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CASE REPORT

AN UNUSUAL PRESENTATION OF DIC

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ABSTRACT: Placenta Praevia is a common obstetric problem which presents with antepartum Haemorrhage and is treated by the obstetrician. It is very rare to see a patient with placenta praevia treated in medical ward for post-partum Haemorrhage due to disseminated intravascular coagulation defect with the added complication of acute kidney injury, Post-partum Hypertension and PRES (posterior reversible encephalopathy syndrome), hence the presentation.

KEYWORDS: Placenta Praevia, Post-partum Haemorrhage, DIC, acute kidney injury, dialysis, PRES (Posterior Reversible Encephalopathy Syndrome).

INTRODUCTION: One of the causes of DIC (Disseminated Itravascular Coagulation) is the obstetric complication of antepartum Haemorrhage due to Abruptio Placentae. It is almost never due to antepartum Haemorrhage due to (Placenta Praevia). This patient was diagnosed to have Placenta Praevia during routine antenatal USG check-up. She presented with bleeding per vaginum in the 30th week of pregnancy and a caesarian section was done and a female live foetus delivered but she developed the rare postpartum complication of DIC, acute kidney injury and PRES which was treated and despite of multiple complications she recovered completely.

CASE REPORT: Mrs. S aged 34 years G4P3L2 came for regular antenatal checkups and was diagnosed to have Placenta Praevia. There was no pregnancy induced hypertension.

This patient presented with painless vaginal bleeding in the third trimester of pregnancy. The uterus was 30 weeks size. The lower segment caesarian section was done and a live foetus was delivered. She developed severe postpartum haemorrhage which was torrential and could not be controlled by usual conservative means.In most of the cases coagulation defect undergoes spontaneous improvement within hours of delivery[1] but in this case it was not so. Ironically, bleeding increase in severity after delivery. She was investigated in detail. She had low platelet count <10000 cells/cumm, prolonged APTT, prolonged PT and raised D-DIMER (10000ng/ml).

She was given FFP, whole blood and RCC to the tune of 16 units to replace the lost blood. The bleeding stopped finally after ten days but she developed acute renal failure with anuria and hypertension due to severe blood loss and DIC. USG showed normal sized kidneys.

Haemodialysis was done 14 times and the urine output improved and serum creatinine reduced from 7.6 to 1.4 mg/dl.

Later she developed generalized seizures and altered sensorium. However, there was no focal neurological deficit. A CT scan of brain was done which revealed cerebral edema predominantly in occipital lobe. Posterior Reversible Encephalopathy Syndrome (PRES) was considered a possibility due to hypertension and her postpartum status. She also developed

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bilateral pleural effusion and ascites which was treated. Finally she recovered from all of these complications, completely.

DISCUSSION: Disseminated intravascular coagulation (consumptive coagulopathy) is a clinicopathologic syndrome characterized by microvascular fibrin and thrombin formation.[2] This also termed as defibrination syndrome The underlying pathology is commonly bacterial sepsis, obstetric causes and malignant tumours. In obstetrics, it is common in abruptio placentae and amniotic fluid embolism.[3] The central mechanism of DIC is the uncontrolled generation of thrombin by exposure of the blood to pathologic levels of tissue factor. There is simultaneous fibrinolysis.

There is fibrin deposition in small and midsized vessels which compromise the blood supply to lungs, kidneys, liver and brain resulting eventually in organ failure. There is consumption of clotting factors and platelets which lead to systemic bleeding.[4] Mortality can range from 30-80%.

