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Severe Septic Shock and Multiorgan Failure Associated with Chikungunya Virus Infection in an Adult

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Abstract **Case Report**

Chikungunya is often a mild condition, with little information on the prevalence of serious clinical consequences. We present the case of a 24-year-old man who developed chikungunya fever-related septic shock and multi-organ failure. Chikungunya can cause severe sepsis and septic shock.

Keyword: Chikungunya virus, Septic shock, Multiorgan failure.

I. INTRODUCTION

An arthropod-borne virus called the chikungunya virus is spread by Aedes mosquitoes. It is a benign febrile sickness accompanied by arthralgia that typically resolves on its own. Although chikungunya seldom results in death, the virus can significantly contribute to central nervous system disease in the event of a widespread outbreak, endangering the old and young [1]. Septic shock brought on by Chikungunya infection has only seldom been documented in adult literature [2-4]. We present a case of adult with diffuse alveolar hemorrhage, acute chikungunya sepsis, septic shock, and multiorgan failure.

II. CASE REPORT

A 24-year-old male patient presented with a high-grade continuous fever associated with chills, multiple small and large joint pains since the last 4 days, and itching followed by the development of a vesicular rash over the abdomen and extremities for 2 days. His vital signs were as follows: body temperature, 101 °F; blood pressure, 80/0 mmHg; pulse rate, 156 beats per minute; respiratory rate, 24 breaths per minute; and oxygen saturation of 78% on room air. He was admitted due to suspected septic shock. On examination, the patient was febrile, pale, oedematous, and irritable with cold extremities. General examination revealed multiple clear, fluid-filled vesicles that were fused and formed bullae over the abdomen, bilateral arms, and both lower limbs, as well as an erythematous patch over the palm and lower limbs. The cervical lymph nodes were not palpable. On auscultation, there were coarse crepitations and a muffled heart sound.

The patient had been given O2 through a nonrebreathing mask. He was given a standard saline rush as well as inotropic support (noradrenaline at 2 mcg/min). Injections of ceftriaxone and doxycycline were started. The central venous pressure and arterial pressure were 13 cm H2O and 80/50 mm Hg, respectively. Arterial blood gas (ABG) revealed pH 7.01, pCO2 35 mm Hg, pO2 169 mm Hg, HCO3 11 mmol/l, and lactate 4.9 mmol/l. A complete blood count revealed a hemoglobin count of 11.7 g/dL, a haematocrit of 33%, a white blood cell count of 15300 cells/mm3 (neutrophils, 82%; leukocytes, 9%; eosinophils, 0.1%; monocytes, 8%; basophils, 0.1%), and a platelet count of 38000 cells/mm3. Blood urea and creatinine were found to be 89 mg/dL and 1.2 mg/dL, respectively. Liver function testing revealed total proteins of 5 g/dL, albumin of 2.7 g/dL, globulin of 1.8 g/dL, total bilirubin of 3.6 mg/dL, and direct bilirubin of 2.7 mg/dL. Levels of aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP) were 79.7 U/L, 69.8 U/L, and 140 U/L, respectively. Laboratory results revealed a blood prothrombin time of 14 seconds, an international normalized ratio (INR) of 1.0, an activated partial thromboplastin time of 28 seconds, serum sodium Na+ 136 mmol/l, potassium K+ 4.8 mmol/l, chloride Cl- 103 mmol/l, C-reactive protein 21.6 mg/l, clotting time of 3

minutes, serum lactate of 32 mg/dl, serum procalcitonin 6. Serology for dengue non-structural protein 1 (NS1) antigen, immunoglobulin M (IgM), and immunoglobulin G (IgG) was negative; Leptospira IgM and IgG antibodies (by immunochromatographic assay) and Weil Felix serology (by latex agglutination) were negative.

On the second day of admission, he still had a high-grade fever and worsening hypotension. Ceftriaxone was then replaced with intravenous meropenem 1000 mg every 8 hours. The patient also developed hypoxemia while receiving oxygen via a nonrebreather mask at 10 L/minute. The patient continued to remain hemodynamically labile and required escalation of inotropic agents (dopamine was started at a rate of 10 mcg/kg/min). A chest X-ray revealed diffuse alveolar hemorrhage, so the patient was started on a high dose of methylprednisolone (125 mg daily). An electrocardiogram showed sinus tachycardia. However, the patient continued to deteriorate with labile hemodynamic status, falling blood pressure on ionotropic support, and worsening organ dysfunctions. Lactate was 17 mmol/l, TLC was 25 600/dl, N was 82%, platelet count was 15000/mm3, INR was 1.80, urea was 120 mg/dl, and creatinine was 2.1 mg/dl. On the third day of admission, he became severely hypotensive (BP of 60/30 mm Hg). 20% albumin boluses of 30 ml/kg were given, and dose escalation of ionotropic support was done for stabilization. The patient suffered progressive tachypnea, tachycardia, and hypoxemia (O2 saturation 85%) on oxygen support. So, he was intubated and put on volume-controlled ventilation. Blood and urine cultures revealed no signs of organism growth. Dengue RTPCR, Salmonella RTPCR, West Nile RTPCR, Plasmodium spp. RTPCR, Rickettsia RTPCR, Leptospira RTPCR, and SARS-CoV-2 RTPCR were negative. HIV-1 and -2, HCV, and HBsAg tests all came back negative.

