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CASE REPORT

A Case of Viral Myositis Simulating Polymyositis

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Abstract

Acute viral myositis is a rare condition that may occur even during the recovery phase of an illness. We report a case of a 72-year-old female who presented with proximal muscle weakness and myalgia three weeks after an episode of fever. A thorough clinical, laboratory, and electrophysiological evaluation was done. The initial creatinine phosphokinase (CPK) was very high. The nerve conduction study (NCS) was normal, and needle electormyogram (EMG) showed some features of denervation with myopathic motor unit action protential (MUAPs). Histopathological findings were also suggestive of inflammatory myopathy. Both anti-cytomegalovirus-IgM and IgG were positive. However, the patient improved rapidly over a short period with normalization of CPK without any corticosteroid or immunosuppressive treatment. Based on the above features and the self-limiting nature of the disease, the patient was diagnosed as a case of Cytomegalo virus (CMV) induced myositis. Viral myositis is a self-limiting myopathy that mimics polymyositis as both having similar clinical and laboratory features. Though it is not common, early diagnosis of viral myositis, if possible can reduce the hazard of long-term use of immunosuppressive medications.

Key words: Cytomegalovirus, creatine kinase, polymyositis, viral myositis.

Introduction

Myositis is defined as an inflammation of the muscle which is characterized by pain, tenderness, swelling, and weakness. The etiology of myositis includes autoimmune disorders, genetic disorders, medications, endocrine disorders, and infections. Infectious myositis may be due to a wide variety of pathogens including bacteria, viruses, parasites, and fungi. Bacterial myositis presents as focal muscle infection, whereas viruses and parasites tend to cause diffuse disease with generalized myalgias and multifocal myositis. Viruses can induce myositis through persistent infections, molecular mimicry, production of immune complexes, immune dysregulation, or other mechanisms. The viruses causing myositis are influenza A/B, parainfluenza, coxsackie, herpes simplex, Ebstein Barr, cytomegalovirus, adenovirus, HIV, and Human T-Lymphotrophic virus type-1. These myositis are associated with spontaneous recovery in most cases but potentially dangerous complications such as rhabdomyolysis, myoglobinuria, acute renal failure, cardiac arrhythmias, and compartment syndrome have been associated with significant morbidity.[1] We report a case of self-limited viral myositis associated with cytomegalovirus infection in an elderly woman.

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The case presentation

A 72-year-old female housewife presented with progressive muscle pain and weakness of all four limbs for 7 days. Weakness was acute in onset and progressive. It was more marked in proximal group of muscles without any tingling, numbness, and diurnal variation. Her strength gradually deteriorated until she developed difficulty in walking and standing from squatting and combing her hair. Three weeks prior she had fever, myalgia, rhinitis, and anorexia. Her fever and sore throat resolved after two days, but muscle pain persisted which evolved into diffuse muscle weakness in her lower extremities. Weakness was not associated with any muscle wasting, skin rash, bowel and bladder dysfunction, and dysphagia. She also denied a history of diarrhea, vomiting, vaccination, or medication use before her illness. She had no similar type of attack in the past and no family history of such type of illness. She is nondiabetic but hypertensive. Her vital signs and general examination were normal. She had no rashes or joint pain. Her neurological examination revealed normal higher psychic function with intact cranial nerves. There was no muscle wasting, mild tenderness was present in both calf muscles and thigh muscles, tone was slightly

reduced but power was in the upper limbs proximally 3 and distally 4, whereas in lower limbs proximally 2 and distally 3. All the deep tendon reflexes were normal and the plantar was bilaterally flexor. All modalities of sensation and cerebellar function were normal. Laboratory results were notable for a CK-MM of 17,493 U/L and serum electrolytes Na-126.1,K-1.74, Cl-78.7mmol/L, low serum calcium (7.6mg/dl) and magnesium (1.1mg/dl). Renal function was normal with a serum creatinine of 0.9 mg/dl. The nerve conduction study was normal and needle EMG showed some features of denervation with low amplitude, polyphasic, short duration MUAPs which may be due to hypokalemic paralysis or inflammatory myopathy.

