CASE REPORT

Giant vulva oedema in pregnancy precipitated by anaemic heart failure: a case report

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ABSTRACT

We report a case of vulva oedema in a 19-year old multigravida at 30 weeks gestational age with severe anaemia complicated by heart failure. The vulva oedema appeared and rapidly increased in size as features of anaemic heart failure set in. Other causes of vulva oedema were ruled out. She was transfused with six units of packed red cells and the oedema subsided by the 10th day. She developed intrauterine fetal death while on in-patient admission, and subsequently, had spontaneous labour and delivery of a macerated stillborn male neonate. The aim of this report is to alert clinicians that severe anaemia with vulva oedema may be a poor prognostic index associated with intrauterine foetal death.

INTRODUCTION

Vulva oedema resulting from anaemia is not a common occurrence. Anaemia during pregnancy is a major public health problem, especially in developing countries.[1] It affects 41.8% of pregnant women globally, with the highest prevalence in Africa.[2] Anaemia is a common cause of maternal and foetal morbidity and mortality. Maternal anaemia is defined as haemoglobin (Hb) <11g/dl and is classified as mild/moderate (7-10.9g/dl) or severe (<7g/dl).[3,4] The cardinal clinical features are malaise, weakness and easy fatiguability. In severe forms, anarsaca, orthopnoea, paroxysmal nocturnal dyspnoea and congestive heart failure may occur.

There is conflicting literature regarding the association between anaemia and vulva oedema. But, severe anaemia may cause a high output cardiac failure resulting in congestion of tissues and organs. Other causes of vulva oedema are pre-eclampsia,

infections, neoplasms, congenital anomalies, trauma, inflammatory diseases, venous stasis, nephritic syndrome and tocolytic agents.[5,6] Vulva oedema should be considered as an important marker for seropositive syphilis during pregnancy.[7]

In patients with oedema caused by chronic severe anaemia there is retention of salt and water, reduction of renal blood flow and glomerular filtration rate and neurohormonal activation. It is suggested that the low concentration of haemoglobin in such patients causes a reduced inhibition of basal endothelium-derived relaxing factor activity and leads to generalized vasodilatation. The consequent low blood pressure may be the stimulus for neurohormonal activation and salt and water retention.[8]

CASE REPORT

We present the case of an unbooked 19-year old gravid 2 para 1^{+0}, 1 alive, who was unsure of her dates but said she was 7months pregnant. She presented at the gynaecological emergency clinic with a history of dyspnoea on exertion of 2weeks, and rapid onset progressive vulva oedema of 5days' duration. She developed progressive dyspnoea initially on exertion and then, later at rest. There was associated cough, headache, dizziness weakness and malaise but no history of fever. There was associated history of generalized body swelling, but no jaundice and gastrointestinal or urinary symptom. There was no antecedent history of trauma, insect bite or rashes on the vulva. She had no prior history of vaginal discharge or bleeding per vaginam.

On admission, she was acutely ill-looking and severely pale. Her pulse rate was 115/minute and blood pressure was 140/90mmHg and her heart sounds were I, II and III. The respiratory rate was 32/minute with vesicular breath sounds and bi-basal crepitations. The abdomen was uniformly enlarged and there was tender hepatomegaly, but the spleen and kidneys were within normal limits. Abdominal examination revealed a symphysio-fundal height of 28cm. The foetal heart sounds were 142beats per minute. Pelvic examination revealed a huge vulva oedema hanging like a bag of worms, measuring 18x16x 12cm.

The result of full blood count showed Hb of 4g/dl with features of both iron and folic acid deficiency anaemiae. Serum electrolytes, urea and creatinine were normal and retroviral screening was non-reactive. Her genotype was AA and blood group was O^{+}. Liver function test result was within normal limits and Venereal Disease Research Laboratory (VDRL) test was negative. Malaria parasites was positive (+) and urinalysis was within normal limits. Urine microscopy, culture and sensitivity yielded no growth, and stool microscopy, culture and sensitivity were normal. Chest x-ray showed normal cardiac index with features of pulmonary oedema, just like electrocardiography and echocardiography were also normal. Bed side ultrasound scan revealed a live foetus, at 30 weeks gestation.

