International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Ehigiegba OE et al. Int J Reprod Contracept Obstet Gynecol. 2024 Aug;13(8):2133-2136
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20242084

Case Report

Successful management of severe anaemia in pregnancy: a rare case report

Oduware E. Ehigiegba[1], Benedict Nwogoh[2], Reuben O. Iweka[1]*

[1]Department of Obstetrics and Gynaecology, University of Benin Teaching Hospital (UBTH), Benin City, Nigeria
[2]Department of Haematology, University of Benin Teaching Hospital (UBTH), Benin City, Nigeria

Received: 31 May 2024
Accepted: 02 July 2024

***Correspondence:**
Dr. Reuben O. Iweka,
E-mail: iwekar@gmail.com

ABSTRACT

Severe anaemia in pregnancy is associated with high maternal and perinatal morbidity and mortality. The life-threatening complications associated with severe anaemia in pregnancy were averted in this case due to the astute management and multidisciplinary approach employed involving the hematologist, dietician, and obstetrician. We report a rare case of a 33-year-old pregnant woman with three living children who presented with severe anaemia, characterized by a hematocrit of 8% (haemoglobin of 2.5 g/dl) in her early second trimester of pregnancy. The peripheral blood smear revealed mixed nutritional deficiency anemia, while the bone marrow aspirate showed megaloblastic changes. She received blood transfusions, haematinics, vitamin supplements, and adequate nutrition. Following discharge, she attended weekly antenatal clinics with antenatal fetal surveillance until term. At term, she was induced and had vaginal delivery of a healthy baby. She was discharged healthy with her baby and had bilateral tubal ligation at 6 weeks postpartum for a completed family size. This case shows that the morbidities and mortalities associated with severe anaemia in pregnancy are preventable with good multidisciplinary clinical management. The severity of this anemia, her remarkable recovery, and successful outcome make this a rare and noteworthy case report.

Keywords: Severe anaemia, Pregnancy, Nutritional deficiency anaemia, Megaloblastic changes

INTRODUCTION

Anaemia in pregnancy is a global health challenge and contributes to maternal and perinatal morbidity and possible mortality.[1] According to the World Health Organization (WHO) anemia in pregnancy is a decrease in the oxygen-carrying capacity of the blood due to reduced circulating erythrocytes or in the concentration of hemoglobin lower than 11 g/dl.[2] Anaemia is further classified into mild (hemoglobin level is between 10.0 to 10.9 mg/dl), moderate (the hemoglobin level is between 7.0 to 9.9 mg/dl) and severe (hemoglobin level <7.0).[3]

A study by the Nigerian Demographic and Health Survey (NDHS) 2018 showed the incidence of 2.3%, 33%, and 25.8% for severe, moderate, and mild anaemia in pregnancy.[4] A study at the University of Benin Teaching Hospital by Orhue et al showed the prevalence of severe anaemia in pregnancy was 2.8% in our surrounding.[5] This shows that severe anaemia is uncommon in our environment.

Anaemia is an indicator of both poor health and poor nutritional health. It has many aetiological factors of which almost 50% of all anemia in pregnancy can be attributed to iron deficiency (ID), the other causes are due to important micronutrient deficiencies such as vitamin B9 (folate), vitamin B12, or vitamin A deficiency, vitamin C and vitamin E.[1,6-9] Other causes include chronic inflammation, infectious diseases such as malaria, HIV/AIDS, hookworm infections, and urinary schistosomiasis.[1,6-8] Genetic disorders (heamoglobinopathies) such as sickle cell anaemia, thalassemia and malnutrition especially where

diets are based mostly on staple foods with little meat intake.[1,6-8]

The maternal complications of anaemia in pregnancy include postpartum haemorrhage, preterm delivery and anaemic heart failure.[7,8] Anaemia in pregnancy may lead to perinatal complications such as birth asphyxia foetal growth restriction, intrauterine foetal demise, and neonatal death.[1,2,7]

Although cases of severe anaemia in pregnancy are few in our environment, the health burden is quite significant. Based on the severity of the index case and its' peculiarity, we are reporting it.

CASE REPORT

We report a 33-year-old with 3 living children who was 19 weeks pregnant and presented following referral with complaints of fever, vomiting and generalized body weakness all of 2 days duration. There was no abdominal pain, bleeding per vaginam or loss of consciousness.

She had a similar symptom in her last pregnancy for which she was managed for moderate anaemia in pregnancy and transfused with 2 units of blood for PCV of 24% for symptomatic anaemia.

Due to her unemployment status, she was not properly fed with adequate balanced diet as her husband was of the low socioeconomic class. She did sleep under a mosquito-treated net but did not take any intermittent preventive treatment for malaria during pregnancy. She was not a known hypertensive, diabetic, or renal disease patient. All previous deliveries were vaginal deliveries and no previous surgery.

