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# A 50-Year-Old Female with Quadriparesis Secondary to Viral Myositis: Unusual Presentation and Diagnostic Approach

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**Abstract:- It's not common to encounter acute viral myositis that occurs during convalescence from illness, most commonly influenza. Myalgia with weakness and isolated laboratory finding of elevated serum creatine kinase (CK) are hallmarks of this disease. The disease is often thought to occur only in children with male predominance and weakness predominantly in calf muscles. Recovery usually takes a few days and is usually spontaneous. This case therefore indicates that it can occur in adults with atypical presentation. Close differentials are inflammatory and hereditary myopathies which require subjecting patients to unnecessary invasive testing. It is well proven in numerous studies that invasive testing such as muscle biopsy has no role in treatment as well as prognosis in cases like benign acute pediatric myositis, therefore physicians should be aware of such cases and avoid unnecessary anxiety-provoking and invasive testing.**

***Keywords:-*** *Quadriparesis, Viral Myositis, Diagnostic Approach, Biopsy.*

## I. INTRODUCTION

Multiple viruses are linked with acute myositis, with influenza being the most common. [(1)] Influenza B is more likely to cause myositis due to the presence of NB protein in the membrane than influenza A, which aids in viral entry and may have a myotropic effect. [(1,2)] Since the viral agent has not consistently been detected in biopsy samples, it is unknown if this is the result of immunological mechanisms, myotoxic cytokines, or direct viral infection of the muscle. [(1)]

Myositis is most common in children and only happens in a tiny percentage of influenza patients (mean age of eight years), known as benign acute childhood myositis, possibly because of virus tropism for immature muscle cells, males are more commonly affected. [(1)] It is associated with a mild to moderate rise in serum creatine kinase levels (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST). [(3)]

During the early stages of the virus' recovery, the most common symptom of viral myositis is the abrupt onset of muscle weakness, discomfort, and soreness. Symptoms are often limited to calf muscles, but other muscle group involvement is also reported. [(4)] Usually, it goes away on its own after a week of symptoms starting when healing starts. However, reports of rhabdomyolysis with renal failure and compartment syndrome are documented. [(5)] Typical symptoms in adults are characterized by muscle pain and severe weakness in the proximal muscles of the upper extremity, which resolve slowly over weeks rather than days. [(6)] Viral and postinfectious myositis in adults are uncommon but have been reported with influenza, mononucleosis, cytomegalovirus infection, echovirus 9, and viral hepatitis. [(1)]

Muscle fibre degeneration and myonecrosis with leukocyte infiltration are the pathological hallmarks of muscle biopsy [(7)] But paediatricians know that invasive tests are futile. [(8,3)] By taking into account the typical clinical and laboratory findings of this disease and identifying more serious pathological conditions that should be ruled out, benign acute childhood myositis can be diagnosed. This can prevent unnecessary diagnostic tests and can ensure an excellent prognosis for patients. [(3)]

## II. CASE DESCRIPTION

A 50-year-old female patient, a resident of rural Karnataka (India) and, a farmer by occupation presented to the ER with complaints of truncal weakness associated with weakness, pain and swelling of bilateral upper and lower limbs for one month. The patient gives a history of fever one month back which lasted for two days associated with upper respiratory tract symptoms and pain in large joints which was aggravated on activity and relieved on rest. The patient sought treatment for the same from a local hospital in her village, and symptoms were relieved for a few days.

Three weeks before admission, the patient complained of persistent pain in bilateral upper and lower extremities along with soreness of her hand and legs, which was not relieved by medication restricting her movements.

In the next two days, the patient noticed swelling in her upper and lower extremities such that her clothes and bangles became tight, and she was not able to put her feet in her footwear.

Two weeks before admission, she developed difficulty in getting up from the squatting position and combing her hair. Weakness slowly progressed over the next week making her

unable to eat, dress or undress by herself along with difficulty walking by herself.

One week before admission, she could not get up from lying down or roll in bed from side to side.

5 days before admission, she could not hold her neck upright and was then referred to us for further evaluation.