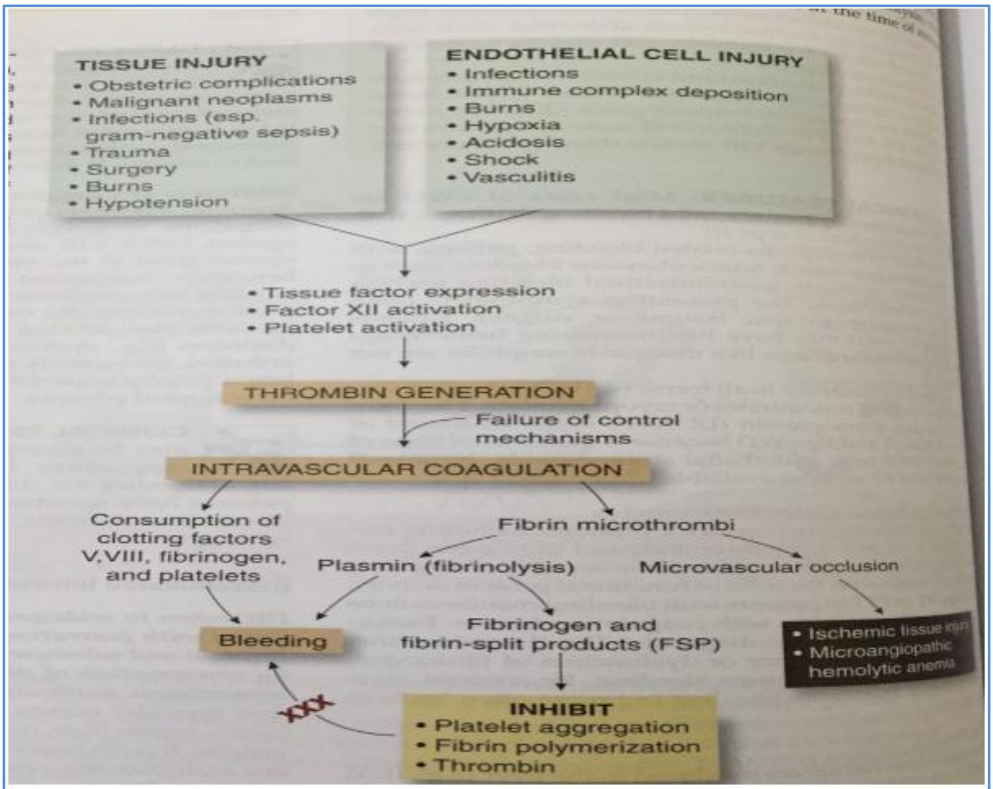

Fig: 1

Diagnoses of DIC are based on laboratory abnormalities of coagulation and thrombocytopenia. Common findings[5] include prolongation of PT, APTT, platelet count<10000/cumm, elevated FDP and fragmented RBCs in peripheral smear. D-DIMER test is a marker of fibrin degradation product. It was markedly raised in this patient, in addition to having raised PT, APTT and low platelets which confirmed the diagnosis of DIC.

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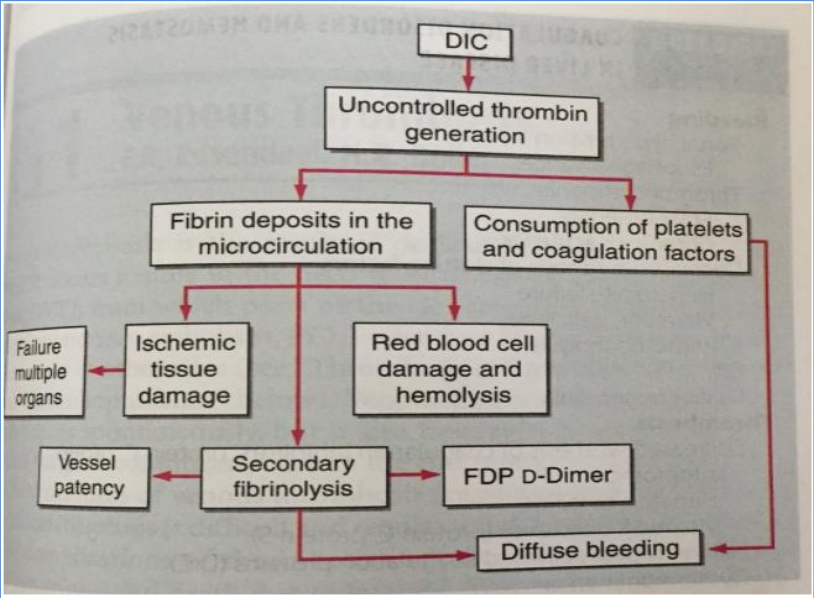

Fig: 2

Due to DIC and the severe blood loss she developed acute renal failure or acute kidney injury. DIC causes acute kidney injury due to glomerular pathology. In addition, the severe blood loss caused ischemic acute tubular necrosis.

The course[6] of ischemic ATN has 4 phases:

1) Initiation
2) Extension
3) Maintenance
4) Recovery

Cellular ischemia results in ATP depletion and oxygen free radicals formation. Recovery phase is characterized by tubular repair and GFR returns to normal.

Another problem was hypertension probably due to the glomerular insult of DIC.

She was also diagnosed to have PRES (Posterior Reversible Encephalopathy Syndrome), the etiology is unclear but postpartum period by itself is one of the causes. There is cerebral segmental vasoconstriction and cerebral edema. Patients have headache and fluctuating neurologic symptoms and signs. The clinical and imaging findings suggest that the ischemia reverses completely.

CONCLUSION: This patient suffered from many rare complications of pregnancy but recovered completely and was discharged after 45 days, healthy and sound.

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