After correction of electrolyte imbalance, her muscle power slightly improved but CPK was persistently high. So patient was thoroughly investigated considering the diagnosis of polymyositis or viral myositis. Muscle biopsy report was mild perimysial inflammation with focal perivascular infiltrate compatible with inflammatory myositis. Detailed immunologic screening for connective tissue disorder and tumor markers yielded negative results. Human immunodeficiency virus (HIV), and hepatitis virus screens were negative. Chest X-ray, abdominal ultrasound, thyroid profile, and CRP all are within normal limits. But cytomegalo viral IgM and IgG both were found strongly positive. She was managed symptomatically with paracetamol and NSAIDs. We were planning to give corticosteroids but dramatically CPK level fell without steroids. After several days her condition gradually improved and CPK decreased to 57U/L. A few days later she was discharged from the Hospital. During discharge, her muscle power was proximally 4+, distally 5, and at normal CPK level. The patient's clinical recovery continued over the course of a few weeks and full muscle strength was regained by 1 month. Though the condition simulates poly myositis but responds spontaneously without any corticosteroid like other viral myositis.

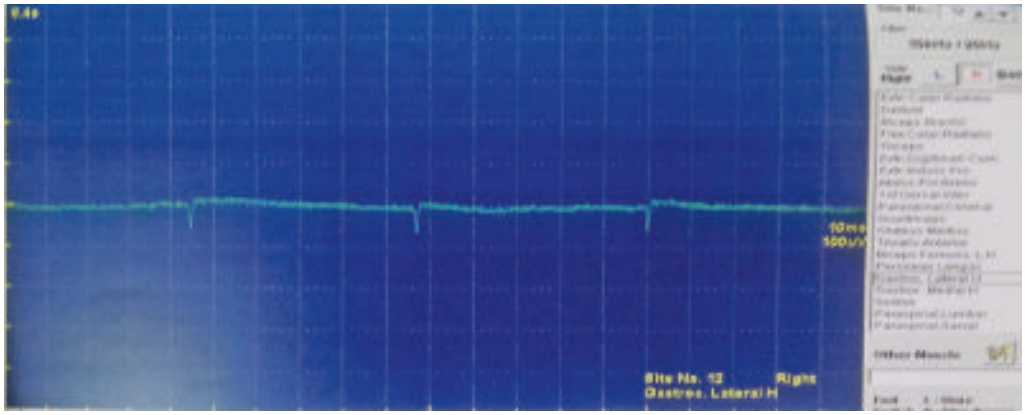

Figure 1: Electroencephalogram showing positive sharp waves (features of denervation)

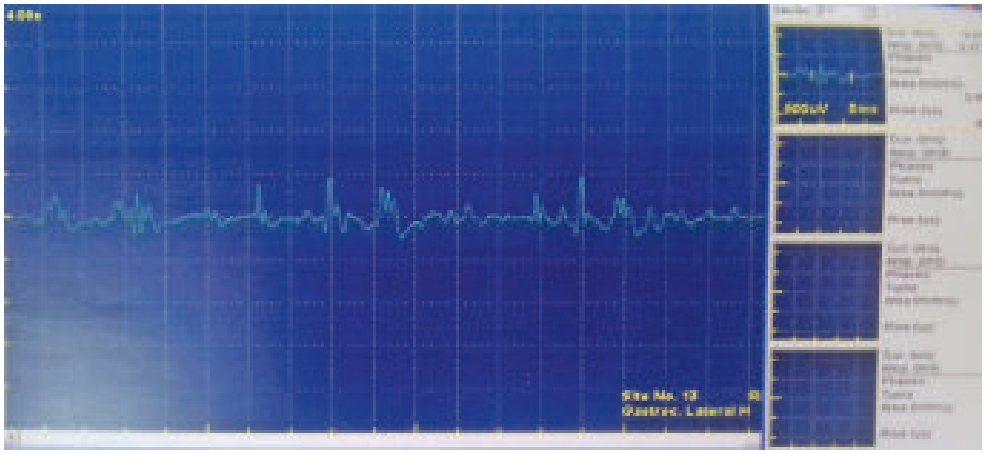

Figure 2: EMG tracing showing myopathic MUAP (poly phasic, low amplitude short duration).

