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Mild Preeclampsia with abruptio Placenta and Acute Kidney Injury– A Case Report.

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ABSTRACT

Acute kidney injury(AKI) is the sudden impairment of kidney function with retention of nitrogenous and other waste products normally excreted by kidneys. AKI is commonly associated with preeclampsia and haemorrhage, especially placental abruption. Case report:A woman at 33wks of gestation in mild anaemia, had preeclampsia. She was treated with antihypertensive drugs and kept under observation. Four hours after admission,she had increased abdominal pain and examination revealed scar tenderness. Emergency LSCS was done in view of threatened scar rupture. But the intra operative finding was abruptio placenta. Since the patient was obese, clinical diagnosis of abruption was difficult. Three hours after surgery she developed AKI.The underlying causes for AKI were mild preeclampsia, abruptio placenta and pre-exist edanaemia.

Keywords: acute kidney injury(AKI), preeclampsia, abruptio placenta, anaemia.

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INTRODUCTION

Acute kidney injury(AKI) is the sudden impairment of kidney function with retention of nitrogenous and other waste products normally excreted by kidneys. The incidence of AKI in pregnancy is decreased in recent years, but still occasionally causes significant obstetrical morbidity and mortality. AKI is commonly associated with preeclampsia and haemorrhage especially placental abruption. Preeclampsia includes gestational hypertension with proteinuria, i.e, the blood pressure ≥ 140/90 mm Hg with persistent 30ml/dl protein in random urine sample(1+ dipstick), after 20wks of gestation. Abruptio placenta is defined as the premature separation of normally implanted placenta, either partially or completely. Both are the major causes of maternal morbidity and perinatal mortality globally and it is of serious concern in the developing countries.This is a case reportofa pregnant woman withpreeclampsia associated with pre-existed anaemia causing abruptio placenta further leading to acute kidney injury.

CASE REPORT:

A 32- year old woman, gravida 2, para 1, live1, previous LSCS done for fetal distresssix years back admittedat 33wks of gestation with complaints of mild lower abdominal pain on & off fortwo hours. Patient was able to perceive fetal movements well.This was her first visit to our hospital. Previously she was taking antenatal checkup from a private practitioner and was diagnosed to have mild gestational hypertension, two weeks back, for which she was adviced admission but she didn't comply. On clinical examination, she was obese (wt – 98kgs) and mildlyanaemic, with bilateral pitting pedal edema grade – 2, BP 156/102mmHg, she had no imminent signs of eclampsia. PR – 88/min. CVS & RS was normal. Abdominal examination showed obese abdominal wall. Uterus corresponds to 32-34wks size with no contraction, small head- mobile,FHS – good. Suprapubic transverse scar present & healthy. Mild tenderness present in hypogastric region.Per vaginal examination showed parous os, membranes present, head above brim, pelvis – gynaecoid. She was started on antihypertensives, tab. Labetalol 100mg BD. One dose of steroid given.BP and PR chart were maintained.Ultrasound showed single live intrauterine gestation corresponding to 33wks, placenta anterior, liquor adequate, FHS – 148/min. Investigation :Hb – 9.8gms/dl, urine routine showed albumin 2+ & no pus cells, urea – 32mg/dl, creatinine – 1.2mg/dl, uric acid – 4.2mg/dl, LFT - normal. Four hours later,she hadsevere lower abdominal pain. On examination, she was mildly anaemic, BP – 130/90mmHg, PR – 104/min. Abdominal examination showed scar tenderness. Uterus not tense, not tender.Per vaginal examination was same as above. Emergency repeat LSCS was donewith the provisional diagnosis of threatened scar rupture. Intra operative finding: uterus was intact, no evidence of scar dehiscence,liquor blood stained, an alive female baby of wt – 1.7kg delivered, 100gms of retroplacental clots seen. One unit of B positive packed cell transfused intraoperatively. Postoperatively hourly urine output maintained. It was about 15 – 25ml/hr in spite of giving intravenous fluids 125ml/hour. Three hours after surgery, there was nil urine output. RFT elevated with urea – 60mg/dl, creatinine–5mg/dl, showing acute kidney injury, for which she underwent four sittings of dialysis. Then she was discharged with stable sr.creatinine – 2.5mg/dl. Patient followed up and the renal parameters were normal (sr.creatinine – 1.2mg/dl) after two weeks.

DISCUSSION

Acute kidney injury is an infrequent but life-threatening complication of pregnancy. Pregnancy has a minimal effect on renal function and is usually not associated with postpartum deterioration or development of end-stage renal disease. The most common cause of renal failure in pregnancy is preeclampsia; however, only about 1.5% - 2% of preeclampsia patients develop failure. Renal failure is typically associated with septic abortion, preeclampsia, or uterine hemorrhage from placental abruption or placenta previa. The initial management of renal failure in pregnancy commences with treatment of the underlying causes.

In this case, even though the patient had mild preeclampsia, which was also under control after medication, she went in for abruption, may be because of pre-existing anaemia. We don't know when the abruption could have exactly happened. Clinical diagnoses of abruption were not made because obese abdominal wall. Though abruption with 100gms of retroplacental(RP) clots is not significant, she went in for AKI. Usually AKI occurs following severe preeclampsia with HELLP syndrome or in placental abruption with profuse bleeding. But this is an atypical presentation. Mild preeclampsia, mild anaemia and 100gms of RP clots as separate entities will not lead to AKI but together caused AKI in this particular case. Hence any complication should not be overlooked.

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CONCLUSION

This is a case of acute kidney injury due to mildpreeclampsia for short duration with pre-existing anaemia and placental abruption. AKI during pregnancy is a rare condition and we do occasionally encounter them. It still remains a serious medical complication of pregnancy. The treatment of AKI during pregnancy is the correction of underlying disease. So the early diagnosis and treatment well save the life of the patient.

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