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Acute Kidney Injury Secondary to Abruptio Placenta: A Case Series Highlighting Maternal Complications.

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ABSTRACT:
Abruptio placenta, also referred to as placental abruption, constitutes a significant obstetric emergency characterized by the premature detachment of the placenta from the uterine wall, frequently resulting in both the mother and the fetus morbidity and mortality . This condition is associated with various complications including disseminated intravascular coagulation, hypovolemic shock, and multi-organ dysfunction. Among these complications, acute kidney injury (AKI) induced by abruptio placenta is a rare yet life-threatening condition that necessitates prompt recognition and intervention. This case series delineates the clinical manifestations, diagnostic difficulties, and outcomes linked to abruptio placenta leading to AKI, thereby underscoring the imperative for a multidisciplinary approach and swift management in such scenarios.
KEYWORDS: *Abruptio placenta , Acute kidney injury , Anuria , Dialysis ,IUFD,Hypertension.*

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I. INTRODUCTION

Pregnancy related Acute kidney injury (AKI) is a serious and potentially life-threatening complication in obstetric emergencies that puts the health of the mother and fetus at serious danger. Abruptio placentae, or the premature separation of the placenta from the uterine wall, is one of the main causes of pregnancy-related AKI.

Abruptio placentae involves the premature separation of placenta from the uterine wall, leading to hemorrhage and compromised blood flow to the fetus. This condition can result in significant maternal morbidity, including development of AKI.

AKI is defined by a sudden kidney function deterioration , which raises S. Creatinine levels and decreases urine production. Although AKI in pregnant women is uncommon, its possible consequences make it a serious issue. Its severity is demonstrated by the reported 30% to 60% incidence of pregnancy-related AKI.

In pregnant women, AKI is often secondary to hemorrhage, hypovolemic shock, or disseminated intravascular coagulation (DIC), all of which are potential consequences of abruptio placentae.

Pathophysiology of AKI in Abruptio Placentae: The onset of AKI in the context of abruptio placentae is due to:

Hemorrhagic Shock: Severe bleeding reduces effective circulating blood volume, leading to decreased renal perfusion.

Disseminated Intravascular Coagulation (DIC): The systemic activation of coagulation pathways can result in microvascular thrombosis, impairing kidney function.

Renal Cortical Necrosis: In severe cases, prolonged ischemia can lead to irreversible damage to the renal cortex, a condition known as renal cortical necrosis.

Symptoms of AKI includes:

i) oliguria or anuria (due to impaired kidney function)
ii) edema(fluid retention leading to swelling,
iii) hypertension (due to fluid overload and renal dysfunction)
iv) elevated S. Creat and BUN (indicators of impaired kidney function)

II. CASE REPORTS

CASE 1 :

24-year-old multigravida at 35 weeks + 2 days gestation who developed a massive placental abruption, leading to intrauterine fetal demise (IUFD), hemorrhagic shock, and acute kidney injury (AKI). The patient presented with severe abdominal pain, hypotension and pallor. On examination, the abdomen was hard and tender,

and no fetal heart activity was detected. Ultrasound confirmed a retroplacental hematoma (13 × 8 cm) and IUFD. Laboratory findings on admission showed a hemoglobin level of 6.6 g/dL, platelets of 1.03 lacs, AST of 40 U/L, D-dimer of 2.0, serum creatinine of 1.6 µmol/L, and urea of 68 mg/dL.

Given the unfavorable cervix and hemodynamic instability, an emergency cesarean section was performed. Intraoperatively, approximately 1L of coagulum was evacuated, and the uterus showed intramural bleeding indicative of early Couvelaire uterus. Uterine atony was managed with uterotonics and B-Lynch sutures, and the patient received blood transfusions and blood products. Postoperatively, on POD 1, the patient developed oliguria with metabolic acidosis, a hemoglobin level of 8.8 g/dL, platelet drop to 88,000, and worsening renal function, prompting nephrology consultation and initiation of IV furosemide (40 mg TDS). On POD 3, the patient became anuric with serum creatinine rising to 3.6 µmol/L, urea 70 mg/dL, uric acid 20 mg/dL, serum sodium 133 mEq/L, and serum potassium 6.6 mEq/L. Two cycles of dialysis were performed, and by POD 5, urine output normalized. The patient remained stable and was discharged on POD 7 with nephrology and obstetric follow-up.