EMG Findings Summary

Muscle/Side		Inc Act	Fibs.	Pos. Wave	Fasc.	MYO. Disch.	Normal MUP	Poly	Low Amp.	High Amp.	Dur.	Recruit	Int. Patt.
Gastroc. Lateral H	L	Normal	0	+1	0	0	0	+++	+2	0	Short	Full	Full
Gastroc. Lateral H	R	Normal	0	+1	0	0	0	++	+2	0	Short	Full	Full
Tibialis Anterior	R	Normal	+1	+1	0	0	0	+++	+1	0	Short	Full	Full
Quadriceps	R	Normal	0	0	0	0	+1	+1	+1	0	Short	Full	Full
1st Dorsal Inter.	L	Normal	0	+1	0	0	0	++	+2	0	Short	Full	Full
Tibialis Anterior	L	Normal	+1	+1	0	0	0	+++	+2	0	Normal	Full	Full
Quadriceps	L	Normal	0	0	0	0	+1	++	+1	0	Short	Full	Full
1st Dorsal Inter.	R	Normal	0	0	0	0	+1	++	0	0	Normal	Full	Full
Extn.Digitorum Com	R	Normal	0	0	0	0	0	++	+1	0	Short	Full	Full
Deltoid	R	Norma	0	0	0	0	0	++	+1	0	Short	Full	Full

Figure 3: EMG findings showed some features of denervation with myopathic MUAPs (polyphasic, low amplitude, and short duration). These features simulate inflammatory myositis.

Discussion

Numerous infections can cause myositis and may be confused with polymyositis. Inflammatory myopathies may be primary, with other connective tissue disease, eosinophilic myositis, sarcoidosis, systemic vasculitis, and infectious causes including viral, bacterial, and fungal. Infectious myositis presents as severe myalgia associated with very high CK levels and nonspecific myopathic changes including fiber necrosis and inflammatory cell infiltration on muscle biopsy. Our patient is a 72-year-old female who presented with severe myalgia with proximal muscle weakness 3 weeks after an episode of fever and very high CKP.

Clinically it is sometimes difficult to differentiate between polymyositis and viral myositis. Hudgson and Walton have stated that the cardinal clinical feature of viral myositis is severe myalgia with little weakness and recovery usually within days.[2] Rowland et al. have observed the clinical diagnosis of polymyositis sometimes included patients with self-limited viral myositis.[3] However, the inflammatory cell infiltrate in muscles is commonly found in typical polymyositis and rarely found in self-limited viral myositis.[4]

Numerous infections can cause myositis but viruses are the most common. Self-limiting viral myositis is more common among children and has been seen to be commonly associated with influenza A and B.[5]

Our patient had features of myositis with very high CK and EMG features suggestive of inflammatory myositis but the muscle biopsy feature was not typical of polymyositis. As there was spontaneous and complete recovery of our patient without steroids and another immunosuppressant we leveled the case as viral myositis rather than polymyositis. We found high Titer of Cytomegalovirus antibody in serum both IgM and IgG.

Symptomatic CMV infection in nonimmuno-compromised hosts has traditionally been considered to have a benign, self-limiting course. However, in the medical literature, there are a considerable number of reports of severe clinical manifestations of CMV infection in immunocompetent patients.[5]

Among the wide range of etiology of acute myositis, viral myositis is the most common. CMV as a cause of acute myositis is rare and commonly reported with immunosuppressive conditions like HIV and after organ transplant.[6] Muscles are affected either directly by invasion of the virus or may be damaged by inflammatory cytokines and autoantibodies triggered by the virus. Acute myositis, caused by CMV infection, leading to severe rhabdomyolysis even in immunocompetent individuals has been reported .[7]

In the literature, less than 10 cases of CMV myositis have been reported and the maximum number of cases were from younger than 40 years.[7-12] Muscle pain associated with very high creatine kinase and muscle biopsy findings along with EMG findings are diagnostic. Laboratory diagnosis was confirmed by seroconversion from IgM to IgG but CMV PCR was negative in maximum reported cases.

