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ATYPICAL PRE-ECLAMPSIA

By

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Atypical presentations in hypertensive disorders of pregnancy.

INTRODUCTION:

Hypertensive disorders are the most common medical disorders in pregnancy with incidence independent of environmental and ethnic influences. They rank second in maternal death annually. Incidence of pre-eclampsia is 5-10% throughout the world. It remains the leading cause of maternal and perinatal morbidity and mortality worldwide. Classically most women develop pre-eclampsia hypertension and proteinuria after 20 weeks of gestation up to 48 hours postpartum. Recent data suggest that in some women, pre-eclampsia and even eclampsia may develop in the absence of hypertension and proteinuria.
Here we report 6 atypical cases encountered in Jeyasekharan Hospital.

Problems with atypical forms of eclampsia is its unpredictable onset. Timely diagnosis and management are critical in avoiding complications.

CASE 1:

KEY POINTS

30 year old, married for 4 years

G2A1

Known case of type 2 diabetes mellitus, post CABG

IUD at 26 weeks in 1st pregnancy

Incidental abnormal doppler at 26 weeks

Absent end diastolic flow

Normal BP and patient blood parameters

Rise in BP 2 days after doppler findings

2 weeks later emergency LSCS

Because of reversal of flow in doppler

- Mrs. X, 30 year old 2nd gravida, booked case came for routine antenatal checkup.
- She is a case of type 2 DM for the past 2 years
- Post coronary artery bypass graft for single vessel block done 2 years back
- Married 4 years with H/O induced abortion at 24 weeks for IUD.
- She had pregnancy induced hypertension during first pregnancy, was on antihypertensive and developed uteroplacental insufficiency as early as 24 weeks
- II trimester expulsion of dead baby was done 1 year back
- Post expulsion, she was investigated for APLA and all investigations were normal
- After an year she conceived again and was under very close follow up.
- We had to terminate her pregnancy at 26 weeks. Since BP was very high and IUD confirmed by scan
- No H/O increased blood pressure during routine check up and she was on prophylactic aspirin and haematinics.
- Again APLA and other investigations were normal
- Dating scan, NT scan, first trimester screening and anomaly scan was normal

- At 26 weeks, she underwent follow up USS (for foetal echo) and at that time she had absent end diastolic flow with cord round neck with a gestational age of 26 weeks, adequate liquor and a baby weight of 791 gm.
- At that time also her BP was normal and there was no proteinuria
- LFT and RFT normal
- Since she was a high risk patient we admitted her.
- 2 days after admission, she developed hypertension and antihypertensives (Labetalol) was started.
- She was given conservative management for 2 weeks in hospital with close monitoring
- After 2 weeks, foetal doppler showed reversal of flow with foetal distress and her BP was also high with proteinuria
- Antenatal corticosteroids were given and IV magnesium sulphate for neuroprotection was also given
- We terminated the pregnancy by emergency LSCS and an extreme preterm alive boy baby of wt 800 gm was delivered and baby was admitted in Neonatal ICU.

PERSONAL COMMENTS:

We usually suspect doppler finding after (clinical features like), increase in BP, oligohydramnios. In this case the sequences occurred retrospectively. After the incidental absent end diastolic flow, she had increase in BP with no other features. We started her on antihypertensives. In two weeks time, her doppler findings showing reversal of flow. Since she had reduced foetal movements and with abnormal doppler we took her for emergency LSCS

CASE 2:

KEY POINTS

Primi

Reduced foetal movements

Altered liver enzymes with no other abnormalities, normal BP

Sudden convulsions followed by increased in BP and proteinuria

In spite on MgSO4 therapy another episode of convulsion

Fetal bradycardia, NST – persistent variable deceleration

Emergency LSCS

- Mrs. Y. a primi at 30 weeks, came to us with history of reduced foetal movement for 2 days.
- She was an unbookedcase and her BP was normal at the time of admission
- She was stable, but her liver enzymes were altered
- USG confirmed – 30 weeks intrauterine gestation with normal doppler
- 2 days later, she developed convulsion followed by increase in BP and proteinuria
- Infection magnesium sulphate was given for eclampsia
- I/V labetalol and antenatal corticosteroids given
- While she was on Inj. Magnesium sulphate again she developed convulsions
- Hence emergency LSCS done and delivered a preterm girl babyof wt1.02 kg. Baby was admitted in NICU.

- Postoperatively, mother did not have any seizures and her BP also came back to normal in 2 weeks.

