ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24JUN378

Severe Pre-Eclamptia Resulting In Abruptio Placentae, Stillbirth and Disseminated Intravascular Coagulopathy in Antenatal Clinic Defauter Woman in Calabar, South-South Nigeria

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Abstract:- This case report examines a poignant instance of severe preeclampsia in a 26 year old with a history of recurrent pregnancy loss and however, a prior vaginal delivery. The patient presented at 29 weeks gestation with acute lower abdominal pain with associated abruptio placentae, resulting in a stillbirth and disseminated intravascular coagulopathy (DIC). The intricate interplay of obstetric history and clinical management reveals the challenges in navigating such complex scenarios.

The patient's obstetric history of recurrent miscarriages and a vaginal delivery with perineal tear signals a predisposition to adverse outcomes. The acute presentation of abruptio placentae underscores the imperative for heightened vigilance in pregnant women even in the absence of antenatal complaints.

Successful resuscitation involved correcting shock, transfusing three units of blood and addressing hemorrhagic DIC. Tranexamic acid, pentazocine, normal saline, misoprostol and oxytocin were employed to stabilize the patient, highlighting the necessity of a comprehensive and multidisciplinary approach.

This case serves as a stark reminder of the unpredictable nature of severe preeclampsia and emphasizes on the critical importance of early detection and intervention. Future research endeavors should focus on refining risk stratification models and exploring innovative interventions to enhance maternal and fetal outcomes in high-risk pregnancies.

I. INTRODUCTION

Preeclampsia is defined as the presence of a systolic blood pressure (SBP) greater than 140 mm Hg or a diastolic blood pressure (DBP) greater than or equal to 90 mm Hg on two occasions at least 4 hours apart in a previously normotensive pregnant woman.[1]

DIC is characterized by systemic activation of blood coagulation, which results in the generation and deposition of fibrin, leading to microvascular thrombi in various organs and contributing to multiple organ dysfunction syndrome (MODS).[2] Consumption of clotting factors and platelets in DIC can result in life-threatening hemorrhage.[2] Preeclampsia is a pregnancy-specific disorder involving widespread endothelial dysfunction and vasospasm that usually occurs after 20 weeks of gestation and can present as late as 4-6 weeks postpartum.[3]

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We encountered a distressing scenario involving a 26-year-old woman from Calabar, South-South Nigeria who was admitted and managed on account of severe preeclampsia, culminating in abruptio placentae, stillbirth, and DIC. This case unveils the intricate interplay of obstetric history, clinical management and the critical need for timely intervention in mitigating adverse outcomes. Through a comprehensive analysis, we aim to glean insights into the complexities surrounding severe preeclampsia and its repercussions on both maternal and fetal health, offering valuable lessons for future clinical practice and research endeavors.

She is a 26-year-old Gravida 5, Para 1^{+3} (one alive) unbooked patient who presented with a complaint of acute lower abdominal pain persisting for 2 hours at 29 weeks gestation during her fifth pregnancy. The pain was sudden in onset, constant all through the day, severe, non-radiating, no known aggravating and relieving factor and no associated history of bleeding per vagina.

She has had three recurrent miscarriages at a gestational age of three months respectively and subsequently was delivered of a live male baby through the vaginal route though she sustained a perineal tear that was repaired. There is no known history of diabetes or other chronic medical condition prior to presentation.

General examination revealed a middle-aged lady in painful distress, well oriented, not calm and cooperative, mildly pale, afebrile with body temperature of 36.7 degree Celsius, anicteric, acyanosed, not dehydrated, nil pedal edema. Respiratory rate was 22 cycles per minute with bronchial breath sounds. Cardiovascular examination showed pulse rate of 108 beats per minute and blood pressure was 150/95milimeter of mercury.

Abdominal examination showed a gravidly enlarged uterus that moves with respiration with generalized abdominal tenderness and a woody-hard uterus, symphysiofundal height was 30 centimeters with a singleton fetus, cephalic presentating and longitudinal lie, the descent was $4/5^{th}$ palpable, uterine contractions were 2 in 10minutes lasting 36 seconds with absent fetal heart rate.

Vaginal examination showed a healthy vulva and vagina, cervix was anterior, soft in consistency, 2cm dilated, station of -2, gloved hands were stained with mucus.

Urinalysis revealed significant proteinuria and a diagnosis of pre-eclampsia with concealed abruptio placentae and intrauterine fetal demise was made. An urgent obstetric scan showed absent fetal heart rate and intrauterine fetal death was confirmed. A full blood count done revealed packed cell volume of 18%, low platelet and severe thrombocytopenia (Fig 2 and 3).

Patient was counselled on the findings and a multidisciplinary management approach involving obstetricians, hematologists, and medical physicians was instituted. She was commenced on oral antihypertensives (methyldopa and nifedipine) and also intravenous magnesium sulfate to prevent seizure. She was counselled on the need for cervical ripening and she consented. Cervical ripening was employed and subsequently she was delivered of a fresh male stillbirth with evidence of retro-placenta clotts noted. She developed uterine atony that resulted in postpartum hemorrhage which was managed appropriately. She was noticed to be bleeding from intravenous punture sites and petechiae hemorrhage were noted in several areas of her skin (Fig 3). Three units of fresh whole blood were immediately transfused and symptoms gradually resolved. She was discharged home six days after delivery and her postnatal clinic visits were uneventful.

