International Journal of Medical and Health Research
ISSN: 2454-9142, Impact Factor: RJIF 5.54
www.medicalsciencejournal.com
Volume 2; Issue 10; October 2016; Page No. 09-11

Rheumatoid myositis: An unusual presentation of rheumatoid arthritis maquerading as an inflammatory myopathy

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Abstract
A 55 year old presented with a seven month history of gradual and progressive weakness of all limbs marked proximally that started at about the same time. No preceding history of fever. However she noticed swelling of both wrists at the onset of symptoms. Examination revealed symmetrical wasting of all limbs with power of 3/5 globally. There was global diminution of her reflexes. Other systems were normal. Creatinine phosphokinase was markedly elevated at 2,550 U/L (26-140). Rheumatoid factor was also significantly elevated-1540.0 IU/ml (0-20.0). Antinuclear antibodies (ANA) was strongly positive at 16.0 U (<0.7-1.0); anti-double stranded DNA (dsDNA)-1.0 IU/ml (<10). Serum aldolase was 0.8 U/L (1.5-8.1). HIV was seronegative. She declined muscle biopsy. Full blood count was basically normal except for raised erythrocyte sedimentation rate of 60 mm/hr. She was commenced on oral prednisolone 60 mg daily with physiotherapy and had full recovery of muscle function after 4 weeks.

Keywords: rheumatoid, arthritis, myositis

1. Introduction
Rheumatoid myositis is defined as the presence of immune inflammatory infiltrates associated with variable degrees of skeletal muscle damage occurring in patients with rheumatoid arthritis [1]. It shares several clinical and laboratory parameters with idiopathic inflammatory myopathy, particularly polymyositis. However, it remains a largely underdiagnosed and undertreated condition [2].
Systemic autoimmune diseases are thought to be very rare in black Africans and very rarely reported [3]. Of recent, however, there have been some reports from Africa [4, 5, 6]. We describe a very rare connective tissue disease that to the best of our knowledge has not been previously described in our environment and also highlight our diagnostic and management experience

2. Case history
A 55-year-old businesswoman, known hypertensive of two years standing with poor drug adherence presented to us via the emergency department with an eight months history of gradual and progressive weakness of all limbs that started at about the same time and difficulty swallowing. She found it extremely difficult to raise her arms above her shoulder, comb her hair, rise from a seated position or climb a flight of stairs. She noticed a change in the quality of her voice with dysphagia which was initial to solids gradually worsened to involve liquids. However, there was no history of drooling. There was no history of sphincter dysfunction, paraesthesias, back pain or neck pain, dysphonia, dyspnea or drooping of her eyelids. The weakness of her limbs was not fatigable and did not progress or worsen over the day. She did not admit to preceding history of fever, cough or body rashes. However, she noticed swelling of both wrists at the onset of symptoms.
On examination, she was chronically ill-looking. There were no cranial nerve palsies. She had symmetrical wasting of the muscles of all limbs with generalized hypotonia. Power was grade 3/5 in all limbs with the weakness most pronounced in her shoulder and pelvic regions. There was a global diminution of her reflexes. Musculoskeletal examination showed tenderness with swelling at both wrist joints. Other systems were normal. Diagnostic considerations entertained initially included motor neuron disease, chronic inflammatory demyelinating polyneuropathy and inflammatory myopathy (Polymyositis).
Her results are as shown in Table 1. Human immunodeficiency virus screening (HIV) was negative for HIV I and II. She declined a muscle biopsy. Electromyography was not done for logistic reasons. Full blood count was basically normal except for raised erythrocyte sedimentation rate of 60mm/hr.
She noticed progressive improvements in muscle strength about two weeks after commencing steroids (60mg prednisolone daily) as she was at this time now able to walk unsupported. Her blood pressure was optimized with lisinopril 10mg and amlodipine 10mg. Physiotherapy was subsequently commenced to further optimize muscle strength. She was discharged in good clinical state after a 4-week period of hospital stay.

Table 1

Test	Value	Normal range
Rheumatoid factor	1540 IU/L	0-20 IU/L
Creatinine phosphokinase	2550 U/L	26-140 U/L
Serum aldolase	0.8 IU/L	1.5-8.1 IU/L
Antinuclear antibody	16	0.7-1.0
Anti-double stranded DNA	1.0	<10.0
Renal indices		
▪ Sodium	135 mmol/l	128-142 ol/l
▪ Potassium	4.0 mmol/l	3.4-4.8 mmol/l
▪ Bicarbonate	25 mmol/l	24-30 mmol/l
▪ Urea	1.4 mmol/l	2.4-6.0 mmol/l
▪ Creatinine	65 umol/l	60-120 mmol/l
Fasting blood sugar	3.7 mmol/l	3.5-5.5 mmol/l
Fasting lipid profile		
▪ Triglycerides	2.6 mmol/l	0.3-1.8 mmol/l
▪ Total cholesterol	4.6 mmol/l	<5.2 mmol/l
▪ HDL-cholesterol	0.4 mmol/l	>1.1 mmol/l
▪ LDL-cholesterol	3.7 mmol/l	<2.6 mmol/l
Thyroid function test		
▪ Thyroid stimulating hormone	2.4 U/L	0.4-6.8 U/L
▪ Free T4	1.7 pg/ml	1.4-4.2 pg/ml
▪ Free T3	1.3 ng/ml	0.8-2.0 ng/ml

3. Discussion

Our patient had symmetrical weakness of all limb muscles that was most significant proximally. This pattern is usually seen with polymyositis. However, she neither had fever or tenderness of her muscles which occurs characteristically in polymyositis. The presence of symmetrical swelling of her wrist which predated her muscle weakness led to the suspicion of rheumatoid arthritis. The elevations of her serum creatinine kinase and aldolase are in keeping with muscle involvement suggestive of an inflammatory myopathy. However, her rheumatoid factor by quantitative method was grossly elevated which further heightened our suspicion of rheumatoid arthritis. Though our patient did not have a muscle biopsy, it is the only investigation that would clearly demonstrate the presence of muscle involvement in rheumatoid arthritis. Muscle involvement is usually moderate affecting mainly proximal muscles in an asymmetrical and patchy manner, hence multiple section analysis is usually needed to diagnose this condition [7, 8]. And also, a biopsy is the only way to definitively differentiate the different types of myositis [9].

As is typical of the inflammatory myopathies, the response to steroid in our patient was quite remarkable. Individuals with inflammatory myopathy have active periods of the disease that occur as flares and typically respond to treatment in a month or two and generally regain strength after two to three months. Corticosteroids have a positive effect on muscle strength [11]. They dampen inflammation and the immune response by interfering with the processing of antigens and with the early triggering of T cell and B cell production [12].

4. Conclusion

Rheumatoid myositis is not a common entity that we encounter daily. Though rheumatoid arthritis is a much commoner disease encountered in clinical practice, a very high index of suspicion is needed to make the diagnosis when it presents atypically as an inflammatory myopathy. Prompt diagnosis and institution of appropriate treatment would go a long way in improving patient's functional capacity.

5. Acknowledgement

Thanks to Dr. Pedro Emem-Chioma for proofreading this work and for his input towards the production of this paper.

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