She was nursed in cardiac position with oxygen by face mask. She was transfused with 6units of packed red cells over 3 days and was counseled on conservative management of the vulva oedema. She was encouraged to have immersion of the vulva in antiseptic solution for 10-15minutes, twice daily. She was commenced on anti-malarials, prophylactic antibiotics, analgesics and haematinics. However, she suddenly stopped perceiving fetal movement on the third day of admission and a bed side ultrasound scan confirmed intrauterine foetal demise.

Her post transfusion packed cell volume on the 7th day was 30%. The vulva oedema resolved on the 10th day. While she was being worked up for cervical ripening and induction of labour she went into spontaneous labour and expelled a macerated male fetus on the 12th day of admission. Labour lasted 6hours and third stage was managed actively.

During the postpartum period, the vulva was inspected for any tear or laceration. She was maintained on oral antibiotics, haematinics and regular *Sitz* bath.

Figure 1. Day 1

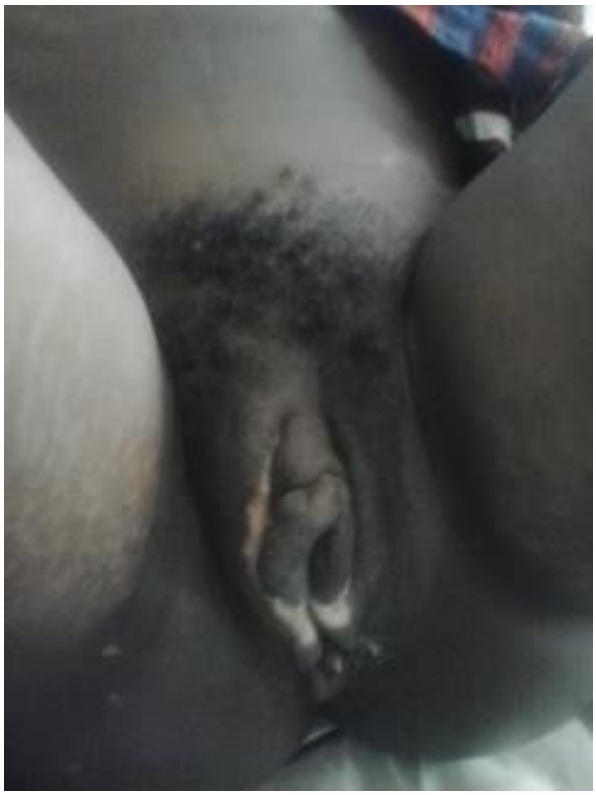

Figure 2. *Day 14*

DISCUSSION

The clinical presentation of patients with anaemia varies appreciably depending on the severity of the anaemia. Anaemia during pregnancy, especially severe anaemia, is associated with increased maternal morbidity and mortality and contributes to 20% of the maternal mortality in Africa.[1,4,9] To our knowledge several reported cases of vulva oedema were associated with pre-eclampsia.[10,11,12] It is thought that women with severe anaemia had a 3.6 times higher risk of pre-eclampsia than women who did not.[3] Though our case had no features of pre-eclampsia, an evolving disease could not be completely ruled out.

Congestive cardiac failure may follow chronic severe anaemia resulting in fluid retention and vulva oedema. The reason why it occurs in selected patients is still unclear. The anaemia in our patient is likely to be multifactorial with nutritional and infective factors being implicated. A suggested pathogenesis for the development of vulva oedema in pregnancy is that renin-angiotensin activation causes fluid retention, which results in oedema, especially in the extremities. The vulva could be affected by the pressure-volume disturbances since it has a thin epithelium and loose connective tissues.[13,14]

Hence, we postulate that vulva oedema in patients with severe anaemia complicated by cardiac failure develops from the common mechanism of hypoalbuminaemia resulting in reduced oncotic pressure and increased capillary permeability, observed in anaemic heart failure. In our case, we presume that, the presence of hypoalbuminaemia, severe anaemia, heart failure and vulva oedema correlated well with the above phenomenon.

On account of this, the correction of the anaemia by transfusion of packed red cells coupled with bed rest resulted in the resolution of the vulva oedema before labour onset. This provided the chance for vaginal delivery in this patient and obviated the need for caesarean section which can be the mode of delivery when the vulva oedema does not resolve. The patient did not sustain any perineal injury during the delivery as the perineum was properly guarded.

CONCLUSION

Vulva oedema may be rapidly progressive in nature with associated pain and marked discomfort particularly if it occurs during pregnancy. Huge vulva oedema may result in ulceration and secondary infection. Identification and treatment of the associated factors and likely causative factor is vital in the successful management of this condition.

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