The index pregnancy was desired and spontaneously achieved, she was yet to do any obstetric ultrasound scan and book the pregnancy in any facility due to financial constraints. Her pregnancy was uneventful until about 2 days before presentation when she developed the above complaint.

At presentation, she was a young woman, lethargic, in obvious respiratory distress, febrile (38.6oc), markedly pale (3++), anicteric, cyanosed, dehydrated, no pedal edema. She was restless with a Glasgow coma score of 13 (E4, V4, M5). The urine was amber-colored. She was dyspnoeic with oxygen saturation of 88% in room air, respiratory rate of 26 cpm and breath sound was vesicular. Her pulse rate was 120bpm (low volume and thread), her blood pressure was 110/70 mmHg, and her heart sounds were S1 and S2 only. The abdomen was uniformly enlarged, about 20 weeks size, there was no abdominal tenderness, no bleeding per vaginam, and the cervical os was closed. An obstetric scan at presentation showed a single viable intrauterine fetus with normal cardiac activity, no fetal anomaly and the placenta was not low lying.

Her malaria parasite blood film showed 2++, while the full blood count done showed reduction in all cell lines (pancytopenia). The white blood cell count was 2,900/ul (3,000-11,000/µl), heamoglbin was 2.5 g/dl (normal is 11 g/dl and above), the haematocrit was 8% (30-45%), platelet count was 73,000/µl (150,000-400,000/µl). Malaria parasite blood film done was negative, the HIV, hepatitis B and C screening were also negative. Her genotype is AA, direct comb test was negative and her blood group is O Rhesus D positive. The electrolyte, urea and creatinine test and liver function test were normal. Stool microscopy did not reveal any ova of parasite. She was given antipyretics, and immediately commenced on blood transfusion with 3units of fresh whole blood initially.

A haematologist reviewed her, and peripheral blood film (Figure 1) showed pancytopenia, dimorphic red blood cells, neutrophil hyper segmentation, and bone marrow aspirate (Figure 2) showed megaloblastic anaemia. She was commenced on high-dose supplemental iron, vitamin C, folic acid, vitamin B complex, tabs cyanocobalamin, and pyridoxine. The dieticians were invited and placed her on a protein diet with fruits and vegetables supplied by the hospital chef. She subsequently received 5 more transfusions making a total of 8 units, after which the transfusion was stopped.

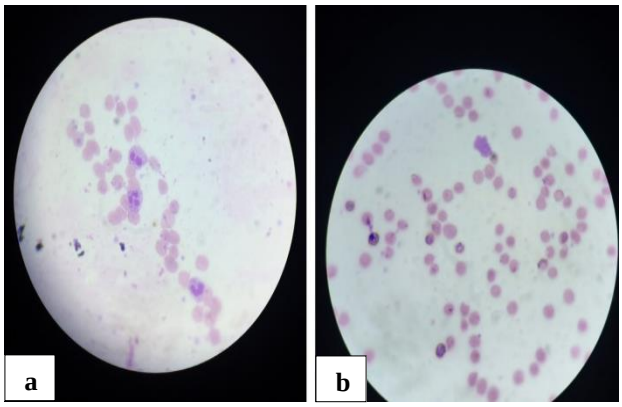

Figure 1 (a and b): Peripheral blood film showing pancytopenia, dimorphic red blood cells and neutrophil hypersegmentation.

Her packed cell volume remained at 24% despite transfusion with 8 units of blood. Further blood transfusion was withheld while she was monitored closely on haematinics and an adequate high-protein diet. After about 6weeks of admission, her packed cell volume rose to 27%, she was asymptomatic and was discharged to the antenatal clinic, where she was seen weekly. At each clinic, the maternal vital signs and clinical state were assessed to be good. She also had antenatal foetal surveillance with weekly biophysical profile and umbilical artery Doppler every fortnight from 28 weeks till term.

At term, her packed cell volume (PCV) was 28%, she was admitted for induction of labour, and transfused with 2

units of blood which raised the packed cell volume to 33%. She subsequently had induction of labour at 37 weeks and 2 days gestation and had a vaginal delivery of a live female 3.5 kg neonate with an APGAR score of 8 and 9 in the first and fifth minutes. The neonate was certified stable by paediatrician and did not require intensive care.

There was no complication following delivery and her postpartum was uneventful. Her PCV postpartum was 30%. She was discharged after 3 days on admission with haematinics and was referred to the postnatal and hematology clinics.

She was seen every fortnight in the postnatal clinic, her PCV ranged between 30 to 33% till 6 weeks postpartum when she was family planning clinic where she had interval bilateral tubal ligation on patients request for completed family size.