PERSONAL COMMENTS

We saw this unbookedprimi who came to us with reduced foetal movements. We evaluated her and we found out increased levels of liver enzymes. All other parameters were normal. Since there was reduced foetal movements we admitted her for close follow up. We placed her on continuous foetal monitoring. On the second day of admission, she suddenly had convulsions followed by increase in BP and proteinuria. We started her on antihypertensives and MgSO4. In spite on MgSO4, she had further episode of convulsions with foetal bradycardia followed by persistent variable deceleration. We took her for emergency LSCS.

CASE-3:

KEY POINTS

G2 A1@37 weeks

Came in labour

BP normal throughout pregnancy

Trace proteins in urine examination at last visit

NVD –4 hours later she had seizures

Increased BP and increased liver enzymes

Platelets marginally reduced

Creatinine raised

- Mrs. X a 20 year old 2nd gravida with abortion – a booked case with regular routine antenatal check up came to us with abdominal pain at 37 weeks.
- Her BP was normal throughout her antenatal check up except for trace protein in urine examination during last check up.
- Her liver function test and renal function test were normal
- She progressed normally and delivered a female baby of wt 3.10 kg normally
- 4 hours after delivery, she developed seizures and after 15 minutes, her BP was 150/100 mmHg
- Injection magnesium sulphate and I/V labetalol was given.
- At that time liver enzymes were increased
- MRI venogram was normal
- Platelets were marginally reduced
- Creatinine was raised

- With magnesium sulphate regime and antihypertensives, BP was under control
- Discharged with oral antihypertensives
- 1 week later, LFT, RFT, and platelets returned to normal
- Tapering of antihypertensives was done.

PERSONAL COMMENTS

We had this lady with uneventful antenatal period who had normal vaginal delivery. Suddenly after 4 hours, she had convulsions with rise in BP, liver enzymes, creatinine and reduced platelets. We started her on antihypertensives and tapered her dose and all her parameter were normal after 1 week

CASE 4:

KEY POINTS

25 years primi

IUGR, Oligamnios

Headache, blurring of vision

Increased BP with proteinuria

Elevated liver enzymes, low platelets, reduced urine output

Emergency LSCS

- Mrs. Y., a 25 year old primi was admitted at 32 weeks with IUGR
- She was an unbooked case with regular ANC outside and till 30 weeks. Her vitals and USG were normal
- At 30 weeks, USG revealed IUGR and oligamnios and she was referred to us.
- At the time of admission, BP and all investigations including LFT, RFT were normal
- BP was normal at the time of admission
- No complaints of headache or blurring of vision
- 3 days later, her BP was 140/90 mmHg. with proteinuria
- Investigations revealed abnormal LFT with low platelets
- Urine output was also less
- A diagnosis of HELLP syndrome was made and emergency LSCS done.
- Baby admitted in NICU.

PERSONAL COMMENTS

This primi was referred to us with IUGR and oligohydramnios at 30 weeks. At the time of admission, her BP and all the blood parameters were normal. 2 weeks later, she had increased BP with proteinuria with altered LFT, reduced platelets and reduced urine output. We arrived at a diagnosis of HELLP syndrome and took her up for emergency LSCS.

CASE 5:

KEY POINTS

4^{th} gravida, 32 weeks

Reduced foetal movements

H/O BOH

Neck pain, headache

Seizures

Increased BP

Emergency LSCS

- Mrs. Y., 32 year old 4^{th} gravida
- Unbooked case come to us at 32 weeks gestation with decreased foetal movements
- First was spontaneous abortion
- Second was inevitable abortion
- Third – IUD at 28 weeks

- During this pregnancy, she was having ANC outside. She was investigated for APLA and was normal.
- According to her BP was normal throughout and her ANC.
- Considering her BOH, we admitted her
- Her BP was normal throughout admission
- She had complaints of neck pain, but no headache
- At that time also her BP was normal and no proteinuria was there
- LFT and RFT normal
- USG normal
- On 5th day of admission, she developed headache for ½ an hour
- Was shifted to labour room and she developed generalized seizures
- At that time BP was 180/100 mmHg
- Since there was foetal heart variation
- Emergency LSCS was done
- Delivered a preterm boy baby of 1.42 kg
- Baby was admitted in NICU
- After LSCS BP returned to normal and no further episode of seizures
- 2 years later, she conceived again and delivered a term baby at 38 weeks with no history of PIH or eclampsia.

