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A Rare Case of Pregnancy with Nephritic Syndrome which can be Misdiagnosed as Preeclampsia

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Abstract

Pregnancy with hypertension, proteinuria, edema detected in third trimester is always preeclampsia unless proven otherwise[1]. We had a similar case with acute onset of hypertension, proteinuia, oedema, mild breathlessness along with frank hematuria at 32 weeks of gestation in a primigravida. She was initially diagnosed as preeclampsia with its complication either HELLP syndrome or DIC. Investigations showed sever anaemia, mild thrombocytopenia normal liver & kidney function normal coagulation studies without any evidence of sepsis. Surprisingly fetal parameters were absolulety normal. There was no evidence of Intrauterine Growth restriction, fetal, and uterine Doppler studies were normal. Renal Doppler showing paranchymal renal disease. Patient was stabilized in ICU with nasal Oxygen, diuretics, antihypertensive, antibiotics, steroids for fetal pulmonary maturity. In view of deteriorating maternal condition uncontrolled hypertension increasing hematuria urgent delivery by caesarian section done as bishops was very poor. Surgery went uneventful with outcome of male child of 1.7 kgs. Post surgery patient was hemodynamically stable hypertension well controlled but hematuria was persistent and fluctuant. Urine culture report was negative. Suspecting some renal pathology with nephritic syndrome like presentation, she was found to be ASO titer positive. Detailed history revealed pharyngitis 4-5 days before with altered voice since then. Repeat renal uitrasonography 15 days postdelivery showed same findings of altered corticomedullary differentiation, raised cortical echogenicity suggestive of medical renal disease with normal renal artery Doppler. Final diagnosis of nephritic syndrome was made and patient was discharged on day 16 with fluctuant hematuria. Hematuria completely cured over 3 months period.

Keywords: *hematuria, nephritic syndrome, renal Doppler.*

Case report

26 years primigravida with 32 weeks of gestation presented to emergency department with sudden increasing edema over lower extrimities and vulval edema with hematuria and slight breathing difficulty. She was not a diagnosed case of hypertension or any other systemic disease. There was a history of fever with sore throat 4- 5days back. On examination she was found to have tachycardia (pulse rate- 139/min), severe hypertension (160/110 mm of hg) with tachypnea (RR 30 /min), pallor, cardiovascular system was normal. On respiratory system examination fine basal crepts were noted. Obstetrics findings were corresponding to weeks of gestation. There was evidence of pitting type lower abdominal wall oedema, pedal oedema upto lower thighs, severe vulval oedema and frank hematuria. Considering provisional diagnosis of severe preeclampsia with its complications, she was treated with antihypertensives diuretics and steroids. As there were no premonitory signs or symptoms of eclampsia Inj. MgSO4 was withhold. Patient was kept in intensive care unit under strict monitoring.

On investigations found to have anaemia (Hb 7.2 gms %) , mild thrombocytopenia (plt count 82000/cmm), hypoalbuminemia with normal total and differential leukocyte count. Urine examination showed plenty of RBCS, protein 4+, with 4-5 pus cells with rbc casts . Renal function tests were slightly deranged with Sr. creatinine level of 1.4 mg/dl, 24 hours urinary protein of 1.2 gms. Liver function tests, coagulation studies were in normal range. Urine culture no growth noted. Obstetrics ultrasound showed single live intrauterine pregnancy with 32wks of gestation no evidence of intrauterine growth restriction with fetal wt of 1.68 kgs. Umbilical, uterine artery and middle cerebral artery Doppler studies were normal. Renal ultrasound was suggestive of raised cortical echogenicity with accentuated corticomedullary differentiation in both kidneys indicating renal medical paranchymal disease. Renal artery Doppler studies were normal. Even after receiving blood and platelet transfusion her hematuria and anaemia was persistent so decision of termination of pregnancy was taken. She was delivered by caesarian section with outcome of male child 1.7 kgs. There were no intra or post operative complications. Postdelivery her blood pressure responded well to antihypertensives but hematuria was persistent. Renal function returned to normal over period of 7 days. Anaemia and thrombocytopenia also reveresd. Only positive finding remaining was hematuria. Further investigations like Sr LDH, Lipid profile, CRP were normal. Sr ASO (antistreptolysin O) titre were slightly raised. Repeat renal ultrasound on day 15 post delivery suggestive of same findings of medical renal disease.

So final diagnosis was made as nephritic syndrome, patient was discharged after explained about self limiting and gradual course of disease and close follow up. On weekly follow up urine examinations showed gradual reduction in hematuria. It took almost 3 months for complete recovery.

Discussion

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. The World Health Organization systematically reviews maternal mortality worldwide. In developed countries, 16 percent of maternal deaths were due to hypertensive disorders. This percentage is greater than three other leading causes: hemorrhage—13 percent, abortion—8 percent, and sepsis—2 percent[1].

Preeclampsia : new onset of hypertension after 20 weeks of gestation with significant proteinuria(more than 300 mg over 24 hours).[1]

Severe preeclampsia – preeclampsia with severe hypertension > 160/110 and/ or with symptoms and /or biochemical and/ or hematological impairment.[1]

Acute nephritic syndrome (glomerulonephritis) - hematuria, hypertension, fluid retention, sustained proteinuria (1-2 grams over 24 hours) with red blood cell casts. Dysmorphic RBCs, raised sr. creatinine redued GFR and oliguria may be seen[2]*. Thin glomerular basement membrane with pores in podocytes large enough to permit protein & RBCs*[3]*. There are varying degrees of renal insufficiency and salt and water retention, which causes edema, hypertension, and circulatory congestion.*

Proteinuria in most adults with glomerular disease is non selective containing albumin and mixture of other proteins[2]*.*

Etiological factors for nephritic syndrome are[4]

1. *Poststreptococcal infection*
2. *Subacute bacterial endocarditis*
3. *Systemic lupus erythematosus*
4. *Antiglomerular basement membrane disease*
5. *IgA nephropathy*
6. *ANCA small vessel vasculitis*
7. *Henoch-Schonlein purpura*

Poststreptococcal glomerular nephritis is common in adults than other types. Following skin infection i.e post impetigo M type streptococci with subtype 47,49,55,2,60,57 are

observed. After pharyngitis M type streptococci with subtypeb 1,2,4,3,25,49,12 are observed. Symptoms are headache, malaise, anorexia, flank pain. 20 % of adults have proteinuria of nephritic range. ASO titres are raised in only 30 % of cases. Diagnosis rarely requires renal biopsy. Treatment is supportive with control of hypertension ,oedema. Dialysis may be needed. No role of immunosuppressive therapy. Overall prognosis is good. Complete resolution of hematuria and proteinuria occurs within 3-6 weeks of onset[5].

Acute poststreptococcal glomerulonephritis is prototypical of these syndromes. Although it rarely develops during pregnancy. Diagnosis is confirmed by history of streptococcal infection within previous weeks and/ or raised ASO titres. Fetal loss is almost invariable renal function returns to normal after delivery[6].

References

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