



CASE REPORT OF ACUTE KIDNEY INJURY IN PREGNANCY- AN ENIGMA

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KEYWORDS :

INTRODUCTION –

Pregnancy related AKI is associated with worsening of kidney function resulting in chronic kidney and end stage kidney disease with hospital and ICU stays. Incidence of AKI in pregnancy is 0.68% ie 68per 10 000 deliveries.Pre – eclampsia, sepsis & prerenal cause due to Hemorrhage & dehydration were most common cause for pregnancy related AKI. With decreasing incidences in developing countries but seemingly increasing incidences in the United states and Canada. The risk factors associated with advanced maternal age, such as hypertension, diabetes, chronic kidney disease, and those associated with reproductive technologies such as multiple gestations, are increasing. Traditional causes of Pr-AKI, such as septic abortions and puerperal sepsis, have been replaced by hypertensive diseases, syndrome (aHUS).

CASE REPORT-

26yr old G2P1 32wks pregnancy with previous LSCS presented to opd with complaints of upper abdomen pain. Backache & dysuria on & of for 2m. Supportive Rx was given. Urine R/M & C/S were normal. USG WA could not visualise renal system. At 34 wks pt again came with severe abdominal pain, loose motions was admitted. IV fluid, antibiotic & steroid was given. Blood reports and OBS usg were normal. Next day pt c/o tightness of abdomen so duvadilon drip was started. By evening pt c/o chest pain, restlessness. Tingling in both UL&LL. O/E severe uterine contraction with fetal bradycardia was noted. Emergency LSCS was done male baby 2kg delivered & shifted to nicu for mild birth asphyxia. Intra op all went well. Next day around 12pm D1-2 pt c/o chest pain, restlessness. ECG & tropI both were normal. Supported treatment was given at 7pm pt shifted to medical ICU with hypotension, tachycardia & oliguria. Pt was put on NA & Dopamine support with higher antibiotic. On investigation TLC 30,900 platlet count 76k, HB 10.9 sr bilirubin Liver enzymes sr creat 1.3 procalcitonin 84 (>2high risk for severe sepsis) coagulation profile wnl. By morning pt went in to anuria. Dialysis was advised by nephrologist. But with settling hypotension urine output 150cc came by 12pm D2-3 TLC went to 39k platlet count was 46k USG showed rt hydronephrosis.

Mild B/L pleural effusion & free fluid in peritoneum. With multidisciplinary approach involving obstetrician, physician, nephrologist, pathologist & radiologist pt was discharge on D8-9 with TLC 18k, platlet count 1.88 creatinine 1 renal usg was normal. This case has been reported to highlight the importance of timely management of near miss mortality and how normal physiological changes can turn to night mare.

DISCUSSION –

AKI in the general population is well defined by the RIFLE (Risk, Injury, Failure, Loss and End stage) criteria, the KDIGO (Kidney Disease Improving Outcomes) guidelines and the AKI Network (AKIN) criteria, pregnancy-related AKI is difficult to determine due to non-validation of these AKI diagnostic criteria in the pregnant population. Pregnancy is a state of increased CO/BV/Plasma volume/GFR and decreased Systemic vascular resistance/ Sr.creatinine.

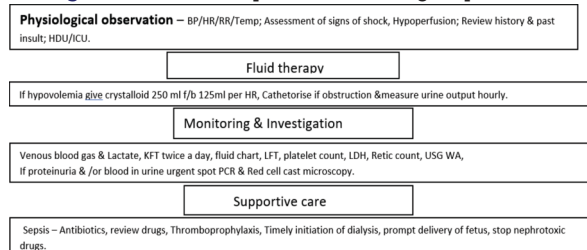
Classification of Pr- AKI according to trimester

1st trimester - Hyperemesis gravidarum, septic abortion.

2nd & 3 trimester / Postpartum - Pre-eclampsia, HELLP, TTP, HUS, AFLP, Lupus nephritis, UTI, Pyelonephritis, Obstructive, uropathy, Placental abruption & Hemorrhage.

Prerenal- Sepsis, Hypovolemia, PPH Renal - NSAIDs, PET, HELLP, TTP, HUS Postrenal - Obstructive uropathy, Uretric injury

Management - Potentially A Medical Emergency.



CONCLUSION -

AKI is not very common in pregnancy but if occurs should be managed with caution due to increase risk of maternal and fetal morbidity and mortality. Close monitoring and multidisciplinary approach is imperative for successful outcome.

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