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WBC	12.8	ADULT: 2.6 - 11.0 x $10^{9/L}$ CHILDREN AT 1YEAR: 4.0 - 15.0 x $10^{9/L}$ NEWBORN : 10 - 25.0 x $10^{9/L}$
NEU%	83	ADULTS (40-75), <7 YRS (25-45)
LYM%	16	ADULTS (20-45), <7 YRS (45-75)
MON%	00	(2 - 8)
EOS%	01	(1 - 6)
BAS%	00	(0 - 1)
RBC		MALE (4.5-5.9), FEMALE (4.1-5.1), CHILDREN AT BIRTH (4.0-5.6)
HCT%	19	MALE (38-52)%, FEMALE (37-47)%, CHILDREN AT BIRTH (44-54)%
MCV		(80.0 - 100.0)
MCHC		(32 - 36)
MCH		(27 - 32)

Fig 1: Full Blood Count

MCV		(80.0 - 100.0)
MCHC		(32 - 36)
MCH		(27 - 32)
PLT	110	(150 - 400)
HGB		12.0-18.0(Male). 11.0-16.0(Female) 16-24(Newborn)

Fig 2: Low Platelet

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Fig 3: Petechiae Hemorrhage

II. DISCUSSION

Pre-eclampsia complicated by disseminated intravascular coagulopathy (DIC) and intrauterine fetal demise is a serious and complex scenario that poses significant risks to both the mother and the fetus.[2] Pathophysiology of pre-eclampsia includes vascular dysfunction, leading to hypertension, proteinuria, damage to multiple organ systems, particularly the liver and kidneys and placental factors triggering an inflammatory response and endothelial dysfunction[3]. Clinical presentation entails hypertension (a hallmark symptom), proteinuria with or without bilateral pedal oedema.[3] Other Symptoms include; headaches, visual disturbances, abdominal pain, and edema.[3]

Pre-eclampsia can lead to organ dysfunction, affecting the liver, kidneys, and the central nervous system.[4] Impaired blood flow to the placenta can result in fetal growth restriction and, in severe cases, intrauterine fetal death.[5] Consumption of platelets and coagulation factors can also occur and leading to bleeding tendencies.[4] DIC involves systemic activation of the coagulation cascade, leading to widespread microvascular thrombosis resulting in a combination of bleeding manifestations due to consumption of clotting factors, microvascular thrombosis and thrombocytopenia.[4] DIC is often triggered by underlying conditions such as severe infections, trauma, or obstetric complications like severe pre-eclampsia and the widespread activation of clotting factors and platelets can lead to the formation of microthrombi, compromising organ perfusion.[4]

Abruptio placentae, is a serious obstetric complication characterized by the premature separation of the placenta from the uterine wall before delivery resulting in significant maternal and fetal morbidity and mortality.[5] The clinical presentation of placental abruption varies depending on the severity of the separation and the amount of bleeding.[6] Common signs and symptoms include sudden onset of intense abdominal pain, vaginal bleeding, uterine tenderness and rigidity, fetal distress and maternal shock depending on the magnitude of circulatory loss.[6]

Placental abruption can be categorised into 3 grades namely; Grade 1 placental abruption involves no vagina bleeding or less than 100mls of blood loss per vagina, no fetal heart rate abnormalities and no evidence of maternal shock.[6] Grade 2 placental abruption involves moderate vaginal bleeding (100-500mls), increased uterine tone (tetanic uterus),

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evidence of fetal distress, maternal tachycardia with narrowed pulse and early signs of coagulopathy (fibrinogen level 150-250dl).[6]

Grade 3 placental abruption involves maternal shock, coagulopathy, vaginal bleeding greater than 500mls and fetal death .[6,7]

Intrauterine fetal demise refers to the death of the fetus that occurs after the age of viability.[8] It usually occurs due to placental insufficiency.[7] In the context of severe pre-eclampsia, placental insufficiency as a result of impaired blood flow due to the pathophysiology of pre-eclampia.[1] Management of a mother with intrauterine fetal demise involves empathy, maternal stabilization, close monitoring and initiation of appropriate interventions.[8]

Delivery of the fetus depends on the gestational age and the decision for prompt delivery may be made to mitigate further risks.[8] Platelet and or fresh frozen plasma may be administered to address thrombocytopenia and correct coagulation abnormalities.[4] Addressing the underlying cause in this case is essential. The emotional impact of intrauterine fetal demise is profound and providing compassionate counselling and support for grieving parents is crucial while continued monitoring is necessary in the postpartum period to address any lingering complications and ensure the mother's recovery.[8]

III. CONCLUSION

Non-compliance to routine antenatal clinic schedule predisposes to maternal and perinatal morbidity and mortality. The unpredictable nature of severe pre-eclampsia and the importance of a comprehensive multidisciplinary approach in managing such complex obstetric scenario cannot be overemphasized.

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