Unusual association of diseases/symptoms

Dermatomyositis masquerading musculoskeletal tuberculosis

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Summary

Dermatomyositis is a rare rheumatic disease which predominantly affects the muscles and skin requiring a protracted course of immunosuppressants which may predispose the patients to opportunistic infections. A 49-year-old lady was diagnosed to have dermatomyositis in August 2010 based on history, significantly raised creatine kinase level and muscle biopsy findings. She had recurrent admissions due to fever, myalgia and muscle weakness. She had spiking temperature despite high dose steroids, broad-spectrum antibiotics and antifungal agents. This prompted extensive investigation which leads us to the additional diagnosis of disseminated tuberculosis involving the lungs, muscles and bones. This case demonstrates the challenge in controlling the disease activity of dermatomyositis with immunosuppressants in the setting of disseminated tuberculosis.

BACKGROUND

To the best of our knowledge, this is the first case report of a patient with dermatomyositis (DM) with concurrent disseminated tuberculosis (TB) involving the lungs and unusual sites such as muscles of the lower limbs and the ribs. Opportunistic infections among patients on high doses of immunosuppressants tend to be more extensive and involve atypical sites as illustrated by this case. Two pathologies occurred concurrently at the proximal muscles of the lower limbs which was initially a diagnostic dilemma followed by difficulties in treatment.

CASE PRESENTATION

In August 2010, a 49-year-old woman was diagnosed to have DM based on a history of progressive proximal muscle weakness associated with rash, laboratory findings of markedly elevated creatine kinase of 3952 U/l, positive anti-Jo 1 antibody and a muscle biopsy showing degenerated and inflamed myocytes. Her investigation for malignancy was negative. Her initial treatment included intravenous methylprednisolone of 1 g daily for 3 days followed by oral prednisolone of 40 mg daily and azathioprine of 50 mg daily. The patient responded well clinically with improvement in her muscle power and a declining trend of creatine kinase.

In September to November 2010, there were several attempts to taper down the steroids but the patient developed fever, myalgia and proximal muscle weakness even with a minimal reduction of 5 mg in the dose of steroids. Hence, the same dose of the immunosuppressants was maintained for months.

In December 2010, the patient presented with spiking fever associated with myalgia predominantly affecting her lower limbs. On examination, there were fine crepitations at bases of the lungs. Her proximal muscle power for the upper limbs was 4/5 whereas for the lower limbs were 3/5.

Her thighs and calves were tender. Her creatine kinase was normal 159 U/l. Chest radiograph showed consolidation in her right upper lobe. All her cultures including bronchoalveolar lavage were negative. Unfortunately, her symptoms did not settle despite broad-spectrum antibiotics and antifungal therapy, that led to the high index suspicion that the symptoms were attributable to active DM. Hence, the patient was pulsed with intravenous methylprednisolone 500 mg daily for 3 days. Besides, she was given intravenous immunoglobulin to treat her recalcitrant disease. Much to our disappointment, there was still no improvement.

In January to February 2011, the patient underwent a CT thorax which showed right-sided consolidation extending from the right upper to middle lobes (figure 1). Besides, a CT scan of the lower limbs revealed extensive

Figure 1 CT thorax showing right-sided consolidation extending from the right upper to middle lobes.

Figure 2 CT scan of the lower limbs showing extensive intermuscular collections extending from the gluteus to hamstring muscles bilaterally.

intermuscular collections extending from the gluteus to hamstring muscles bilaterally (figure 2). She had a bone scan done with findings consistent with chronic osteomyelitis of the left lower tibia and lesions in the right posterior ribs suspicious of metastasis (figure 3). As the ribs are an atypical site for skeletal TB or even other forms of infections, we were concerned about underlying malignancy and went on to investigate her further by doing a positron emission tomography (PET scan). The PET scan showed disseminated infection most likely TB involving her lungs, ribs and muscles of her lower limbs (figure 4). There was no evidence of malignancy. The right lung consolidation was biopsied whereas the collection in the right thigh was drained under ultrasound guided drainage. The histopathological examination of the right lung biopsy showed granulomatous inflammation with Ziehl-Neelson stain highlighting numerous acid fast bacilli. Pus from the right thigh abscess too stained positive for acid fast bacilli. A diagnosis of disseminated TB was made. She was started on antituberculous therapy that is, ethambutol, isoniazid, rifampicin and pyrazinamide. Her temperature settled and regained full proximal muscle power. Her steroids were tapered to 20 mg daily of prednisolone (0.5/mg/kg for body weight of 39.8 kg).