Our case also had severe myalgia followed by proximal weakness, high creatine kinase, positive Cytomegalovirus IgM to IgG, and muscle biopsy favors myonecrosis which confirms the diagnosis of acute myositis. We had ruled out toxins, trauma, drugs, autoimmune disorders, endocrine and other viral causes. Spontaneous recovery occurred within a few days without steroids or other immunosuppressive drugs.

Both viral myositis and polymyositis can present with similar biochemical parameters and electrophysiological and muscle biopsy features. However in clinical practice, it is sometimes very difficult to differentiate between polymyositis and viral myositis. Viral myositis should be suspected when the patient presents with weakness of acute or subacute onset after an antecedent upper respiratory or gastrointestinal infection with spontaneous improvement and normalization of CPK without steroid or immunosuppressant. Thus it is essential to differentiate viral myositis from polymyositis as the former is benign and self-limiting and the latter requires long-term corticosteroid with other immunosuppressive treatment.

Conclusion

Viral myositis is a self-limiting myopathy that mimics polymyositis as both having similar clinical and laboratory features. However early differentiation is important because viral myositis recovers spontaneously whereas polymyositis needs aggressive treatment with high-dose corticosteroids and immunosuppressants. Though not common early diagnosis of viral myositis if possible can reduce the hazard of long-term use of these drugs.

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References

1. Bagley WH, Yang H, Shah KH. Rhabdomyolysis. Intern Emerg Med 2007;2:208-10. DOI: 10.1007/s11739-007-0060-8.
2. Hudgson P, Walton JN. Polymyositis and other inflammatory myopathies. In: Vinken PJ, Bruyn GW, eds. Diseases of muscle: part II (Handbook of clinical neurology, vol. 41). Amsterdam:
3. Rowland LP, Clark C, Olarte M. Therapy for dermatomyositis and polymyositis. Adv Neurol 1977; 17: 63–97. PMID: 888742
4. Naylor CD, Jevnikar AM, Witt NJ. Sporadic viral myositis in two adults. Can Med Assoc J 1987; 137: 819–21. PMID: 2832046
5. Jacobs BC, Van Doom PA, Groeneveld JHM, Tio-Gillen AP, and Van der Meche FGA. Cytomegalovirus infections and anti-GM2 antibodies in Guillain-Barre´ syndrome. J Neurol Neurosurg Psychiatr 1997;62:641–643. DOI: 10.1136/jnnp.62.6.641
6. Crum-Cianflone NF. Bacterial, fungal, parasitic, and viral myositis. Clin Microbiol Rev. 2008;21(3):473–94. DOI:10.1128/CMR.00001-08
7. Gindre H, Féasson L, Auboyer C, Cathébras P. Severe rhabdomyolysis associated with a primary cytomegalovirus infection in an immunocompetent patient. BMJ Case Rep. 2013;13:bcr2012008140. DOI: 10.1136/bcr-2012-008140
8. Sato K, Yoneda M, Hayashi K, Nakagawa H, Higuchi I, Kuriyama M. A steroid-responsive case of severe rhabdomyolysis associated with cytomegalovirus infection. Rinsho Shinkeigaku. 2006 May;46(5):312-6. Japanese. PMID: 16886796.
9. Maeda M, Maeda A, Wakiguchi H, et al. Polymyositis associated with primary cytomegalovirus infection. Scand J Infect Dis. 2000;32:212–4. DOI: 10.1080/003655400750045367
10. Yasumoto N, Hara M, Kitamoto Y, et al. Cytomegalovirus infection associated with acute pancreatitis, rhabdomyolysis and renal failure. Intern Med. 1992;31: 426–30. DOI: 10.2169/internalmedicine.31.426
12. Hirohama D, Shimizu T, Hashimura K, et al. Reversible respiratory failure due to rhabdomyolysis associated with cytomegalovirus infection. Intern Med. 2008;47:1743–6. DOI: 10.2169/internalmedicine.47.1349