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Case Report

Young Primigravida with Acute Renal Failure in a Case of Abruptio Placentae: A Case Report

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ABSTRACT

Abruptio placentae is an important obstetrical complication occurring in 1% of pregnancies. The basic underlying factor is hemorrhage at the decidua-placental interface. If severe, it significantly effects on fetal and maternal morbidity and mortality, with the most frequent complications being fetal death, severe maternal shock, disseminated intravascular coagulopathy, and renal failure. Ischemic damage to the kidneys is a well-known complication of severe Abruptio placentae and can be either acute tubular or acute cortical damage or both. And in extreme oliguria or anuria, it becomes difficult to distinguish between the two. Vigorous therapeutic regimens instituted when coagulation defects are identified lead to better maternal and fetal outcome.

Keywords: Abruptio placentae, obstetrical complication, acute renal failure, DIC-disseminated intravascular coagulopathy, coagulation defects.

INTRODUCTION

Abruptio placentae complicates almost 1% of all pregnancies. Renal failure is an important complication of Abruptio placentae. Disseminated intravascular clotting is the result of a widespread exposure of the circulating blood to procoagulant activity capable of activating fibrinolytic enzyme system converting fibrinogen into the fibrin. Fibrin may in turn cause small blood vessel occlusion resulting in tissue necrosis, and as the phenomenon occurs more often in the glomerular capillaries acute renal failure may ensue. [1]

CASE SUMMARY

We present a case of a 19 year's old unregistered, prim gravida, with IUGR at 28.3 weeks of gestation who presented with complaints of leaking per vagina since 2 days. The patient was vitally stable. Ultrasonography report s/o asymmetrical IUGR with 3 weeks lag and AFI -7cm. The patient was admitted for further investigation and management. A Colour Doppler was done which showed normal indices. Biophysical profile showed a score of 10. Patient was given corticosteroids for fetal lung maturation. The next day, patient started complaining of severe pain in abdomen and on examination uterus was tonically contracted with fetal heart decelerations. Patient's blood pressure was recorded to be 150/ 100 with urine albumin on dipstick test 2+. A provisional diagnosis of Abruptio placentae was made and patient was taken for emergency LSCS. INTRA OPERATIVELY: 80 grams of blood clots

(approx. 2000 ml of blood) were evacuated and diagnosis of Abruptio placentae confirmed. A still born Male baby of 1.5 kg was delivered Urine output in OT was 100 ml. Post LSCS patient had decreased urine output for which IV fluids and Inj. Lasix 40mg was given despite of which output remained 10ml in 2 hours.

INVESTIGATIONS

- Serum creatinine

Pre-operative- 1.0

Post-operative- 1.4 → 2.0 (by evening) → 3.5 (next morning)

- Blood urea- 46.0→74.0→86.9
- BUN-21.4→34.0→40.6
- USG- s/o bilateral echogenic kidneys with dilatation of pelvic calyces.
- D-dimer- 5.92 micro g/ml (0.0-0.5)
- FDP- 10 (<5.0)
- Fibrinogen – 251
- PT 16 INR 1.2
- Hb- pre op- 9.0 gm%
  Post op- 6.7 gm%

MANAGEMENT

- With the help of physician patient was managed jointly and conservatively with IV fluids and blood products.
- Two units of Whole blood was transfused.
- Strict monitoring along with input and urine output charting was done.

OUTCOME

- Urine output-

With good fluid management, UO had risen from 400 ml in first 24 hours of LSCS to 800 ml on Day 2 to 1200 ml on Day 3.

- Serum creatinine-

2.9→ 1.1→ 1.0 (repeated 12 hourly)

Patient was discharged on day 10 of LSCS with normal renal function tests.

DISCUSSION

Abruption placentae is one of the most dangerous obstetrical complications posing threat to both mother and fetus. Vascular disease plays a predisposing role in the disruption of the placental bed in some abruptions. Because hypertension has been shown to cause arteriolar degeneration, the compromised afferent blood supply to the placenta has been emphasized. In addition, superimposed arteriolar spasm would tend to aggravate the ischemia. Perhaps the entire entity may be a progressive stress or accelerated degeneration of an already defective vasculature of the placental bed. [2] Nelson and associates speculated that an acute reduction in the uterine volume and intrauterine surface area as a consequence of preterm PROM could ultimately lead to a disruption in the site of placental attachment, thereby resulting in abruption. [3]

Pregnancy related acute renal failure can include reversible tubular necrosis as well as irreversible cortical necrosis in which DIC probably plays a primary role. [4] Ischemic damage to the kidneys is a well-documented complication of severe Abruptio placentae and takes the form of acute tubular necrosis, bilateral cortical necrosis, or both. The pathologic changes are explained on the basis of inadequate blood supply and hypoxia from hemorrhagic hypovolemia or obstruction secondary to focal fibrin deposits in the precapillary arterioles from DIC. Other suggested mechanisms are vasospasm of the supplying arteries caused by humoral (serotonin release) or reflex (utero-renal) factors. When extreme oliguria or anuria develops, it is impossible to initially distinguish between acute tubular necrosis and acute cortical necrosis. The key to prevention of renal ischemic damage is vigorous blood and fluid therapy to combat hypovolemic shock. [2]

CONCLUSION

The frequency of Abruptio placentae with adverse maternal and fetal outcome is high even after advances in

obstetrical and neonatal care. Gestational hypertension, premature rupture of membranes, un-booked status, and rural residence are important risk factors. Early intervention and providing antenatal services particularly in rural areas, will help to reduce and prevent such incidences.

Hence, maternal morbidity and mortality can be reduced only with regular antenatal care. Anticipating the complications, early diagnosis and management can improve the outcome.

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