In April 2011, while on the above treatment regime, the patient was readmitted with spiking temperature associated with myalgia of the lower limbs and generalised bone pain. The proximal muscle power for her upper limbs was 4/5 whereas for the lower limbs was 3/5. Her creatine kinase level was normal but the C-reactive protein was markedly elevated; 14 mmol/l. There was no clinical evidence of concomitant infection. Her temperature and muscle power recovered after the prednisolone dose was doubled to 40 mg daily (1 mg/kg).

OUTCOME AND FOLLOW-UP

In May to June 2011, the patient was seen at the rheumatology outpatient clinics. She was doing well with full muscle power. Her steroids were tapered slowly by 5 mg per month. Azathioprine 50 mg was added. There was significant improvement radiologically. The antituberculous therapy was switched from intensive regime consisting of ethambutol, isoniazid, rifampicin and pyrazinamide to maintenance phase with isoniazid and rifampicin only.

DISCUSSION

This is the first case report of DM with disseminated TB involving the lungs, muscles and bones. Among immunocompromised patients diagnosed with TB, more than 50% have extrapulmonary involvement. However, extraspinal musculoskeletal TB has a rare frequency of only 1% to 2%.¹

The co-occurrence of musculoskeletal TB in the forms of tubercular fasciitis, tubercular myofascitis and tubercular myositis with DM has been reported previously. Interestingly, in almost all cases including this; the diagnosis of musculoskeletal TB was made within 6 months of the diagnosis of DM. This shows that patients are at higher risk of developing opportunistic infections during the initial phase of treatment which involves higher doses of immunosuppressants. The other similarities noted in most of these cases were the predominant involvement of the thigh muscles.²⁻⁶ The thigh muscles are proximal muscles which are commonly affected in DM. We believe that in severe DM, there is necrosis of muscles which acts as a focus for infection particularly opportunistic infections.

In this case, the patient was admitted three times over a period of 8 months. It was indeed a dilemma as her presenting symptoms for the second and third admission were the same that is, fever with myalgia but the final diagnosis

Figure 3 Bone scan showing increased tracer uptake in the left lower tibia and right posterior ribs.

Figure 4 PET scan showing hypermetabolic areas in the right lung, right posterior ribs and extensive involvement of the muscles of the lower limbs.

was different. We felt that her second admission was due to disseminated TB while the third was due to active DM as a consequence of rapid reduction of the dose of steroids. In addition, the simultaneous use of rifampicin reduced the

effects of the steroids. In one study, rifampicin was found to increase the plasma clearance of prednisolone by 45% and reduce the amount of drug available to the tissues (area under the plasma concentration time curve) by 66%.⁷

Yoshida *et al.* studied their dermatomyositis cases who suffered from TB fasciitis and found 11 weeks delay in arriving at the correct diagnosis.⁴ This case demonstrates the importance of considering tuberculous infection in all immunocompromised hosts and biopsy of suspicious lesions should be performed as soon as possible. Early diagnosis and prompt administration of antituberculous therapy can reduce morbidity and mortality.⁵

A multidisciplinary team comprising rheumatologist, chest physician, infectious disease physician and an orthopaedics surgeon were involved in this patient's management. We faced the challenge of controlling the disease activity of dermatomyositis with immunosuppressants without causing further spread of the tuberculous infection. The dose of immunosuppressants had to be titrated carefully within a narrow window as too low a dose could cause relapse of DM while higher doses may delay the recovery from TB. A previous case report of DM with TB recommended the use of rituximab in this setting.³ Fortunately, our patient responded to high doses of steroids with antituberculous therapy.

Competing interests None.

Patient consent Obtained.

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Learning points

- Persistent fever, muscle weakness and myalgia in a patient with underlying dermatomyositis which fail to respond to high doses of immunosuppressants should prompt the search of an alternative diagnosis such as musculoskeletal infection.
- There is a rare association between musculoskeletal TB and DM.
- It can be tricky to decide on the optimal dose of immunosuppressants to maintain remission in DM without worsening the spread of the coexisting tuberculous infection.
- The concurrent use of rifampicin with steroids reduces the efficacy of steroids and hence, causes relapse of the autoimmune disease treated.

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Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Rajalingham S, Mohd Said MS, Shaharir SS, AbAziz A, Periyasamy P, Anshar FM. Dermatomyositis masquerading musculoskeletal tuberculosis. *BMJ Case Reports* 2011;10.1136/bcr.08.2011.4675, date of publication

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