Chikungunya RTPCR was positive in him. The patient was closely monitored and continued to receive invasive ventilation. After 3 days, the patient was extubated. Endotracheal tube tip culture revealed the presence of Coagulase- negative Staphylococcus, which was only sensitive to Teicoplanin. He was started on 400 mg injectable Teicoplanin 12 hours a day. No adverse or unanticipated events occurred. His urine output improved. The repeat chest X-ray became normal. The body's temperature returned to normal. The skin lesions on the abdomen and both thighs became crusted hemorrhagic vesicles 7 days later. The patient was discharged on the 12th day with a final diagnosis of Chikungunya sepsis with septic shock and multiorgan failure: hepatitis, acute renal failure, and diffuse alveolar hemorrhage.

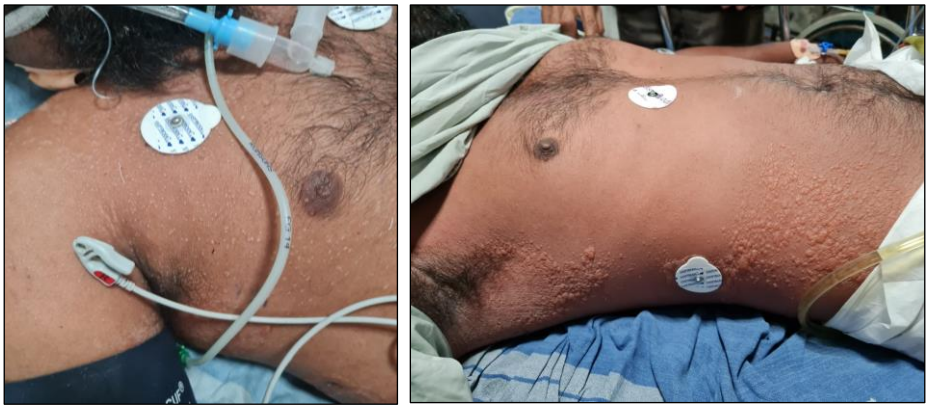

Figure 1: Multiple small clear vesicles over chest and flanks

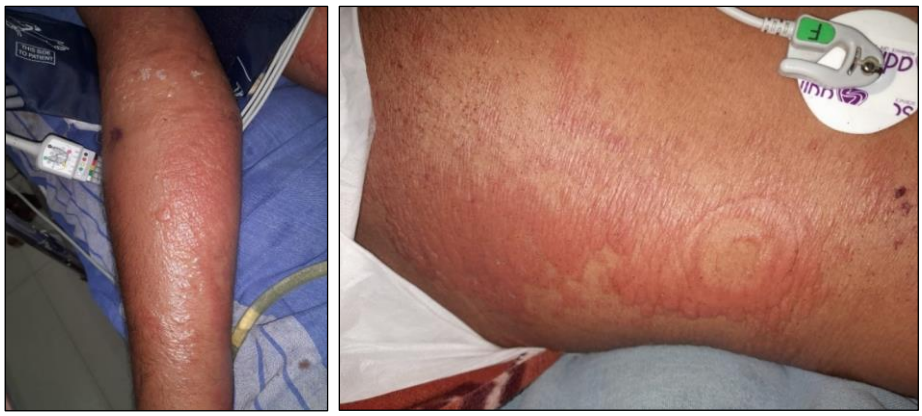

Figure 2: multiple clear fluid filled vesicles with hemorrhagic background which were fused and formed bullae over abdomen and right upper limb