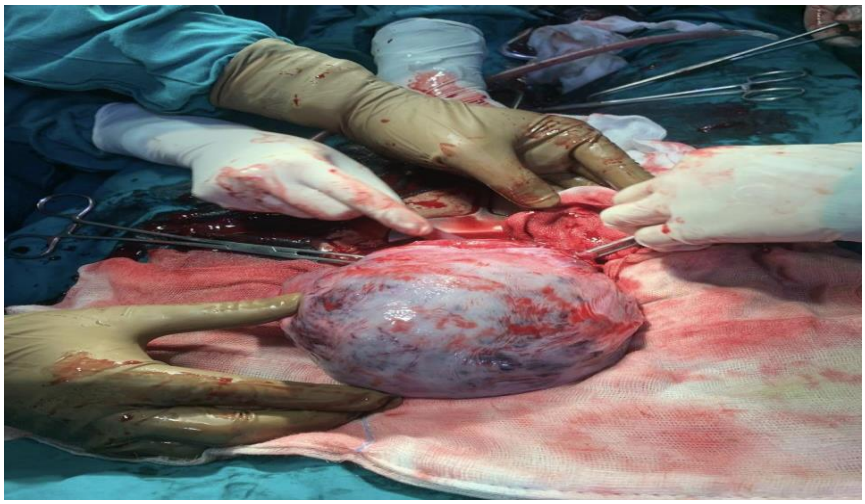

Fig 1: Couvelaire uterus showing intramural bleeding.

CASE 2:

32-year-old multigravida was admitted at 35 weeks of gestation, presenting with vaginal bleeding, abdominal discomfort and CTG showed reduced fetal movements. On clinical assessment the uterus was tonically contracted. Patient was hypotensive , very pale. Lab showed Hb 7.0g/dl, Plt 1 lakh, S. Creat. 1.7; urea 30 . She subsequently developed severe hypovolemic shock, necessitating vigorous fluid resuscitation and emergency caesarean delivery. Intraoperatively retroplacental clot of 500cc was present baby had delayed cry handed over to pediatrician , blood and blood products started intraoperatively .postoperatively patient was stable but developed to oliguria , and laboratory analyses indicated elevated creatinine levels and ABG showed metabolic acidosis, confirming the diagnosis of AKI. , nephrology opinion taken patient started on lasix, fluid balance was calculated input output was measured. Patient developed anuria with rising serum creatinine . She required 5 cycles of hemodialysis on day 14 she was discharged with normal urine output and was asked to follow up on discharge.

CASE 3 :

A 27 yrs old patient multigravida admitted in 35.6 weeks presenting with raised blood pressure, pain in abdomen , vaginal bleeding , CTG showed no fetal sound found. Clinical assessment showed tonically contracted uterus. Patient was hypertensive ,pale . Labs- Hb-5.6g/dl, PLT 1.2 lakh . RFT, sr.creat 1.2 g/dl uric acid 30, urea 40. Ultrasonography was confirmed intrauterine demise and retroplacental clot was present. Digital vaginal examination showed unfavorable cervix .emergency section was taken. Intraoperatively retroplacental sepearationseen 380 cc of clots seen. Blood and FFP was started intraoperatively. Postoperatively the patient was monitored, there was anuria. lab parameters along with ABG repeatedly showed metabolic acidosis hb 8.0 mg/dl plt 70000, sr creat 2.3,urea 40 , uric acid 18. D dimer 3 aptt 27. patient shifted to ems icu .nephrology opinion taken strict input output charting and inj lasix 40 mg tds advised, still no anuria perist day 2 rft repeat rft in increasing trend sr potassium kept increasing dialysis adviced anuria persisted despite giving diuretics hemodialysis planned . On day 21 urine output started coming within a few days aki reversed and urine output was back to normal. Patient discharged on day 28th with follow up advice.

