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A Case of Acute Renal Failure Secondary to Injudicious Use of Diuretics in a Case of Severe Pre-Eclampsia

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Abstract: *Hypertensive disorders complicate 5-10% of pregnancies. They are associated with poor maternal and foetal outcomes. Prompt diagnosis and judicious treatment is essential for a better foetomaternal outcome. We are reporting A case of primigravida with 34weeks of gestation diagnosed with preeclampsia at 26weeksof gestation who was on irregular, inadequate treatment in whom diuretics were used injudiciously ultimately landed with acute renal failure.*
Key words: *Pre eclampsia, acute renal failure, diuretics.*

I. Introduction

Hypertensive disorders complicate 5 to 10 percent of all pregnancies, and together they form one member of the deadly triad, along with hemorrhage and infection, that contribute greatly to maternal morbidity and mortality rates [(1)]. Most of the complications of pre eclampsia in developing countries are due to irregular antenatal care leading to late diagnosis, improper care due to inadequate awareness amongst care providers regarding the difference in the treatment of hypertension in pregnant & non-pregnant patients. Maternal complications include eclampsia, HELLP syndrome, renal failure, cerebral hemorrhage, DIC & maternal death. Fetal complications are IUGR, oligoamnios leading to fetal distress, thereby increasing the neonatal morbidity & mortality[(2).] We are reporting a case of injudicious use of the diuretic Furesemide in the antenatal period in view of controlling hypertension & improving the renal output who presented to us as acute renal failure.

II. Case Report

Mrs. X aged 19 years, a Primigravida presented to Dr. PSIMS & RF on 10/8/2013 with history of completion of 8 calendar months of amenorrhea. She complained of swelling of both feet since one month not relieved on overnight rest with puffiness of face since one week suggestive of pathological edema. She was appreciating fetal movements well. There was no history of pain abdomen, watery discharge P/V or bleeding P/V.

She has been married since 2 years, spontaneous conception. She had irregular medical checkups with her local general practitioner, not an obstetrician. She was informed that her blood pressures were high at 6 completed months & started on antihypertensives (Tab. Methyl dopa 250 mg bid). The patient was not regularly taking her medication & discontinued the medication on her own about one month prior to admission.15 days prior to admission, she was started on the same medication & since one week she has been receiving Inj. Furesemide once daily in view of decreased urine output & uncontrolled blood pressures. Patient reported that her urine output improved with the injections. She had received one dose of the diuretic prior to referral to this hospital. The history was correlated by contacting the referral doctor & the medications received were confirmed as the documentation was inadequate. There was no positive history suggestive of impending eclampsia.

Her menstrual cycles were regular & her dating scan at 12 weeks of gestation corresponded to the gestational age of 34 weeks + 3 days by dates. There was no significant past, family or personal history relevant to her present medical condition. On examination, the patient was conscious & coherent. She had grade 4 edema.

Vitals were stable with a blood pressure recording of 120/80 mm of Hg.CVS & RS examination revealed no abnormality. Per abdomen examination revealed abdominal wall edema, fluid thrill present, fundal height was corresponding to 28 weeks of gestation. Her blood pressure started rising one hour later & the antihypertensive dose was increased to 500 mg tid.Laboratory investigations:Hb% - 15.5 gms%, Blood group & type: B negative, PCV: 45 % TC & DC: Within normal limits. Platelet count: 1.5lakhs/cmm Coagulation profile: Within normal limits. A/G: 0.7: 1. LFT: within normal limits RFT: Blood urea: 103mg%(normal levels in pregnancy: 18 – 20 mg%). Serum creatinine: 2.2 mg% (normal values: 0.8 mg%)Urine examination: albumin 2+ Serum electrolytes: Na+: 132meq/lt, K : 3.5meq/lt USG: Single live intrauterine fetus of 32 -34 weeks. Gestational age in breech presentation with AFI of 3 cms, with maternal ascitis Fundoscopy : normal study.

Patient was monitored closely for the next 6 hours & Inj. Betamethasone 12mg IM was given & her urine output was 700ml. Blood pressure range was between 140/90 to 150/100 mm of Hg. A diagnosis of primigravida with 34 weeks of gestation with severe pre eclampsia with IUGR was made & since the urine output was good we decided to wait to give the benefit of steroids to the foetus. 6 hours later urine output decreased to about 20 ml / hour over the next 4 hours & 15ml in the next 2 hours in spite of adequate intake. Repeat RFT revealed a slight elevation in the renal parameters with a creatinine levels of 2.6 mg%. UOP in 12 hours was 800ml with a rapid reduction in the past 6 hours. This could be explained with the effect of diuretic wearing off. An Emergency LSCS under spinal anesthesia was planned in view of severe pre eclampsia with acute renal failure. Preoperatively, since prerenal cause was suspected for renal failure, central venous pressure line was introduced to help us guide the amount of fluids to be infused. This was to prevent overload & subsequent pulmonary edema which could be fatal. Her initial CVP was 0 cms of H_2O. IV Fluid treatment was aimed at maintaining the CVP at 5- 7 cms of water. A live female baby of 1.5 kgs was delivered by breech with minimal clear liquor. On table blood loss was minimal. Baby's APGAR score was at 1 min: 5/10 & at 5 mins : 6/10. Urine output was 15 ml for the next 2 hours & the patient was anuric over the following 6 hours. IV fluids were maintained at 50 ml / hour. RFT revealed urea of 100mg% & S. creatinine of 2.4 mg%. Dialysis was considered but deferred in view of the renal parameters remaining the same with no further deterioration. Patient was monitored for vitals & a strict intake output chart. She did not require further antihypertensives as her blood pressure was within the normal range. Her urine output gradually started improving 8 hours after surgery. 24 hours later she had an output of 50ml/hour. RFT was monitored twice daily. S. creatinine was 1.1 mg% on day 2 of surgery. Her further post operative period was uneventful & she was discharged on the 10th post operative day. She did not require antihypertensives in the post operative period.

