



DUAL HORROR IN PREGNANCY : SICKLE CELL DISEASE WITH DENGUE FEVER.

Obstetrics & Gynaecology

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ABSTRACT

Seldom have reports of sickle cell disease cases associated with dengue during pregnancy been made. One of the most prevalent genetic illnesses that are inherited, particularly in some parts of India, is sickle cell disease (1). Sickle cell disease varies in clinical severity, particularly during pregnancy. It could potentially result in major problems and have a detrimental impact on the results for both the mother and the fetus. Dengue is a frequent illness in tropical nations. The dengue virus, which is spread by the *Aedes aegypti* mosquito, has four serotypes: 1, 2, 3 and 4. A 24-year-old primigravida at 36 weeks of gestation, a known sickle cell disease patient, reported to the emergency room with a history of several blood transfusions and fever lasting for four days with chills and body aches. After sending in her fever profile, the patient's diagnosis was confirmed as Dengue. She received conservative treatment for pyrexia followed which she went into spontaneous preterm labour and delivered. Pregnancy-related pathophysiological changes such as elevated blood volume and blood viscosity, and hypercoagulability, combined with dengue fever complications in a sickle cell disease patient leads to higher rates of morbidity and mortality.

KEYWORDS

INTRODUCTION

Sickle cell carriers make up 1 to 40% of the population in India's various tribal groups [2]. In the past, pregnancies were linked to higher rates of fetal/maternal death, and patients with Sickle cell disease (SCD) received subpar care. These days, overall illness outcomes and patient survival have improved, and rates of maternal and newborn death have significantly decreased because of advanced screening tools, maternal screening and preventive interventions including immunization and antibiotic prophylaxis since birth[2]. All the advancements made, however, have not eliminated the association between dengue and increased clinical and obstetric problems in SCD pregnant women as compared to the general population. The physiological adaptations that occur in the circulatory, hematologic, renal, and pulmonary systems during pregnancy can overburden organs that already have chronic injuries secondary to SCD, increasing the rate of obstetric complications like anemia and preeclampsia, worsening of vaso-occlusive crisis, and acute chest syndromes. Microvascular damage and decreased uteroplacental circulation in these mothers leads to an exaggerated rate of spontaneous abortions and stillbirths[3]. Dengue fever is a public health issue in the subtropical and tropical regions [4]. Its incidence has increased dramatically around the world in recent decades [5]. The number of dengue cases per year is estimated at 390 million, of which 96 million are symptomatic [6]. There is also evidence that suggest a higher percentage of severe dengue infections in pregnant women than non-pregnant women. Many pregnant women with dengue infections may progress to develop Dengue Shock Syndrome (DSS), and the mortality rate triples in these scenarios[7]. According to the WHO guidelines on dengue fever, pregnant woman with sickle cell disease is considered to be a risk factor for development of severe dengue fever[8].

CASE

A 24 year old primigravida at 36 weeks of gestation, a known case of Sickle cell disease with history of multiple blood transfusions since childhood presented to the emergency department with the fever for 4 days, which is associated with chills and rigor. It was also associated with retroorbital pain, epigastric pain and generalised body ache. She had regular antenatal visits throughout her pregnancy, and there were no complaints. On admission, her body temperature was 102°F, Blood pressure was 110/80 mmHg, Pulse rate was 110/min, Respiratory rate was 24/min and SpO2 was 99% on room air. She had signs of mild dehydration. She had normal breath sounds. Petechiae sized 1–2 mm in diameter were noted around her face, forearms and both pretibial areas. On per abdominal examination it was noted that her liver and spleen was mildly enlarged and tenderness was observed. Her fundal height was around 36 weeks of gestation, uterus was relaxed, with a fetal heart rate of 164 beats/min. On per vaginal examination her cervix was soft, anterior, 50% effaced, 2 centimetres dilated and her station was -2 with no discharge seen (Bishop's score - 7). The joints of the patient were examined to rule out sickle cell crisis, all the joints of the upper and lower limbs along with hip joint were examined and no signs of joint tenderness were elicited. She had vague generalised body pains. Blood

investigations like Complete blood count, liver function test, renal function test, urine routine and microscopy, urine culture, high vaginal swab, Coagulation profile and fever profile of the patient was sent. Laboratory analysis on admission gave the following results: Haemoglobin 8.3 gm%, haematocrit 35% ,total leucocyte count of 13000/cumm ; Platelet count was 70,000 /cumm, she tested positive for dengue NS1 Ag, Ig M, total bilirubin was 1.4mg/dL, LDH was 300U/L, the urine culture and high vaginal swab had no growth of bacteria.