On the day of admission, she was afebrile with stable vitals. On further probing, there was no history of loss of sensation, dysphagia, dysarthria, nasal regurgitation, facial asymmetry, diplopia, visual disturbances or headache. There was no history of bowel or bladder involvement or a band-like sensation, shooting pain, or involuntary movement/seizure-like activity.

There was no history of neurological illness or any other comorbidity. There was no history of drug intake or significant family history. Her last COVID-19 vaccination dose was 6 months back.

On examination, she had a normal general physical examination apart from bilateral pedal edema extending to her thighs which was tender on touch. There was no rash or subcutaneous nodule or any other neurocutaneous marker.

Table 1 Case Examination

| Parameter | Reference Range | Admission | Day 1 | Day 3 | Day 7 | Day 15 | Day 24 |
|---|---|---|---|---|---|---|---|
| Haemoglobin (g/L) | 12-15 | 17.2 | | 17.5 | 12.0 | 11.3 | 12.2 |
| PCV (%) | 37-47 | 52.5 | | 54.2 | 35.8 | 36.7 | 38.1 |
| RBC (x 10^6/microL) | 3.80-4.80 | 5.89 | | 6.00 | 3.64 | 3.87 | 3.86 |
| Platelets (x 10^3/microL) | 150-450 | 339 | | 314 | 189 | 199 | 239 |
| Total Counts (x 10^3/microL) | 4-10 | 18.13 | 17.80 | 18.34 | 11.93 | 9.93 | 6.89 |
| Urea (mg/dl) | 17-49 | 32 | | 51 | 30 | 19 | 25 |
| Serum Creatinine (mg/dl) | 0.60-1.17 | 0.7 | | 0.8 | 0.9 | 0.6 | 0.6 |
| Total Bilirubin (mg/dl) | 0.0-1.1 | 0.3 | | 0.4 | 0.2 | 0.6 | 0.7 |
| SGOT/AST (U/L) | 0-35 | 1133 | | 1298 | 187 | 27 | 25 |
| SGPT/ALT (U/L) | 0-45 | 352 | | 375 | 311 | 18 | 16 |
| Alk. Phosphatase | | 159 | | 195 | 56 | 84 | 56 |
| CPK (U/L) | 20-180 | - | 3688 | 2194 | 9938 | 107 | |
| Sodium (mEq/L) | 136-145 | 144 | 138 | 136 | 142 | 135 | 138 |
| Potassium (mEq/L) | 3.5-5.10 | 4.0 | 4.46 | 3.7 | 3.9 | 3.6 | 4.1 |
| LDH | | 1524 | | | | | |

Her higher mental function, cranial nerve examination, sensory examination, and reflexes were normal along with no cerebellar signs, and intact cortical function with no signs of meningeal irritation. Although, there was a motor deficit in the upper limb– shoulder (2/5), elbow (2/5), wrist (3/5), and finger (4/5) with poor handgrip. Her lower limb power – hip (1/5), knee (2/5), ankle (4/5) was present along with poor neck and truncal flexion.

On the day of presentation, she had raised haemoglobin levels of 17.2 g/dl (normal 12-15g/dl) with elevated hematocrit of 52.5% (normal 36-46%) suggesting a dehydrated state, along with elevated ALT of 352 U/L (normal 1-33 U/L) and AST of 1133 U/L (normal 1-32 U/L). Her total count was 18.13 x $10^9$/L (normal 4.5-11.0 x $10^9$/L), and she was started on IV antibiotics along with IV fluids to treat any superadded infection and dehydration. Her HIV, Hepatitis B and Hepatitis C results came back negative. Dengue, leptospirosis and typhoid were also ruled out.

On Day 2 of hospitalization, she had elevated serum creatine kinase levels of 3688 U/L along with an elevated LDH value of 1524 U/L with normal serum PCT, thyroid profile, serum electrolytes and urine examination. Diagnosis of myositis secondary to unknown etiology was made however any invasive testing like EMG, NCV or muscle biopsy was deferred.

On day 3 of hospitalization, her serum cortisol levels were within normal range, ANA profile, myositis panel and rheumatoid factor were sent, which was negative.

