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Case Report

An atypical case of fatal chikungunya infection in pregnancy

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ABSTRACT

Chikungunya viral infection is a mosquito borne illness which is known to have a limited course and complete recovery is seen in most of the patients. However the virus has been reported to have atypical manifestations and lethal complications have been reported in patients suffering from chikungunya infection. In the present outbreak of chikungunya virus in the national capital territory of Delhi we report a case of chikungunya fever in a pregnant female with no significant medical history. The patient developed thrombocytopenia, hepatic injury and disseminated intravascular coagulation and ultimately succumbed to the illness due to cardiovascular collapse.

Keywords: Chikungunya, Disseminated intravascular coagulation, Hepatic, Pregnancy, Thrombocytopenia

INTRODUCTION

Chikungunya virus is a mosquito borne alphavirus which is transmitted by the bite of Aedes aegypti and Aedes Albopictus. The virus causes an acute febrile illness and has been responsible for causing outbreaks in India since 2005. The most recent outbreak of the disease has been reported in Delhi in September 2016.[1]

Here we report a case of a previously healthy pregnant female without coexisting morbidity who succumbed to the disease with development of thrombocytopenia, hepatic injury and disseminated intravascular coagulation following chikungunya viral infection.

CASE REPORT

An unbooked female with 34 weeks period of gestation was admitted in the obstetric emergency of our hospital with the complaint of bleeding from mouth and gums and minimal bleeding per vaginum along with pain abdomen since 4 to 5 hours. Patient gave history of fever with multiple joint pains for the last 8 days. She did not consult a physician for the fever and there was no history of medication for the fever. There was no other history of cough, retro orbital pain, seizures, loss of consciousness or diarrhoea. Patient gave history of similar febrile illness with joint pains in her neighborhood. There was no other significant antenatal or past medical history. On examination patient was afebrile and icteric with heart rate of 84/minute and blood pressure of 110/68 mm of Hg. She had maculopapular lesions over the lower extremities. However no joint swelling could be appreciated.

The fetal heart rate was normal and on pervaginal examination the cervix was 3 centimetre dilated and 70% effaced. She was administered injection betamethasone and a complete hemogram, liver and renal function profile and coagulation profile were sent for analysis. She was administered injection ceftriaxone 2 gram intravenous after intradermal sensitivity testing. The

complete blood count showed hemoglobin of 8.3 g/dl, total leucocyte count of 5700/ microlitre, Differential leucocyte count (P 58, L 38, E 2, M2), platelet count of 30000/ microlitre. The blood urea was 50 mg/dl with a serum creatinine concentration of 1.3mg/dl.

The hepatic and coagulation profile reports were still awaited. In the meanwhile the labour progressed quickly. The patient was transfused 1 unit whole blood and 4 unit platelet concentrates and had a normal vaginal delivery of live male preterm twins who were admitted in the neonatal intensive care unit. She had postpartum haemorrhage of 1 litre which was managed with 1 unit packed red blood cells transfusion. The hepatic profile showed serum bilirubin of 4.1 mg/dl (total), 1.9 mg/dl (direct bilirubin), aspartate aminotransferase levels of 101.7 U/l and alanine aminotransferase levels of 48 U/l and alkaline phosphatase levels of 221 IU/L. The prothrombin time was 19.1seconds (Control 13 seconds, INR 1.5).

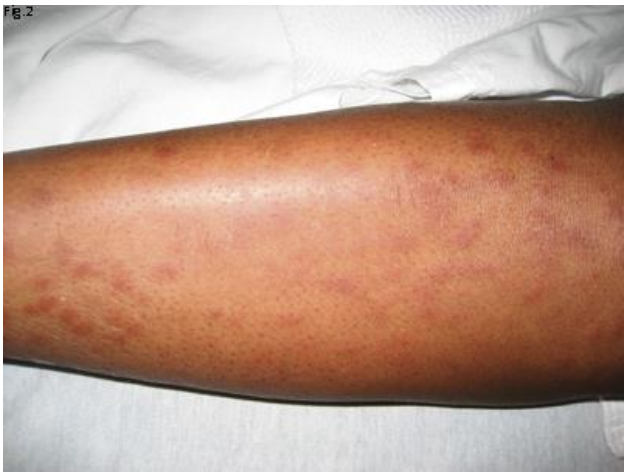

Figure 1: Maculopapular lesions over the lower extremity.

The patient was transfused 4 units of fresh frozen plasma. However her condition gradually deteriorated in the labour room. The heart rate increased to 120/minute and blood pressure decreased to 92/54 mm Hg. Patient developed breathlessness and bleeding from the mouth and she was transferred to the intensive care unit for further management.