The patient was shifted to high dependency unit, she was immediately started on intravenous fluid replacement therapy to hydrate her along with a prophylactic dose of intravenous antibiotic (Ceftriaxone 1 gm twice a day for 5 days), intravenous Acetaminophen 1000mg twice a day was started and Dexamethasone 4 mg twice a day was given for 2 days. She was closely monitored, her vitals were stable and she did not show any signs of deteriorating. Twenty-four hours later, the epigastric pain was relieved and the vital signs were within normal limits. The haemoglobin 8.3 gm%, haematocrit was 30% and the platelet count was 60,000 /mm³. Transfusion of packed red cells was done under strict monitoring. The patient had spontaneous rupture of membranes, due to which the patient went into preterm labour. The labour was actively managed. The patient was positioned in left recumbent propped up position and started on 2 litres of oxygen prophylactically throughout the labour. A 2.4 kilogram female child was born, who cried immediately after birth. There was around 350 ml of blood lost during labour. APGAR scores of the baby were 8 and 9 at 1 and 5 minutes of life. The baby was attended by neonatologist. No abnormality was detected in the new-born. The patient was monitored continuously for 24 hours, her bleeding per vaginam was checked every 2 hourly. platelet concentrate was kept ready but not transfused as there was no severe bleeding. On the third day of admission, the patient gradually recovered. The haemoglobin was 9.2 gm%, haematocrit was 31% with platelet count 85,000 /cumm. She was discharged on the seventh day when her haemoglobin was 9.2 gm%, haematocrit was 31% with platelet count 1,00,000 /cumm with no fever spikes, signs of sickle cell crisis and after contraceptive counselling. On 1 week follow-up, she was healthy with no episodes of fever, well involuted uterus and haemoglobin 9.2 gm%, haematocrit was 32% with platelet count 1,74,000 /cumm.

DISCUSSION

The dengue virus, which is spread by the *Aedes aegypti* mosquito, has four serotypes: 1, 2, 3, and 4. It is a significant issue for public health in tropical nations. The World Health Organization has revised its classification of dengue (WHO). Dengue fever (DF), dengue hemorrhagic fever (DHF), and undifferentiated fever were the three categories used to categorize symptoms of dengue in 1997. DHF was divided into four grades based on its severity. DSS applied to Grades III and IV. A person must have a fever, hemorrhagic manifestations, thrombocytopenia, and signs of plasma leakage in order to be diagnosed with DHF [9]. Patients who develop DSS also exhibit signs

of shock, hypotension, a further constriction of the pulse pressure, and a rise in hematocrit (HCT) levels, in addition to the symptoms observed in DHF [10,11]. The adoption of new criteria in 2009 was prompted by the challenge of meeting the stringent requirements of DHF.

The revised criterion divided symptomatic dengue into three categories: severe dengue (SD), dengue with warning symptoms (DWS), and dengue without warning signs (DWS). Abdominal pain and tenderness, continuous vomiting, mucosal bleeding, tiredness, restlessness, liver enlargement greater than 2 cm, and test results showing an elevated HCT value together with an abrupt reduction in platelet count are all warning signals. When severe bleeding, serious organ involvement, or severe plasma leakage resulting in shock or respiratory difficulty are present, SD is diagnosed [12].

Due to their vulnerability and increased worry of the outcome in pregnancy, pregnant women are one of the most affected groups by this virus [13,14]. Pregnancy-related dengue fever increases the risk of both more serious infections and mother-to-child transmission of dengue [15].

Among the various genotypes of Sickle Hemoglobin C disease (SC) are Sickle beta plus thalassemia (S β +Thal), sickle beta zero thalassemia (S β 0 thal), sickle cell anemia with alpha thalassemia (SS α thal) and sickle cell anemia with high fetal hemoglobin (SS+HbF). A homozygous mutation (hemoglobin S) is the cause of sickle cell anemia (SCA), which manifests as a chronic anemia with painful episodes. The main defect that causes these occurrences is poor microcirculation caused by erythrocyte sickling.

The patients in this facility are susceptible to infections and all types of maternal problems, including hemolytic and thrombotic sickling crisis.

Dengue fever can manifest in persons with sickle cell disease (SCD) in an unusual and severe way. When these two disorders coexist, the clinical picture may become more complex. For instance, the fever and inflammatory response linked to dengue infection might cause or worsen the vaso-occlusive crises that are frequently observed in SCD. Furthermore, the dengue virus has the potential to exacerbate hemolysis in patients with sickle cell disease (SCD), which increases the risk of severe anemia and blood transfusions. This syndrome is associated with increased maternal and fetal morbidity and mortality (16).

The possibility of worsening clinical consequences makes the relationship between dengue fever and SCD very worrisome. The pathophysiology of dengue fever may exacerbate the infections and consequences that already pose a risk to mothers with sickle cell disease (SCD).

It is imperative to recognize and promptly address potential consequences, including acute chest syndrome, multi-organ failure, and severe bleeding. Multidisciplinary management and intense care may be necessary for certain problems.

Depending on the severity of the underlying sickle cell disease as well as the dengue infection, people with SCD can have a guarded prognosis for dengue fever. Results can be enhanced by aggressively managing problems and recognizing them early. Studies indicate that the interaction between the chronic inflammatory state in SCD and the inflammatory response in dengue may result in more severe disease presentations, requiring a high index of suspicion and early intervention.

There have been examples of dengue virus vertical transmission, however infrequent [17, 18].

CONCLUSION

This instance emphasizes how critical it is for sickle cell disease patients to identify and treat dengue fever in pregnant women as soon as possible with vigorous care. Optimizing patient outcomes requires multidisciplinary care from hematologists, infectious disease specialists, and critical care teams.

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