Our differentials were myositis secondary to autoimmune aetiology or viral aetiology, she was started on steroid pulse therapy (Injection methylprednisolone 1g/day for 5 days) followed by tablet methylprednisolone 1mg/kg body weight in a tapering manner for the next 4 weeks. If symptoms recurred on a tapering dose of steroid, we would incline towards autoimmune aetiology.

On day 8 of hospitalization, her muscle power improved and by day 15, her liver function test normalized.

On day 24 of admission, her serum creatine kinase levels were within normal limits (107 U/L), and she was discharged after 4 weeks of hospitalization with normal bilateral upper and lower limb power of 5/5.

## III. DISCUSSION

The case of a 50-year-old female presenting with a gradually progressive weakness predominant in the truncal region and bilateral proximal upper and lower limb muscles with no other abnormal central nervous system examination along with elevated serum creatine kinase levels, deranged

ALT, AST succeeding a fever episode with a prolonged recovery time describes a disease remarkably similar to benign acute childhood myositis (BACM) in terms of onset during convalescence phase of a viral illness, distribution of pain in limbs and elevated creatine kinase levels, however, it also highlights the unique features such as the age and gender of the patient, time taken for onset of symptoms and recovery and the ability of diagnosis without the use of invasive tests such as EMG and biopsies.

In 1955, LUNDBERG first described an illness that started with high fever and headache. When the temperature nearly returns to normal and the headache subsides, usually after three to four days, the pain appears in the calf. It was termed 'myalgia cruris epidemica' and was reported in 70 children and four adults, however later the adult cases were overlooked and 'myalgia cruris epidemica' was renamed benign acute childhood myositis. [(9)] Since then, many studies have been carried out in the pediatric population and similar diseases in adults have been poorly understood, therefore understanding a case of an adult presenting with a similar constellation of symptoms and laboratory values becomes important.

A similar study done by Mackay et al. in 1999 described benign acute myositis of childhood as a disorder of mid-childhood (mean age of 8.1 years), typically affecting young males. Symptoms include calf pain and difficulty walking after a viral illness with spontaneous recovery in one week. He also highlights that biopsy done revealed nonspecific degenerative changes, focal muscle fibre vacuolation, and extensive muscle necrosis, however, several biopsies did not show any inflammatory changes as the process of the disease is 'patchy' and it is exceedingly rare to isolate influenza or other viruses from the muscle [(10)] which is in concordance with our approach of refraining from invasive testing such as biopsy.

Initial investigation results of elevated CK, LDH, AST and ALT along with thorough history taking and clinical examination ruled out several causes of quadriparesis. They inclined us towards the diagnosis of myositis of unknown aetiology, close differentials such as inflammatory, hereditary, and metabolic causes were ruled out by a negative myositis panel, negative ANA profile and a normal thyroid function test. However, a similar conclusion was drawn by Neocleous et al. in 2012 which emphasises that the correct diagnosis of BACM is by evaluating the clinical and laboratory findings and by recognising other serious medical conditions, which must be excluded from the diagnosis by a detailed patient workup including a detailed CNS examination, blood investigations including serum CPK and CRP values and a urinary analysis (to rule out rhabdomyolysis) are enough for the diagnosis and can avoid unnecessary diagnostic tests and reassure parents and patients of a good prognosis. [(3)]

Chanson et al. in 2018 described a BACM-like illness in a 32-year-old patient with severe pain and oedema in both legs associated with lower extremity motor impairment, The biopsy of the left anterior tibial muscle showed necrosis and muscle cell regeneration without inflammatory infiltration, the patient recovered after a few days. According to him, the diagnosis of benign viral myositis was first disregarded since it was thought to be a condition that only affected children.

A muscle biopsy was thus performed while it is not useful for the diagnosis of benign acute childhood myositis which is following our theory of non-invasive diagnostic algorithm. He also said that any inflammatory or genetic myopathy would have relapses. [(8)] Our case is also of the adult age group, however, there are some differentiating features such as the gender (female) and time duration of onset and recovery, which is longer in our case. A causal infectious agent was also not found in our case; however, it can be explained as the search for a virus is done after the symptomatic period when the pathogen has presumably been eliminated.

