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MYOSITIS AS A COMPLICATION OF DENGUE VIRAL INFECTION

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ABSTRACT
Dengue fever is a febrile illness caused by an arbovirus transmitted by the Aedes aegypti mosquito. It occurs as a seasonal epidemic every year in Pakistan. Apart from febrile illness and its associated hemorrhagic complication, dengue fever is associated with multi-system involvement and their respective complications, including myriad neurological complications. In this case report we describe two patients who developed acute viral myositis.

Keywords: Dengue, Dengue fever, Myositis

INTRODUCTION
Dengue is a febrile illness caused by Dengue virus transmitted by Aedes aegypti mosquito bites. Dengue infection may present as asymptomatic or sometimes lead to life-threatening illnesses like dengue hemorrhagic fever and dengue shock syndrome. Dengue is associated with many neurological complications, which include myositis, myalgias, rhabdomyolysis, and hypokalemic periodic paralysis.[1] The critical phase lasts for 24-48 hours. Clinical manifestations which occur during the critical phase may include liver failure, CNS manifestations like Guillain Barre Syndrome (GBS), dengue encephalitis, myelitis, musculoskeletal manifestations like myositis, myocardial infarction, and acute kidney injury. Globally about four billion people are susceptible to dengue virus infection and about 70% of them are in Asia.[2] Myositis with viral infections occurs but myositis associated with dengue fever is rarely reported and it can lead to life-threatening complications like acute renal failure and rhabdomyolysis. The literature review showed about 30 published reports on dengue-associated myositis, one study reported that 4% of patients with dengue fever may develop neuromuscular complications such as myositis, GBS, and rhabdomyolysis of varying severity.[3]

CASE PRESENTATION
Case 1:
A 21-years old female, student, with no known prior comorbids, presented in ER of Pakistan Institute of Medical Sciences with the complaint of high-grade fever for five days, which was acute in onset, progressive in pattern, continuous with no diurnal variations, associated with episodic headache which was involving frontal and temporal region. Headache lasted for 4-5 hours, relieved by taking paracetamol. It was not associated with nausea, vomiting, lacrimation, stuffy nose. It was not aggravated by coughing, sneezing, chewing, or walking. Fever was associated with generalized body aches and severe myalgias, which were associated with bilateral lower limb weakness leading to bed-bound status. Weakness was gradual in onset, progressive, symmetrical, initially involving of distal muscles of lower limbs and gradually progressed to proximal muscles of upper limbs. Muscles of facial expression were spared. There were no complaints of difficulty in swallowing, speech difficulty, or double vision. There were no rashes on the face and eyes or body. There was no history of recent seafood or shellfish intake, tick bite, preceding upper respiratory tract infection, diarrhea, vomiting, sepsis, or multiorgan failure. The patient developed shortness of breath that started on the 3rd day of illness, which was gradual in onset, progressive, New York Heart Association (NYHA) class 4, not associated with cough, paroxysmal nocturnal dyspnea, orthopnea, or chest pain.

In the emergency department, the patient had the following vitals: Pulse 87 beats/min, B. P 124/87 mm Hg, temperature 102 degree Fahrenheit, and respiratory rate 32 breaths/min. SpO2 was 66% on room air. On general physical examination, the patient was lying on the bed, severely tachypneic, and in respiratory distress. The patient was cyanosed but there was no jaundice or clubbing. JVP was not visualized. No enlargement of the thyroid gland, lymph nodes, or edema was noted. On neurological examination, her GCS was E3V5M6-14/15, drowsy,

her pupils were bilaterally equal and reactive to light, and her plantars were bilateral down going. On motor examination, bilaterally normal bulk and lower tone, power was 3/5 in both upper limbs and 2/5 in both lower limbs. Her individual muscles strength could not be assessed in both proximal and distal muscles as the patient complained of severe pain during examination (grade 3 tenderness in all limb muscles). Deep tendon reflexes were Grade 2 in all four limbs, Cerebellar signs, cranial nerves, and eyes examination could not be assessed due to critical condition of the patient. On Respiratory Examination, the patient was in acute respiratory distress; there were nasal flaring, intercostal recession, and use of accessory muscles. Cardiovascular and abdominal exams were unremarkable.

We did a preliminary investigational analysis: blood complete picture showed an Hb of 14. 2 g/dl, TLC of 5700 per mm^3 and platelets of 79000 per mm^3. Dengue IgM antibody was positive by Elisa, and COVID-19 PCR was negative. Creatine phosphokinase (CPK) levels of 6212 mg/dl (highly raised). EMG and NCS were done, which were unremarkable. Muscle biopsy was in the plan but couldn't be done as the patient developed both type I respiratory failure followed by type II respiratory failure with respiratory acidosis with a PCO2 of 87 mm Hg signifying severe respiratory failure, and was transferred to ICU.

