SOMI MEET (08/07/2014)

Minutes of meeting:

CASE 1: DISCUSSION ON CHRONIC KIDNEY DISEASE

To summarise the case, this 37 year old chronic hypertensive, who did not have evaluation for secondary causes of hypertension or involvement of target end organs prior to IVF, hence missed opportunity for prepregnancy counselling for CKD, missed out another chance of diagnosis when a routine s.cr of 1.8mg/dl at the end of first trimester was not evaluated further. She developed early onset Preeclampsia and FGR (Fetal growth restriction) at 28wks with Doppler compromise.

She travelled from siliguri in West Bengal to Hyderabad for further management but found to have IUFD at admission with worsening of renal parameters with metabolic acidosis and hyperkalemia needing termination of pregnancy and expelled still born. Serial labs postpartum dint show any further worsening of renal parameters and as her vitals were stable, she was discharged after nephrologist consult, with a plan to evaluate further when her preeclampsia related changes subside initially at 6weeks and again at 3months postpartum. Her s. Creatinine and potassium can be repeated at 2weeks postpartum with physician review. She will require daily BP monitoring for atleast 1week, then weekly for 3months then monthly.

The final diagnosis is CKD with chronic hypertension and anemia of chronic disease with superimposed early onset preeclampsia and IUFD.

She will need a renal biopsy to know the staging of CKD and probable etiology, stabilisation of her risk factors (HTN, CKD) before she can plan another pregnancy with prior prepregnancy counselling regarding the effects of CKD on pregnancy and vice versa, fetal outcomes in CKD depending on the severity of CKD.

If this case had come prepregnancy, she would have been advised to do the following investigations for chronic hypertension:

CBC, CUE, 24 hour urinary protein and creatinine clearance, USG abdomen, renal artery Doppler, serum creatinine and electrolytes, FBS, PLBS, lipid profile, ECG +/-2D Echo (if ECG showed any changes)

She would have been referred to nephrologist for further evaluation of CKD, opinion regarding plan for pregnancy, titration of medication, ideal time to conceive etc. The couple would have been counselled regarding

1. Risk of FGR and preterm delivery, superimposed preeclampsia and worsening of renal function in pregnancy.
2. If the couple still would like to go ahead with plan for pregnancy, they would be advised to have ANCs and deliveries in a place experienced in dealing with high risk pregnancies and preterm infants (NICU care
3. The need for LDA through out pregnancy, need to switch antihypertensives from ACE inhibitors or ARBs to other safer drugs in pregnancy like alphadopa/labetalol or nifedepine SR.
4. Close monitoring of HTN (with a target BP of </= 140/90) and renal function, with daily BP, monthly s.Cr , S. Potassium and CUE.

5. ANCs monthly till 28weeks then fortnightly till 32weeks then weekly till 34-36wks, to deliver by 36-37weeks if all goes well.
6. If there is superimposed preeclampsia, pt would be monitored for FGR by serial scans and placental insufficiency by fetal dopplers from 26weeks.
7. If there is rapid worsening of renal function, would be monitored as in-patient with daily SGPT, platelets, Creatinine and potassium and a decision for delivery would have been taken with worsening fetal dopplers or nonreactive NSTs or fluctuating BPs or worsening labs or imminent symptoms.

If she had presented in the first trimester, still the chronic htn evaluation and evaluation for the cause of CKD could be undertaken with nephrologist consult. Renal biopsy can be done if needed upto 28weeks gestation as later it would be technically difficult.

Chronic kidney disease is defined as kidney damage for more than three months as evidenced by structural or functional abnormalities with or without decreased glomerular filtration rate (GFR) and manifested either as pathological abnormalities or kidney damage markers in blood or urine or in the imaging tests. A GFR of <60ml/ min/1.73m²body surface area for greater than three months per se also indicates chronic kidney disease.

