Case report

Acute renal failure following chorioamnionitis

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Case report: Renal failure is a rare entity encountered in obstetrics commonly found as complication of septic abortion, severe PIH, APH and PPH and rarely as a result of hyperemesis gravidarum, chorio-amnionitis (1%) and idiopathic postpartum renal failure. Chorio- amnionitis is a frequent occurrence as a result of premature rupture of membrane, however acute renal failure is unusual consequence of chorio-amnionitis. We are presenting this case of renal failure as a result of chorio-amnionitis because of its rarity in the obstetric practice.

Key words: Renal failure, chorioamnionitis, premature rupture of membrane

Introduction

Acute renal failure is a clinical syndrome characterized by sudden and marked decreased in glomerular filtration rate, rising plasma urea and creatinine levels and urine output <400ml in 24 h. Mostly acute renal failure in obstetrics occurs in persons with previously healthy kidneys but it may occur as a complication to pre existing renal disease. Renal failure is a rare entity encountered in obstetrics commonly found as complication of septic abortion, severe PIH, APH and PPH and rarely as a result of hyperemesis gravidarum, chorio-amnionitis (1%) and idiopathic postpartum renal failure. Chorio- amnionitis is a frequent occurrence as a result of premature rupture of membrane, however acute renal failure is unusual consequence of chorio-amnionitis. We are presenting this case of renal failure as a result of chorio-amnionitis because of its rarity in the obstetric practice.

Case

A 22 Yrs old housewife form Gorkha was admitted with diagnosis of G_2P_1 at 41^{+2} weeks of gestation in early stage of labour with high leaking for 3 days. It was a booked pregnancy, first booking being done at 24 weeks gestation and the last at 38 weeks and she had an uneventful antenatal period. At admission, she complained of on and off passage of clear fluid per vaginum since the last three days and labour pain for one day. She denied of having fever and foul smelling discharge per vaginum. Examination at admission confirmed that she was afebrile and other vital parameters were within normal limits. Obstetric examination showed that abdomen was of term size with fetus in longitudinal lie and cephalic presentation and no uterine tenderness, fetal heart rate was normal. Pelvic examination revealed frank leaking of liquor and cervical dilatation of 1.5 cm. High vaginal swab was taken and sent for culture. Cardiotocography was done which was reactive after which she was augmented with oxytocin as she had only mild uterine contraction.

After 4 hours of admission, she was detected to have fever (39^oC) and investigations showed normal total and differential leucocyte count. Blood and urine culture sensitivity were also sent and antibiotics were started. Amniotomy was done that time at cervical dilatation of 3cm, which revealed thick meconium stained liquor and so cesarean section was done. Intraoperatively, the liquor was moderate meconium stained adequate in amount but not foul smelling. Operative period was uneventful except that uterus was periodically atonic and required additional dosage of oxytocin and ergometrin. She delivered a baby girl weighing 3550gm with good Apgar Scores. Vitals remained stable and she had a total blood loss of 250ml.

After half an hour of surgery, call was attended for bleeding per vaginum, but there had been no fall in vitals and uterus was contracted. Three pads had been partially soaked and

about 80 gm clots removed from vagina. To avoid uterine atony, 800 µgm of PG E_2 and 250 µgm of PG F_2a. were given prophylactically.

The first post operative day was uneventful. She was afebrile and there was no further bleeding per vaginum, Hb was 10.8gm%. She maintained urine output of 65ml per hour; was then started on oral antibiotics and oral feeding. The second day following surgery was complicated by development of several episodes of diarrhea (4 to 5 times). There was no dehydration but fever of 38°C was detected. Serum electrolytes sent had been normal and she was managed with oral rehydration solution and oral metronidazole. Unfortunately, she missed to have her urinary output measured on this day on account of being treated as mild diarrhea. Following day was even worse with further more episodes of diarrhea, two episodes of vomiting, severe myalgia and shortness of breath. The urine output had been absent for last 24 hours which had gone unnoticed. Examination of chest showed that there were bilateral diffuse rhonchi and a soft systolic murmur in the mitral area. Investigations sent showed raised neutrophilic leucocyte count, raised blood urea (2.5 folds) and serum creatinine (4 folds). With diagnosis of acute renal failure following sepsis and diarrhea, she was transferred to nephrology unit for needful haemodialysis. She underwent the first session of haemodialysis on the third day following cesarean delivery and second day of severe diarrhea.