III. OBSERVATION:

Parameter	Case 1	Case 2	Case 3
Age (years)	24	32	27
Gravida	Multigravida	Multigravida	Multigravida
Gestational Age (weeks)	35+2	35	35+6
Presenting Symptoms	Severe abdominal pain, no fetal heartbeat, vaginal bleeding	Vaginal bleeding, abdominal discomfort, reduced fetal movements	Raised BP, abdominal pain, vaginal bleeding, no fetal sound
Clinical Findings	Hard, tender abdomen, hypotensive, pale	Tonically contracted uterus, hypotensive, pale	Tonically contracted uterus, hypertensive, pale
CTG Findings	No fetal heartbeat	Reduced fetal movements	No fetal sound
Ultrasound Findings	Retroplacental hematoma (13 × 8 cm), IUFD	Retroplacental clot (500cc), fetal distress	IUFD, retroplacental clot
Laboratory Findings	Hb 6.6 g/dL, Plt 1.03 lacs, Sr. Creat 1.6 µmol/L, Urea 68 mg/dL	Hb 7.0 g/dL, Plt 1 lakh, Sr. Creat 1.7 µmol/L, Urea 30 mg/dL	Hb 5.6 g/dL, Plt 1.2 lakh, Sr. Creat 1.2 µmol/L, Urea 40 mg/dL
Mode of Delivery	Emergency C-section	Emergency C-section	Emergency C-section
Intraoperative Findings	1L coagulum, intramural bleeding (Couvelaire uterus)	Retroplacental clot (500cc), baby had delayed cry	Retroplacental separation, 380cc clots
Intraoperative Management	Blood transfusion, uterotonics, B-Lynch sutures	Blood transfusion, vigorous fluid resuscitation	Blood and FFP transfusion
Postoperative Complications	Oliguria, metabolic acidosis, AKI, anuria	Oliguria, metabolic acidosis, AKI, anuria	Anuria, metabolic acidosis, rising creatinine, hyperkalemia
Nephrology Consultation	Yes	Yes	Yes
Treatment for AKI	IV Lasix, 2 cycles of dialysis	IV Lasix, 5 cycles of dialysis	IV Lasix, hemodialysis

IV. DISCUSSION:

- **Key Observations and Clinical Implications:**
- **Hemodynamic Instability and Hemorrhagic Shock**:

- All three patients were hypotensive and pale, indicating significant blood loss.
- Massive retroplacental hematomas (ranging from 380cc to 1L) were observed intraoperatively, contributing to hemodynamic instability.
- Aggressive fluid resuscitation and blood transfusions were critical in stabilizing the patients

- **Acute Kidney Injury (AKI) as a Major Postoperative Complication:**

- All three cases developed oliguria/anuria postoperatively, with rising serum creatinine, metabolic acidosis, and hyperkalemia, consistent with AKI.
- The degree of renal impairment correlated with the severity of hemorrhagic shock, suggesting that prolonged hypoperfusion was the primary etiology of AKI.

- Despite early intervention with diuretics (IV Lasix) and nephrology consultation, dialysis was required due to persistent anuria and worsening biochemical parameters.
- Case 3 had the most prolonged course of AKI, requiring hemodialysis until Day 21, emphasizing the variability in recovery timelines.

- **Role of Multidisciplinary Management:**

- The early involvement of nephrologists plays a crucial role in patient survival and renal recovery.
- Strict hemodynamic monitoring, serial renal function tests, ABG analysis, and fluid balance assessment were essential in guiding management decisions.

- **Maternal and Neonatal Outcomes:**

- Unfortunately, two cases resulted in IUFD, underscoring the high fetal mortality associated with severe placental abruption.

- **Long-term Considerations:**

- While all patients showed renal recovery and were discharged in stable condition, long-term follow-up is crucial to monitor for chronic kidney disease (CKD), hypertension, and postpartum anemia.
- Patients with placental abruption and AKI are at increased risk for recurrence in future pregnancies, emphasizing the need for preconception counseling and close antenatal surveillance.

V. CONCLUSION:

- **Key observations from this case series include:**

- Placental abruption remains a major obstetric emergency that can lead to severe maternal morbidity, including AKI, coagulopathy, and multi-organ dysfunction.
- AKI is a serious complication in hemorrhagic shock and requires early recognition, aggressive fluid management, diuretic therapy, and, in severe cases, dialysis.
- Multidisciplinary management involving obstetricians, nephrologists, and critical care teams is crucial for optimal patient outcomes.
- Timely intervention, strict hemodynamic monitoring, and appropriate postoperative care significantly improve maternal recovery and prevent long-term renal complications.
- This case series emphasizes the need for prompt diagnosis, rapid surgical intervention, and proactive postoperative management to prevent maternal mortality and morbidity. Future research should focus on early predictive markers for AKI in obstetric emergencies and protocol-driven management strategies to improve maternal outcomes in cases of severe placental abruption.

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