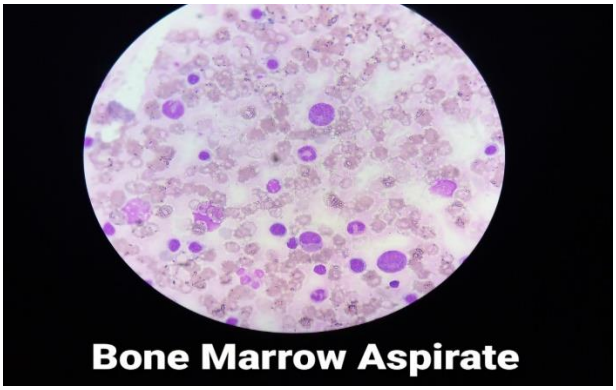

Figure 2: Bone marrow aspirate showing megaloblastic anaemia.

DISCUSSION

Severe anaemia in pregnancy remains a public health challenge, especially in developing countries. This report is particularly of interest due to the severity of the anaemia, her recovery and the outcome despite the poor resource setting.

Most cases of anemia in pregnancy are a result of iron deficiency (ID), the other causes may include micronutrient deficiencies such as vitamin B9 (folate), vitamin B12, or vitamin A deficiency, vitamin C and vitamin E.[1,6-9] Other causes include chronic inflammation, infectious diseases such as malaria, HIV/AIDS, hookworm infections, and urinary schistosomiasis; and genetic disorders (heamoglobinopathies) such as sickle cell anaemia, thalassemia and malnutrition.[1,6-8] In this patient, poor nutrition, severe malaria parasitaemia and micronutrient deficiencies such as vitamin B12 and iron played significant roles in the aetiopathogenesis. Poverty occasioned by her unemployment status and her spouse's low socioeconomic class was the major predisposing factor. Also, her low level of knowledge of anaemia in pregnancy, despite a previous history of anaemia in pregnancy which necessitates blood transfusion and was yet to book for antenatal care at 19 weeks gestation was contributory as she probably felt it was a waste of the family's meager resources.

Her peripheral blood smear (Figure 1) and bone marrow (Figure 2) findings were consistent with mixed nutritional deficiency predominantly megaloblastic anaemia. Megaloblastic anemia occurs due to impaired DNA synthesis during the process of erythropoiesis due to deficiency of vitamin B12 and or folate.[12] Due to this halt, the cells are unable to progress into the mitotic (M) phase of the cell cycle when DNA synthesis is inhibited.[12] The clinical manifestation of this disorder is macrocytosis and megaloblastosis. Megaloblastic anemia is a kind of nutritional anemia that ordinarily responds to B12 and or folate therapy however in some instances as in the index case may not be responsive.[12] As in this case, previous reports have highlighted megaloblastic anaemia as a cause of severe anaemia.[12,13] However, the packed cell volumes of these 2 reports were not as low as 8%.

The maternal and foetal complications of severe anaemia in anaemic heart failure, preterm delivery, postpartum haemorrhage, foetal growth restriction, foetal demise, birth asphyxia, and neonatal death.[1,2,7-9] These were not observed in this case due to astute care instituted in managing this patient.

The multidisciplinary approach employed in this case involving the haemaotologist, dietician and obstetrician was key to the successful outcome recorded. The patient was transfused with blood which was closely supervised by the haematologist, she received supplements such as iron supplements, vitamin B12, folate (B9), vitamin C and pyridoxine. Although, vitamin B12, folate and iron levels could not be accessed due to cost (Financial constraint), these supplements were instrumental in raising her packed cell volume to an acceptable state.

During this period, pregnancy was monitored closely to ensure that sustenance of pregnancy was not at the detriment of the mother. This was achieved through close foeto-maternal monitoring by checking maternal vital signs closely and examination of her clinical state. Also, the use of obstetric devices like hand-held doppler, cardiotocography, biophysical profile, and umbilical artery doppler when appropriate. All these were done to ensure that the well-being of the mother and her baby was good at all times.

The decision of how to deliver a woman with anaemia in pregnancy has been the subject of debate due to complications of delivery such as postpartum haemorrhage. A systematic review by Bunch et al showed that there was weak evidence that induction/ augmentation of labour for patients with severe anaemia in pregnancy could increase the risk of postpartum haemorrhage.[14] This was not recorded in our case because her packed cell volume and other haemostatic parameters were optimized

before delivery. The process of labour is actively managed culminating in the delivery of a healthy baby by a healthy mother.

CONCLUSION

Severe anaemia in pregnancy is a significant burden due to its poor prognosis. To avoid this, adequate nutrition with appropriate vitamin supplementation in pregnancy is essential. The management of this case illustrates the importance of good obstetric care, proper treatment and the role of multidisciplinary approach which was instrumental in the successful outcome.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Ehigiegba OE, Nwogoh B, Iweka RO. Successful management of severe anaemia in pregnancy: a rare case report. Int J Reprod Contracept Obstet Gynecol 2024;13:2133-6.