Scaber et al. in 2020 highlighted that in adults, proximal muscles of bilateral upper and lower limbs can be involved in an illness like BACM which resolves over weeks rather than days which is similar to our case where recovery took an entire month. Additionally, he hypothesises that the most plausible explanation is that the virus directly causes muscle cell necrosis through its action on muscle cells, or that the effects of the virus's immune response are responsible for the short 3-day delay between the start of the flu-like sickness and the onset of neurological symptoms. [(11)]

Naylor et al did a study and found a usual presentation of two female adults who presented with insidious onset bilateral upper and lower limb weakness following an episode of viral infection, the latency period in each case was about 10-14 days (about 2 weeks), with recovery taking few weeks rather than days which is similar to our case where patient took around three weeks to develop a full constellation of symptoms and recovered in one-month duration. He also says biopsy shows features suggestive of necrosis and inflammation and rarely a virus has been isolated from a biopsy specimen [(6)] thereby again proving the fact that even in cases which are atypical in terms of duration, investigation like biopsy has no role to play in treatment or prognosis. He also mentions two cases of myositis secondary to viral infection where corticosteroids were started, to which both patients responded well like our case.

A study done by Gibson et al. during the H1N1 epidemic of 2009 highlights the fact that H1N1 disproportionately affects younger and healthier patients when compared to typical influenza, postulating that presentation depends on the strain of the virus. He also mentions that a negative muscle biopsy does not rule out viral myositis and therefore muscle biopsy is not routinely recommended in such cases. [(12)] This does makes us wonder whether the atypical presentation in our case could be due to different strains of influenza prevalent at the time in that geographical region or like epidemics of the past, the recent epidemic of COVID-19 has a lingering effect.

To answer our last question, more research on a wide scale including more patients from different regions needs to be done.

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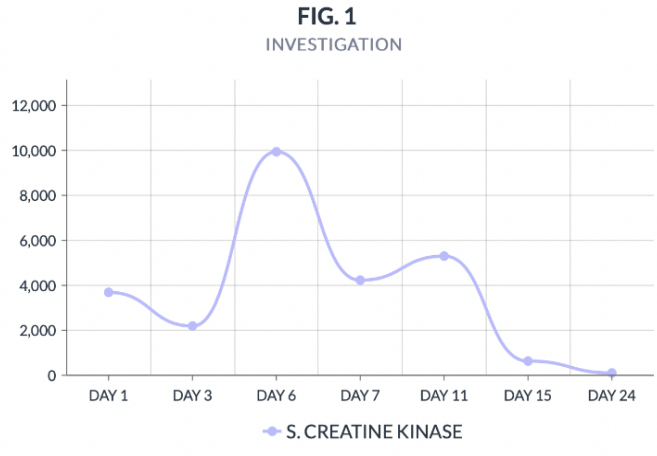
## IV. INVESTIGATION




## V. CONCLUSION

The patient presented with atypical presentation of a disease which is like benign acute childhood myositis (BACM), the diagnosis was made based on a thorough history taking ruling out an extensive list of differential diagnoses along with detailed physical examination including central nervous system examination and positive laboratory results. After ruling out inflammatory causes of myositis, our differential diagnosis was myositis secondary to viral vs autoimmune cause. The decision of not subjecting our patient to unnecessary invasive procedures like EMG, NCV and muscle biopsy was made which would not have any significant difference to the treatment. Instead, the patient was given steroid pulse therapy followed by a tapering dose steroid keeping in mind that any autoimmune cause would flare up on withdrawal of steroids. The patient responded well and made a full recovery with 4 weeks of steroid therapy along with supportive care, aggressive physiotherapy and rehabilitation.

The patient was followed up at 1 month, 3 months and 6 months of duration, there was no evidence of any recurrence with the patient able to carry out her usual activity without any difficulty, helping us retrospectively incline towards the diagnosis of myositis secondary to viral infection.

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