Stages of Chronic Kidney Disease to guide managemnet

Stage	Description	GFR (ml/min/1.73m2)	management
1	Kidney damage with normal or increased	GFR >90;	Specific Rx, Rx comorbid conditions & CVD
2	Kidney damage with mild decreased	GFR 60-89;	Slowing rate of loss of kidney function
3	Kidney damage with moderate decreased	GFR 30-59;	Prevention & Rx of Complications
4	Kidney damage with severe decreased	GFR 15-29;	Preparation for renal replacement therapy
5	Kidney Failure	<15;	Renal Replacement therapy

Severity classification to guide pregnancy counseling

1. Mildly impaired renal function (Cr 1.4) – good outcome for pregnancy and renal disease
2. Moderately impaired renal function (Cr 1.4 – 2.8) – risk of progression of renal failure, increased fetal risk
3. Severe renal insufficiency (Cr > 2.8) – high fetal/maternal morbidity/mortality, low likelihood of successful outcome, pregnancy discouraged

CASE 2: JAUNDICE COMPLICATING PREGNANCY from Navi Mumbai sent by Dr. Sucheta

Case summary:

- 22yr old primi, presented at ?8months gestation, with one spike of high grade fever and suspected PPROM (later ruled out as pad was dry and no oligohydramnios), Started on ciproflox empirically inview of mild pyuria, with C/S pending.

- Developed tachycardia, hypotension, oliguria and thrombocytopenia on D3 of illness, admitted and treated for sepsis with IV fluids and ceftriaxone. Found to have elevated transaminases >10times normal, normal bilirubin, ALP, hypoalbuminemia with AKI (Cr-1.7). Dengue serological tests were negative.

- Transferred to MICU care under a physician; received vasopressors in view of septic shock. She had persistent oliguria despite frusemide infusion 40mg/hr, metabolic acidosis(treated with sodabicarb) and worsening RFT, LFT with hypoglycaemia on day 5 of illness. Later pt developed pulmonary oedema and received NIV (noninvasive ventilation) for 2 days followed by mechanical ventilation; she also received hemodialysis in view of excess positive fluid balance.

- Found to have Hep E positive; malaria, leptospira negative, hep A/E/B negative.ANA, ACL negative.

- Patient had preterm labour and SVD of still born AGA baby with (INR 1.8) prophylactic RDP and FFP transfusion. Had altered sensorium following delivery.

- 2 PRBC transfusions given and antibiotics changed to meropenem, teicoplanin, steroids in view of persistent hypotension and increasing leucocytosis, with sofa score of 15/24, worsening thrombocytopenia, creatinine, LFT. Procalcitonin was high (62.7). Urine and blood cultures were sterile. 2D echo showed hypokinetic LV; CXR was suggestive of ARDS.

- Later antibiotics switched to Colistin, Metronidazole, Caspofungin. On Day 7 sepsis was not resolving yet, but LFTs started improving. Started on plasmapheresis after which she showed rapid improvement over the next 3-4 days with AKI, thrombocytopenia, leucocytosis, hypotension resolving. Extubated on d11 and discharged on d19 with normal LFT, RFT and CBC

Discussion : diagnosis – most probably urosepsis+ Hepatitis E with septic shock with MODS (AKI, ARDS, thrombocytopenia, hypoglycaemia, metabolic encephalopathy) with HUS (as it responded to plasmapheresis) and preterm labour with still birth.

Since the patient first presented with fever and with past history of probable UTIs, hypotension in the absence of hemorrhage or volume loss, sepsis needs to be considered as the first diagnosis (thrombocytopenia, high procalcitonin levels and the rising leukocytosis are in favour of sepsis). With Hep E positive (assuming it is IgM antibody positive and not IgG as this information is not available from the case), rise in transaminases, can be explained. Even if it is not positive, prolonged septic shock can result in ischemic injury to the liver resulting in such elevations in transaminases including LDH. Hypoglycaemia is seen in any hepatic injury but not in HELLP. AFLP is a

possibility but DIC is more common and prominent in AFLP which is absent in this case. Altered sensorium can be explained by metabolic encephalopathy secondary to hypoxia, uremia, septic shock +/- hyperammonemia.