III. Discussion

Acute renal failure is defined as a rapid decrease in the glomerular filtration rate over minutes to days, acute renal failure is termed acute kidney injury by the American Society of Nephrology.[3]

Definition introduced by the Acute Kidney Injury Network (AKIN), specific criteria exist for the diagnosis of AKI.[4]

Reduction of kidney function

- Rise in serum creatinine, defined by either:
  - Absolute increase in serum creatinine of ≥0.3 mg/dl (≥26.4 μmol/l)
  - Percentage increase in serum creatinine of ≥50%
- Reduction in urine output, defined as <0.5 ml/kg/hr for more than 6 hours

Pre -eclampsia, eclampsia and HELLP syndrome accounted for 5 (11.6%).[5]

Patients who develop acute renal failure, prerenal and obstructive causes must be excluded. Particularly important causes of prerenal azotemia in pregnancy include hyperemesis gravidarum and uterine hemorrhage, especially if it is unsuspected as in abruptio placentae .[6]

In our case the acute renal failure was probably precipitated by the use of diuretics resulting in volume depletion in an already contracted intravascular space which is seen in pre eclampsia. The prerenal cause added to the pathology seen in pre eclampsia which is vasospasm resulting in elevated renal parameters & oliguria. An acute increase in serum creatinine is usually due to renal ischemia. Oliguria is an important sign of acutely impaired renal function. In obstetrical cases, both prerenal and intrarenal factors are commonly operative. Furosemide (Lasix) is a loop diuretic.[7] The mechanism of action is by potently inhibiting the reabsorption of sodium and chloride at the ascending loop of Henle in glomerulus. It acts by direct Venodilation in Pulmonary edema

1. Reduces venous return (preload)
2. Reduces central venous pressure
3. Synergistic effect with morphine and Nitroglycerin

Reduces Intravascular Volume

4. Reduces cardiac output
5. Hypotension may lead to Myocardial Infarction

Adverse Effects:

1. Risk of central volume depletion (dehydration)
2. Hypotension
3. Electrolyte abnormalities
   1. Metabolic Alkalosis
   2. Hypokalemia
   3. Hypomagnesemia
   4. Hypocalcemia
   5. Hyponatremia

6. Hyperosmolality

Indications:

1. Symptomatically reduce pulmonary and Peripheral Edema
    1. Renal Insufficiency
    2. Congestive Heart Failure
2. Emergency Management of Pulmonary Congestion
    1. Left Ventricular Dysfunction (CHF)

In our patient the pre eclampsia presented with normal biochemical parameters except for elevated RFT. This progressed to result in acute renal failure which was diagnosed by serum creatinine levels of more than 2 mg% & patient being oliguric for 6 hours. Further delay in delivering the patient would probably have resulted in irreversible kidney injury.

This patient had irregular antenatal checkups with inadequate evaluation of her condition. Though a diagnosis of hypertension in pregnancy was made promptly, the medication & anticipation of the complications was found to be lacking. In non pregnant patients with chronic hypertension, diuretics are one of the first line medications. But it is contraindicated in pregnancy. It is a category C drug to be used in cases of pulmonary edema & CCF. It may cause metabolic derangements in the mother & abortions in early pregnancy. The injudicious use of diuretics further aggravated the insult to the kidneys resulting in a shut down. The timely referral of the patient to an institution & prompt recognition & intervention prevented expensive dialysis & potentially fatal condition of irreversible kidney failure.

Prevention:

According to varies studies the leading causes of acute renal failure are pre eclampsia-eclampsia, obstetrical hemorrhage, abortions with the incidence of septic abortion on the decline. Whereas in earlier years obstetrical cases composed 33 percent of all patients requiring dialysis, more recently these accounted for only 10 percent. [(4)]

Acute tubular necrosis may often be prevented by the following means:

1. Prompt and vigorous replacement of blood in instances of massive hemorrhage, such as in placental abruption, placental previa, uterine rupture, and postpartum uterine atony
2. Termination of pregnancies complicated by severe preeclampsia or eclampsia and careful blood replacement if loss is excessive
3. Close observation for early signs of sepsis syndrome and shock in women with pyelonephritis, septic abortion, chorioamnionitis, or sepsis from other pelvic infections
4. Avoidance of potent diuretics to treat oliguria before initiating appropriate efforts to ensure that cardiac output is adequate for renal perfusion
5. Avoidance of vasoconstrictors to treat hypotension, unless pathological vasodilation is unequivocally the cause of the hypotension.

IV. Conclusion

In India, due to lack of resources and awareness , most of the antenatal women seek medical help from general medical practicioneers.It is utmost duty of every medical personnel to identify high risk pregnancies and refer them to centers equipped to handle such cases.In the above discussed case there was a delay in diagnosis,inadequate treatment,irregular follow up and injudicious use of diuretics,to treat pre-eclampsia resulting in acute renal failure.Proper diagnosis and timely treatment is essential for good foetomaternal outcomes.

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