Coagulation profile was also sent which were deranged with lowered platelets count (27,000/cumm) and raised prothrombin time, activated partial thromboplastin time and Fibrin degradation products. Supportive care was given with FFP and fresh blood transfusion, antibiotics (ceftriaxone) and diuretics. The culture reports had been available by that time. Though blood and urine showed no growth of any organism, placental tissue culture that had been sent post cesarean delivery showed growth of citrobacter species sensitive to ceftriaxone with which she had been already treated.

She developed anuria for the following five days despite high doses of diuretics and biweekly sessions of heamodialysis. After more than a week following cesarean section and having diarrhea, urine formation occurred as evidenced by the urine out of upto 120ml a day. She also developed hypertension secondary to renal failure. Over the following days she continued to be managed with haemodialyis, diuretics and antihypertensives. She remained oliguric despite all these measures even till the end of 6 weeks following acute renal failure. The serum creatinine level persistently kept rising and ultrasound showed increased cortical echotexture with renal parenchymal involvement. She stayed in the hospital for 58 days during which she underwent 33 sessions of haemodialysis. She was ultimately referred for renal transplant before which was advised to follow up for regular dialysis.

Discussion

Renal failure resulting from obstetric complications and interventions is uncommon. It occurs in 1:10000 [1] pregnancies and 22 % of cases of acute renal failure are of pregnancy related with mortality of 48%[2], Similar result has been reported by Naqvi R et al, according to which 24% of renal failure is of obstetrical origin resulting 26% irreversible damage and 23% of mortality.[3] In Nepal such figures are not available regarding pregnancy related renal failure.

There have been many causes which are attributable to renal failure during pregnancy. The causes being septic induced abortion, hyperemesis gravidarum, severe PIH, APH, PPH, Chorioamnionitis, Pyelonephritis, and idiopathic postpartum acute renal failure. In the past, the most common cause of acute renal failure was septic induced abortion however as a result of legalization of abortion the incidence of acute renal failure in obstetrics has reduced along with the incidence of septic abortion. A study done by Prakash J et al showed a declining trend of pregnancy related acute renal failure from 15% in 1982-1991 to 10% in 1992-2002.[4] Recently preeclampsia-eclampsia had replaced as a principal cause of pregnancy related renal failure.[5] Chorio-amnionitis following premature rupture of membrane has been attributed as a rare cause of renal failure (1%). Chorio-amnionitis leading to bacteraemia and septicaemia leads to disseminated intravascular coagulopathy and ARF in 3-12% and finally 9-22% leading to chronic renal failure requiring dialysis and renal transplant like our patient who needed several sittings of dialysis during puerperium finally requiring renal transplant. The onset of renal failure due to infection is usually acute in nature as our patient had also acute high grade fever, diarrhea and severe myalgia along with decreased urine output. ARF resultant of sepsis is mainly due to nephrotoxins produced by the organisms where the common organisms are E. coli and Clostridium[6] and in our case it was Citrobacter species.

In women when renal failure occurs in postpartum period management is not complicated by fetal consideration. Renal cortical necrosis was suspected (though histopathological evidence was not available) in our case as it occurs commonly during puerperium, several days of anuria and is associated with intense intravascular coagulation as evidenced by blood pictures which were treated with fresh blood and fresh frozen plasma. The initial prolong (5 days)

anuria and irreversible damage is suggestive of renal cortical necrosis as literature shows that anuria more than three days represents bad prognosis.[7]

Early diagnosis appears to reduce the mortality and may enhance the recovery of renal function. In our case also early initiation of dialysis has prevented the mortality but unfortunately could not prevent the morbidity associated with chronic renal failure.

Conclusion

Acute renal failure which was high in the past has decreased in the recent years. The most important measure is prevention of the condition and if it occurs aggressive management of obstetric complications reduces maternal mortality and morbidity associated with it.

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