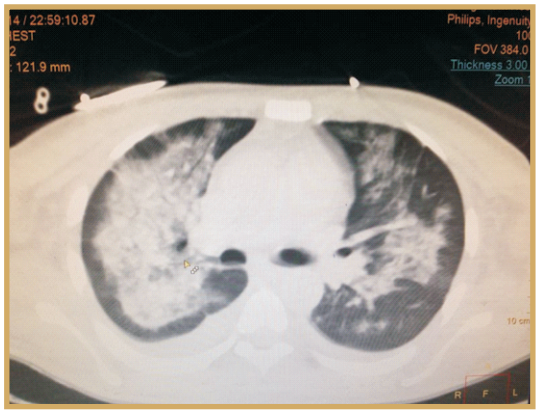

Fig 3 : Non contrast CT thorax showing bilateral infiltrates suggestive of alveolar haemorrhage.

Laboratory investigations revealed iron deficiency anaemia with hypoalbuminemia (Table 1). Blood and urine cultures were sterile. Pro inflammatory markers such as serum CRP (C reactive protein), and erythrocyte sedimentation rate (ESR) were elevated. Viral markers for infection with HIV, Hepatitis B, and Hepatitis C were negative. Hormonal essay to test the integrity of hypo-thalamo-pituitary-adrenal axis revealed that it was maintained.

The patient was diagnosed as a case of Systemic Lupus Erythematosus (according to Systemic Lupus Collaborating Clinics criteria, Petri et al 2012). The diagnosis was based on positive Anti-nuclear antibodies (ANAs 1:100; cytoplasmic); markedly elevated anti dsDNA antibodies (>150); positive direct Coomb's test; positive extractable nuclear antigens and Anti-SSA autoantibodies/ anti-Roautoantibodies (3+); Anti-ribosomal P protein antibodies ((3+) along with the clinical features. In addition, patient had lupus nephritis which was diagnosed on renal biopsy revealing focal proliferative glomerulonephritis (WHO class III), proteinuria and low complement levels. Nerve conduction velocity of limbs showed asymmetric large and small fibre sensory-motor axonal neuropathy involving left ulnar nerve and predominantly motor axonal neuropathy involving right median and ulnar nerve. Muscle biopsy was suggestive of myositis whereas sural nerve biopsy was normal. Echocardiography was normal with ejection fraction of 65%. Anti cardiolipin and lupus anticoagulant antibodies were negative.

During the 7th day of hospitalization, patient suddenly deteriorated, became hypoxic and his chest radiograph showed bilateral infiltrates more on right side (Fig 2). HRCT (high resolution computerized tomography) confirmed bilateral infiltrates possibly due to diffuse alveolar haemorrhage (DAH) (Fig 3). As his condition worsened he was intubated and shifted to ICU. Bronchoscopic lavage contained frank blood confirming the diagnosis of alveolar haemorrhage. He was managed aggressively and pulsed with Methyl Prednisolone (1 gm daily for 3 days), Mycophenolate Mofetil (1.5 g/day) and other supportive treatment. Bronchial lavage on cyto-pathological analysis revealed RBCs (haemorrhagic) with mostly polymorphs. By 10th day he was clinically better and his intubation was removed. The examination of his eyes revealed

cotton wool spots in both of his eyes suggestive of Lupus. He was finally, diagnosed a case of Systemic lupus Erythematous with Lupus Nephritis with Lupus Retinitis with Neuropathy and Pulmonary Haemorrhage. He was discharged on oral steroids and Mycophenolate Mofetil. He has completed eight weeks of follow up and is doing well.

Discussion

Leprosy is a great masquerade and mimics SLE therefore one has to be vigilant in diagnosing SLE in leprosy endemic areas (Petri et al 2012). It can be speculated if this patient had Lupus to start with earlier, and Lupusauto immunity was moderately controlled with Dapsone and Clofazamine both of which have anti-inflammatory and immunomodulatory activity. However, vice versa may also be true when cases of lupus may be diagnosed as leprosy due to the immunological features and overlap.

SLE in men can involve any organ in the body but men have more lymphopenia, thrombocytopenia, positive direct Coombs test, positive anti-Smantibodies, low C3 levels, elevated anti-dsDNA antibodies, and renal involvement than female counterparts (Teixeira et al 2011). The involvement of lung in both genders can result in pleuritis, diffuse interstitial lung disease, pulmonary embolism, pulmonary hypertension and pulmonary haemorrhage. Pulmonary haemorrhage is a rare complication of SLE with a reported frequency ranging from 1 to 5.4%, and mortality up to 23 - 92% (Tan et al 2012). The risk factors for alveolar haemorrhage include multi organ involvement, high SLEDAI (systemic lupus erythematosus disease activity index) scores (Gogia et al 2007). The patient had SLEDAI score of 12. Most cases of alveolar haemorrhage have lupus nephritis. The outcome of such patients is affected, and they require mechanical ventilation, treatment of secondary infection. Cyclophosphamide is used for the treatment of such auto immunity in addition to the control of above (Badsha et al 2004). The prevalence of peripheral neuropathy with SLE varies from 5 to 27% according to most of the studies (Canus et al 2007). Polyneuropathy in SLE occurs more frequently in patients with central nervous system involvement and high SLEDAI. Patients with SLE related neuropathy tend to be younger, and have a shorter duration of the disease. There is a somewhat a greater tendency for involvement of lower limbs nerves especially peroneal and sural nerves. However, in a study reported by Campello et al (2001), incidence of ulnar neuropathy was 30.4% of all cases of peripheral nerve involvement in SLE cases (Florica et al 2011). There are very few studies reporting on the pathogenic mechanisms of diffuse alveolar haemorrhage (DAH) in SLE. The proposed mechanisms include immune-mediated damage of small blood vessels and alveolar space. There are no standard treatment guidelines of DAH in SLE because of the rarity of the condition. The combination of Cyclophosphamide and Methyl Prednisolone has been shown to have good outcomes in some studies. Plasmapheresis can be used in refractory cases not responding to pulse therapy and Methyl Prednisolone. In a study one patient was successfully treated with Rituximab although its role in DAH is still not clear (Nellessen et al 2007).

Conclusions

Leprosy is prevalent in many parts of world including India. It can occasionally be misdiagnosed, and/or confused with other auto immune diseases like SLE. It should be considered in the Differential Diagnosis of such diseases, During severe life threatening complications requiring urgent interventions, such autoimmune

diseases need to be kept in mind to make the correct diagnosis and institute prompt and effective treatment.

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