PERSONAL COMMENTS

We had this 4th gravida who came to us at 32 weeks with reduced foetal movements. Her initial antenatal check up were outside. Her records showed normal BP and normal blood parameters throughout her pregnancy. In view of BOH, APLA profile done which was normal. We admitted her for continuous foetal monitoring and on 5th day of her admission, she had neck pain and headache followed by generalized seizures. Then she had increased BP with foetal heart rate variation and underwent emergency LSCS.

CASE 6:

KEY POINTS

Primi, 34 weeks

MCDA twins

Moderate anaemia

Epistaxis

Increased BP

Mrs. X, primi at 34 weeks, MCDA twins with moderate anaemia

Unbooked case, referred from outside with first episode of epistaxis

Previous ANC – normal according to patient

No H/O increased BP or she was not on medications

Was admitted and was having recurrent attacks of epistaxis for which conservative management was given

Next day – BP was 140/90 mmHg

LFT and RFT normal

Antihypertensives started

USG and doppler normal

Antenatal corticosteroids given

No proteinuria and all other investigations normal

Another bout of epistaxis on 3rd day

BP was high in spite of antihypertensives

She developed headache

She underwent emergency LSCS was done

Two alive preterm babies were delivered

Postoperatively, she did not have epistaxis

BP also became normal after 1 month.

PERSONAL COMMENTS

We had this primi who was referred from outside at 34 weeks with first episode of epistaxis. Her antenatal records were normal. We admitted her since she had recurrent attacks of epistaxis. The next day after admission, her BP was 140/90 mmHg with normal RFT, LFT and USG. We started her on antihypertensives and in spite of that there was epistaxis and she was taken up for LSCS followed which her BP returned to normal and there were no further episodes of epistaxis.

DISCUSSION:

- Classic triad of preeclampsia is hypertension, proteinuria and oedema.
- Approximately 1/3 of eclamptic women do not develop oedema
- In recent years the new term atypical preeclampsia is used to describe non-classical forms of hypertensive disorder
- Atypical eclampsia constitutes about 8% of eclamptic cases
- Atypical preeclampsia is defined as any clinical presentation of preeclampsia <20 weeks of gestation and >48 hours after delivery.
- Also in variability as described in the following table

TABLE:

ATYPICAL PREECLAMPSIA:

Gestational hypertension and one of the following items:- Symptoms of preeclampsia: 1. Hemolysis 2. Thrombocytopenia (less than 1 lakhs/mm^3) 3. Elevated liver enzymes (2 times the upper limit of the normal value for aspartate aminotransferase or alanine aminotransferase).

Gestational proteinuria + 1 of the following items:- Symptoms of preeclampsia 1. Hemolysis 2. Thrombocytopenia 3. Elevated liver enzymes 4. Early signs and symptoms of preeclampsia – eclampsia at <20 weeks. 5. Late postpartum Preeclampsia – eclampsia >48 hours of delivery

When this clinical presentation is described – capillary leak syndrome should also be known.

Capillary syndrome is ascites, pulmonary oedema, proteinuria associated with multiogran dysfunction.

In classic disease usually first involves the arteries and kidneys manifesting as hypertension and proteinuria before other systems are involved.

In atypical cases however the organ involvement may start with other symptoms such as cerebral involvement which present initially as eclampsia

Suspicion findings may include marginally elevated BP or liver enzymes, foetal distress, blurred vision and headache.

CONCLUSION:

The purpose of this review is to increase the awareness of atypical forms of hypertensive disorder of pregnancy

The classic teaching that eclampsia is the end point of a disease process. Starting subclinically and proceeding to mild preeclampsia and then severe preeclampsia implies that hypertension and proteinuria should precede the onset of eclampsia

In contrast to this paradigm eclampsia can potentially be encountered at the beginning of the disease process before hypertension and proteinuria develop.

Eclampsia, foetal distress may be on unusual presenting scenario in atypical cases before overt hypertension or proteinuria