On arrival in ICU, the patient was intubated and put on a ventilator, CVC and arterial line were passed, as she was hemodynamically unstable and was unresponsive to fluid so she was put on vasopressors. ECG was done which showed sinus tachycardia, in-house echocardiography was done which showed right ventricular (RV) dilation and increased pulmonary artery pressure. Suspicion of pulmonary embolism was made. Pro-BNP and D-dimers were sent which were not suggestive of pulmonary embolism or cardiac failure. The patient developed acute renal failure with a creatinine of 5.2 mg/dl. The patient was treated for her acute kidney injury (AKI) but despite all this, the vasopressors requirement was increased until she collapsed. Cardiopulmonary resuscitation (CPR) was done with full ACLS protocol but she couldn't survive. The patient died due to multi-organ failure and respiratory failure.

Case 2:

A 27-years old male, known case of hypertension, presented in ER with the complaint of high-grade fever for 7 days, which was associated with generalized body aches and lower limb weakness. Weakness was acute in onset, progressive, and symmetrical, initially involving distal muscles of lower limbs and gradually progressing to proximal muscles of upper limbs. The patient was unable to bear weight or stand. Facial muscles were spared. There was no bulbar weakness or cranial nerve involvement. There were no other significant points in history.

On arrival in ER, the patient had the following vitals: Pulse 86 beats/min, B.P 154/71 mm Hg, temperature 102 degree Fahrenheit, respiratory rate 22 breaths/min. SpO2 was 94% on room air. On examination, a young male was lying comfortably on a bed having an I/V cannula and catheter in place. There was no cyanosis, jaundice, or clubbing. JVP was not visualized. No enlargement of the thyroid gland, lymph nodes, or edema was noted.

CNS examination:- GCS was E4V5M6 15/15, well oriented, pupils were bilaterally equal and reactive to light. Plantars were downgoing bilaterally. On motor examination, bulk normal, tone normal, power was 5/5 in upper extremity and 3/5 in the lower extremity and deep tendon reflexes were intact in all four limbs. Cerebellar signs, cranial nerves, and eyes examination couldn't be done due to the critical condition of the patient. All Sphincters were intact. Respiratory Examination, CVS, and abdominal examination were unremarkable.

Blood CP showed an Hb of 16.6 g/dl, with cytopenia with a white blood cell count of 1970 per mm3 and platelets of 15000 per mm3. Dengue NS 1 antigen was positive, liver enzymes were mildly elevated with normal serum bilirubin. Renal function tests, serum electrolytes, and coagulation profile were normal, and CPK and LDH were elevated at 2304 mg/dl and 630 U/l respectively. Nerve conduction studies did not show any evidence of polyneuropathy. The patient was then shifted to the medical ward and treated conservatively. The patient recovered within a week and was discharged. His CPK normalized to within normal limits over the following four weeks.

DISCUSSION

Dengue fever is a viral illness and viral myositis is a widely reported and well-known disease process and is seen much more commonly since COVID-19 pandemic.[4] However, dengue related myositis is rarely reported as compared to other causes.[5] Musculoskeletal manifestations of dengue fever include myalgias, polyarthralgia, rhabdomyolysis, and myositis with markedly elevated CPK. Acute severe myositis occurs with other viral infections as well such as HIV-1, human T lymphotropic virus 1 (HTLV-1), influenza, Coxsackie virus, and echoviruses. Myositis can be

complicated by rhabdomyolysis, and myoglobinuria and acute renal failure may lead to multiorgan failure and dysfunction. Dengue virus is rare but a potential cause of myositis. Myositis which occurs because of dengue can mainly be due to the production of inflammatory cytokines like tumor necrosis factor and interferon alpha.[6] Muscle biopsies in dengue patients show inflammatory infiltrates leasing to myonecrosis.[7]. A study done on dengue-related neuromuscular complications showed an age range from five years to 65 years and CPK was elevated in all with recovery in two weeks in all cases.[8]

In this report, we discussed two cases, In our first case 21 years old girl presented in the critical phase, and baseline investigations showed CPK levels of 6212 mg/dl, but she developed acute renal shutdown and pulmonary edema. In our second case a 27-year-old male, with baseline raised CPK levels, was managed with fluids and a short course of steroids and he recovered gradually with normal CPK levels within eight days of hospital stay. Studies have reported benefits of short course steroids in these cases.[9] This emphasizes early recognition of dengue complications as raised CPK levels is the most sensitive indicator of myositis. During the dengue epidemic, all patients presenting with fever, tenderness of muscles, and weakness should be screened for dengue viral infection and CPK as soon as possible to prevent deadly complications. [10,11]

CONCLUSION

In countries like Pakistan where there is a high prevalence and incidence of dengue fever especially during the epidemic months every year, doctors should be made aware of potential complications of dengue fever and keep a low threshold for screening of complications of myositis with CPK. Early diagnosis of such cases will help to provide adequate care.

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All the authors have approved the final version of the article, and agree to be accountable for all aspects of the work.