The only evidence backing for plasmapheresis is available for cases of HUS/TTP. Though the peripheral smear did not show evidence of hemolysis, raised LDH and drop in haemoglobin can be secondary to HUS. Urine for haemoglobin would also help in confirming intravascular hemolysis.

The group was not in consensus with the choice of antibiotics used (highest end being used)

Nephrologist felt there is no role of frusemide infusion and that too at such a high dose of 40mg/hr; infact it can sometimes be detrimental to renal function. When there is no response to intermittent doses of frusemide (40mg tid), dialysis should be considered especially with excess fluid balance resulting in pulmonary oedema. Also there is no role of sodabicarb supplementation in acute kidney injury, the cause has to be treated and dialysis should be helpful.

Post critical illness flaccid quadriparesis has often been observed but usually recovers rapidly with good nutrition and physiotherapy. Role of micronutrients?

Following are the answers to the questions sent along with the case details by primary consultant who treated this patient:

1. What did we miss in AN visits ?
    - Recurrent UTI in pregnancy warrants USG abdomen o rule out complicated UTI. If cultures are sterile despite persistent significant pyuria and perineal infections ruled out, genitourinary Kochs needs to be considered.
2. What should be considered as screening modality of UTI in pregnancy?
    - Urine C/S at 16wks (2^{nd} trimester) (RCOG standard of antenatal care)
3. Should bacteriuria $< 10^5$ be treated
    - Only if symptomatic with dysuria/fever ;
4. Was Ciproflox a bad choice to begin with
    a. We don't use in our set up as our antibiogram (urine C/S show70-85% resistance; 100% sensitivity to amikacin). If in your set up, you have E. coli sensitive to ciproflox, may be not a bad choice considering it is a category C drug in pregnancy.
5. Is there any role of antimalarials in all high grade fevers?

a. according to NMEP (national malaria eradication programme), empirical antimalarials are no longer indicated since the sensitivity of rapid antigen tests is 99%; hence antimalarials are to be atrted only if the tests are positive unless the patient is in a remote place where tests take longer time to do/reported. Avoiding empirical antimalarials will prevent development/spread of resistance.

6. Can HELLP be the diagnosis in absence of HT- HELLP can be in the differential diagnosis even in the absence of HTN but hypoglycemia and rise in transaminases to such an extent is not seen in HELLP

7. What if she did not go in spont labour? Physicians were recommending induction / relatives in retrospect were questioning about NOT doing LSCS.

   The group felt that fetal monitoring in the MICU of the pregnant lady should be aggressive as the maternal mileu was not favourable to the fetus (in view of hypoglycemia, metabolic acidosis, renal failure, septic shock, pulmonary oedema). She could have been taken up for delivery at an earlier stage even if HELLP and AFLP were not being considered, for the above reasons mentioned (maternal parameters) as baby was AGA and 1.7kg.The maternal and fetal outcomes in Hep E positive patients are better if fetus is delivered earlier rather than later with possible fetal immunogenicity contributing to further liver damage according to some studies. Hep E in Asians is considered to be more virulent in pregnancy compared to those in Egypt and other Mediterranean countries where they have better maternal and fetal outcomes. There is ~80% maternal mortality and morbidity associated with Hep E in pregnancy in our parts. Delivery in Hep E has to be considered when the patient is relatively better than when she is worse off.

   When the diagnosis is not clear and HELLP/AFLP are possibilities (3rd trimester acute hepatic failure, thrombocytopenia, hypoglycemia) delivery is always indicated as soon as possible.

8. Can we give uterotonics in ARF : yes, when indicated

9. Anesthesia?

   If platelets >80,000 regional anaesthesia si not contraindicated, if < 70,000 general anaesthesia is preferred.

10. Plasmapheresis … was it a right choice?

We wouldn't have done plasmapheresis in this case , but seeing that it helped in this case and patient appears to have improved only after starting plasmapheresis, the group felt, retrospectively, there may be an element of HUS secondary to sepsis which might have resulted in the improvement, as otherwise, plasmapheresis is not shown to be beneficial in AFLP/ Hep E except for a few case reports. We must start thinking of plasmapheresis in such cases.