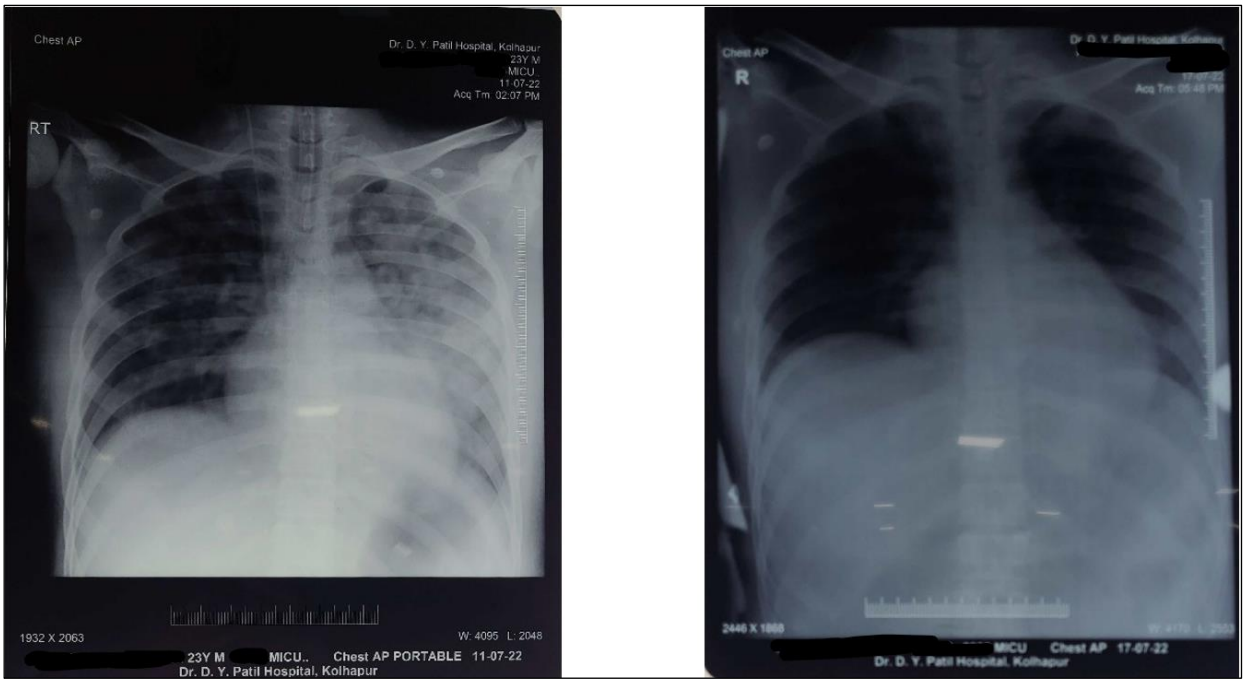

Figure 3: Chest Xray of patient suggestive of diffuse alveolar haemorrhage which is resolved on subsequent chest Xray

III. DISCUSSION

An arthropod-borne virus called the chikungunya virus is spread by Aedes mosquitoes. Infection with the chikungunya virus frequently causes fever, rash, and debilitating arthralgia. The location and appearance of the chikungunya virus' cutaneous presentation in adults vary, although the majority is characterized by a maculopapular rash, the development of bullae, and skin blistering. Vesicles most likely develop as a result of viral replication in the epidermis, which leads to localized necrosis, ballooning degeneration, or nuclear rupture, which is then followed by an inflammatory reaction and consequent leukocyte recruitment. Although uncommon, recorded neurological signs include retrobulbar neuritis-related blindness, encephalopathy, encephalitis, Guillain-Barre syndrome, focal seizures, febrile convulsions, and encephalopathy. Conjunctivitis, neuroretinitis, iridocyclitis, myocarditis, pericarditis, pneumonia, dry cough, nephritis, hepatitis, lymphadenopathy, and pancreatitis are other described abnormal clinical characteristics [6].

Serious chikungunya virus consequences include shock and sepsis. Our patient had an atypical presentation of a severe acute Chikungunya virus infection. The serious infections only manifested in 0.3% of symptomatic patients during the Le Reunion outbreak in 2005–2006, and 36% of atypical cases were deemed critical, with an estimated case fatality rate of 1 in 1000 [7]. Infections with the Chikungunya virus rarely result in septic shock reports. The three adult series just recently linked the chikungunya infection to severe sepsis and septic shock. Additionally, in these three series, chikungunya septic shock mortality was high (50–75%) [2-4]. Our patient presented with symptoms compatible with septic shock [7]. Laboratory tests indicated that the disease was caused by a chikungunya virus infection. Bacterial and dengue infections, both of which are major causes of tropical disorders, were ruled out. The differential diagnoses in this case were bacterial septic shock and dengue shock syndrome; however, other than chikungunya virus, we did not isolate any other organism as a probable cause of septic shock. This data implies that chikungunya infection in adults can only cause severe sepsis and septic shock in rare circumstances. The virus can cause sepsis and microvascular leakage either directly through cytopathic effects or indirectly through generating systemic cytokinemia and endothelial barrier dysregulation, resulting in increased vascular permeability and immune cell migration into organs [8].

IV. CONCLUSION

The patient in our case report had a positive chikungunya virus RT-PCR test result as well as severe sepsis, septic shock, and multiorgan failure early in the course of the disease. There was no other organism identified as a possible cause of sepsis in the patient. This discovery strongly shows that the chikungunya virus can cause severe sepsis, septic shock, and multiorgan failure in rare cases—an observation that had not previously been documented. More research is needed to uncover any background factors that may be linked to the onset of severe sepsis or septic shock.

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