Even minor clues in diagnosis such as a marginally elevated BP

or trace of proteinuria

or headache/neck pain

or blurring of vision

or gastritis

or epistaxis

May be critical for appropriate timely management

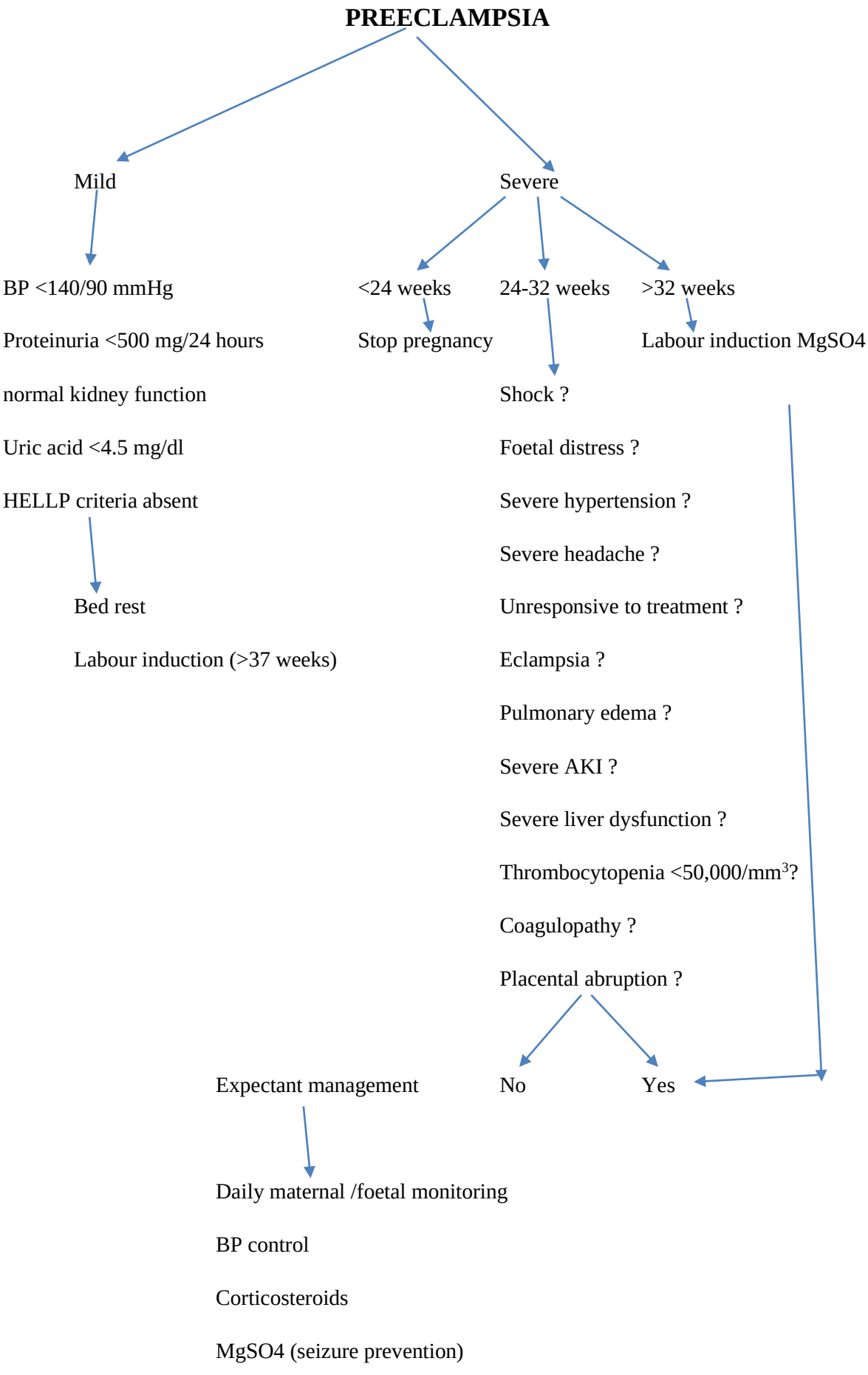
The famous quote

“The eyes cannot see what the mind does not know”holds good in this situation

Obstetricians should be aware of atypical presentations maintain a high level of suspicion and be ready to take immediate steps.

We should be alert in dealing with each and every patients and a close monitoring is essential even for normal looking patients

These women should be seen frequently at least twice weekly. Rather than relying solely on the presence of hypertension or proteinuria, the patient history, physical examination, and laboratory and imaging studies may be critical in not missing atypical forms.


PREECLAMPSIA
Mild
Severe
BP <140/90 mmHg
Proteinuria <500 mg/24 hours
normal kidney function
Uric acid <4.5 mg/dl
HELLP criteria absent
Bed rest
Labour induction (>37 weeks)
<24 weeks
24-32 weeks
>32 weeks
Stop pregnancy
Labour induction MgSO4
Shock ?
Foetal distress ?
Severe hypertension ?
Severe headache ?
Unresponsive to treatment ?
Eclampsia ?
Pulmonary edema ?
Severe AKI ?
Severe liver dysfunction ?
Thrombocytopenia <50,000/mm3?
Coagulopathy ?
Placental abruption ?
Expectant management
No
Yes
Daily maternal /foetal monitoring
BP control
Corticosteroids
MgSO4 (seizure prevention)