An arterial blood gas analysis was suggestive of severe metabolic acidosis. Patient was intubated in the ICU and put on assist control mode of ventilation. She was infused intravenous fluids rapidly in view of hypotension but no improvement was seen and Noradrenaline infusion was started. Patient was administered injection sodium bicarbonate in view of severe metabolic acidosis. She developed cardiac decompensation and was put on triple inotropic support with Noradrenaline, Dopamine and Dobutamine. She continued to bleed from the nose and suctioning of the endotracheal tube revealed intrapulmonary bleeding. The coagulation profile was grossly deranged. Prothrombin time was 49.7 seconds (Control 13 seconds, INR 4.15) and activated partial thromboplastin time was 72.5 seconds (Control 34 seconds) and serum fibrinogen levels were 108.4mg/dl (Normal range 373-619 mg/dl in third trimester). The D dimer levels were 2110 ng/ml and platelet count was 60000/microlitre. Patient was transfused fresh frozen plasma, cryoprecipitate and platelets but did not show clinical improvement. Despite all the intensive management patient continued to bleed and developed significant derangement in the hepatic function tests (Aspartate aminotransferase 3710 U/l, Alanine aminotransferase 3690 U/l and alkaline phosphatase 421 IU/l). She developed acute renal failure with anuria and succumbed to the illness in the ICU. Blood, urine and endotracheal suction culture specimens failed to identify bacterial pathogens. IgM serology was positive for chikungunya virus and negative for dengue. The NS 1 antigen test and PCR test for dengue was negative. Malaria, typhoid, leptospirosis, meningococcemia and other common bacterial, viral parasitic diseases prevalent locally were also excluded.

DISCUSSION

Chikungunya is an arbovirus belonging to the togaviridae family. The virus was first isolated in Tanzania in 1952.[2] The first urban outbreaks of Chikungunya were reported from Bangkok in early 1960s and India from 1963 to 1973. After a long period of 32 years Chikungunya fever reemerged in India in 2005. Chikungunya fever is caused by the bite of an infected mosquito of the Aedes genus which is known to bite during the daylight hours. The fever starts abruptly and may last for several days upto 2 weeks. It is associated with severe symmetric polyarthralgias along with other constitutional symptoms and rash lasting for 1 to 7 days. The fever has been reported to show remission for 1-2 days after a gap of 4 to 10 days and may show a saddle type fever curve.[3,4] Cutaneous manifestations include a rash which is commonly maculopapular and involves the face and limbs.

The disease usually runs a limited course and most patients recover fully. However recently medical literature has reported Chikungunya virus to cause exacerbation of pre-existing systemic diseases resulting in multiorgan failure. Sam et al reported a case of chikungunya infection resulting in exacerbation of pre-existing heart disease and causing multi organ failure and death of a patient.[5] It has been reported to cause cardiac complications (myocarditis, pericarditis), neurological syndromes (Guillain Barre syndrome, meningitis, encephalitis) and retrobulbar neuritis.[6,7] In the Reunion island outbreak the incidence of atypical cases was 0.8% and the case fatality rate was 10.7%.[7] Chikungunya virus infection can cause severe clinical manifestations, even in young patients with no significant medical history. It is strongly suspected to have myocardial, neurologic and hepatic tropism with severe complications.[8] Chua et al

reported a fatal case of chikungunya fever resulting in liver involvement and multiorgan failure.[9] Torres et al reported a case series of 4 patients with life threatening chikungunya infection causing rapid clinical deterioration resulting in shock, multiorgan failure and death of the patients.[10] Unlike dengue fever, coagulation abnormalities are rare in chikungunya fever. However disseminated intravascular coagulation has been reported in chikungunya viral infection in neonates.[11] In present case the patient had no significant past medical history and the antenatal course had been uneventful till the onset of the fever. Present patient developed thrombocytopenia, hepatic derangement, disseminated intravascular coagulation and multiple organ failure and succumbed to the infection. Dengue haemorrhagic fever which is very common in the Indian subcontinent was ruled out by repeated negative serology and negative PCR test. Pregnant women with chikungunya infection can pass the virus to the fetus. The Reunion South Hospital groups have reported 84 pregnant women with Chikungunya infection. In all of these cases the chikungunya infection had been distant from the delivery time. In 74 pregnancies, the newborns were aymptomatic. However in ten pregnancies the neonates had severe chikungunya infection after birth and required intensive care support. Four neonates developed meningoencephalitis and three developed intravascular coagulation. One infant developed severe thrombocytopenia and intracranial haemorrhage.[12]

Lenglet et al reported findings on 160 pregnant women with chikungunya infection. They observed 3 miscarriages in women with chikungunya infection before 22 weeks of gestation. In 33 pregnancies with positive viremia at delivery, chikungunya infection was reported in 16 neonates. However in our case both the twins tested negative for chikungunya infection.

To the best of our knowledge this is the first case which reports mortality due to chikungunya infection in a pregnant woman in the national capital region of Delhi. In our country where repeated chikungunya epidemics have occurred since 2005, the physician should be well aware of the atypical manifestations of the disease which would help in better management of these patients.

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