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ABSTRACT
In rural areas obstetric care is mainly provided by general physicians, and it is important for physicians to stay up to date with the latest obstetrics guidelines. Preeclampsia is one of the well known hypertensive disorder related to pregnancy and guidelines for diagnosis and treatment are also well established. However, non classical forms of preeclampsia have been less established. As these cases are rare and less studied, its classification, presentation, constituents and when to be concerned with is an important point to be considered for better management and recovery of these patients.

In this article, we focused on a 21 yr old pregnant female who was not a classical case of preeclampsia i.e. without the presence of gestational hypertension and proteinuria. But this patient developed eclampsia and HELLP syndrome in immediate post-partum period. According to recent data, now a days cases are being reported where pregnant females are presenting with atypical form of preeclampsia and even eclampsia. These cases are of more concern, as because of their atypical presentation there occurs a delay in diagnosis and management leading to life threatening complications there by increasing maternal morbidity and mortality.

INTRODUCTION
Preeclampsia is a multisystem disorder unique to human pregnancy. It is characterized by diffuse endothelial dysfunction with maternal complications including placental abruption, pulmonary oedema, acute renal failure, liver failure, stroke, and various neonatal complications. Clinically, preeclampsia is defined as development of hypertension after 20 weeks of gestation, with the presence of proteinuria. The incidence is estimated to be between 3-10% of all pregnancies. Worldwide, preeclampsia and related conditions are among the leading causes of maternal mortality. Recently, a new term atypical preeclampsia- eclampsia has been used to describe non classical forms of hypertensive disorder during pregnancy. Although, there is no strict definition of this atypical variety; it has to include cases with minimal or no proteinuria but with hypertension; or proteinuria with no or marginally elevated blood pressure; or without hypertension and proteinuria; or presentation before 20 weeks or more than 48 hrs postpartum and also include cases those resistant to MgsO4 or associated with haemolytic anaemia, elevated liver enzymes, and low platelet count (HELLPsyndrome) and its variant.

Due to its atypical form and unpredictable onset; it is thus difficult to make timely diagnosis to initiate management, which is found to be critical in avoiding complications and decreasing maternal morbidity and mortality related to these atypical presentations of hypertensive disorder during pregnancy.

CASE REPORT
A 21 yr old primigravida was admitted to antenatal ward of obstetrics and gynaecology for routine check up, as she was 5 days post-dated according to her documented expected date of delivery. Her antenatal course was unremarkable and remained normotensive throughout this period. However, she complained of severe headache and epigastric pain 2 to 3 days prior to hospital admission with marginal rise of blood pressure. All laboratory investigations including haemoglobin, platelets were within normal limits and rubella IgG and virology were also normal. Urine analysis showed no proteinuria during antenatal visits.

Emergency lower segment caesarean section was conducted on second day of hospital admission for oblique lie and cephalopelvic disproportion.

Pre-operative period was uneventful and her vitals were also within normal limits. Caesarean section was performed under spinal anaesthesia. Intra-operatively her blood pressure was within normal range but she complained of headache and severe epigastric pain which was managed with mild sedoanalgesia.

Immediate post-operatively, her blood pressure started decreasing which was managed with vasopressors and then there was sudden rise of blood pressure up to 170/110mmHg. This sudden rise of blood pressure was followed by generalized tonic-clonic seizure which lasted for about 2 minutes accompanied with frothing from mouth, up rolling of eyes and rise of temperature.

Seizure was controlled with inj. MgSO4, inj. Propofol and Midazolam and she was shifted to central ICU for further management. On admission to ICU, she had one more episode of seizure. She was drowsy since first fit. During this period her blood pressure remained towards higher side around 160/100mmHg without any pressor support with gradual fall from day 1 postpartum. Her laboratory Workup for Day 0 postpartum showed Hb - 14.6 g/dl, Serum creatinine – 1.7 mg/dl, Platelet counts –80,000/mm³, serum potassium – 3.5mmol/L.

Gradually, her consciousness deteriorated and she developed AKI (Acute kidney injury) and crepitations in chest (Pulmonary oedema) with deterioration of her saturation and features of HELLP syndrome for which she was intubated on day 1 postpartum. Her laboratory Workup for day 1 postpartum were as follows: Hb – 9.2 gm/dl, platelet count – 80,000/mm³, AST – 4341units per litre, ALT – 1351units per litre, S. Creat – 3.9mg/dl, S. Potassium – 5.4mmol/L, Total bilirubin - 1.81mg/dl.

Her CT scan brain was done which showed – Hypodense area involving cortical and subcortical areas of frontal and posterior parietal region. There was no evidence of...
intracranial haemorrhage, cerebral infarction, or cerebral venous thrombosis. Her further investigations and MRI couldn’t be done as patient started deteriorating rapidly and developed cardiac arrest on day 4 postpartum from which she could not be resuscitated back.

DISCUSSION
American college of obstetricians and Gynaecologists considered hypertension as most important factor, which is required for the diagnosis of preeclampsia with either proteinuria, end organ dysfunction or both. In contrast to this, hypertension and or proteinuria may be absent in 10-15% of preeclamptic patients.

Atypical preeclampsia comprises 4 clinical groups. Nonproteinuric gestational hypertension or symptoms or laboratory signs suggestive of microangiopathy/haemolysis; normotensive gestational proteinuria with the presence of symptoms or laboratory signs suggestive of microangiopathy/haemolysis; normotensive and non proteinuric with symptoms or laboratory signs suggestive of microangiopathy/haemolysis; presence of preeclampsia - eclampsia or HELLP syndrome after 48hrs postpartum and before 20 weeks of gestation.

Our case developed severe features of preeclampsia due to delay in diagnosis and absence of target management. Severe features are defined as follows; systolic blood pressure of at least 160mmHg or a diastolic blood pressure of at least 110mmHg, new onset cerebral or visual disturbances, severe epigastric pain, thrombocytopenia (100000/mm3), s. creatinine> 1.1mg/dl or doubling of s. creat), pulmonary oedema, altered liver enzymes (AST,ALT) two times the upper limit of normal.

This patient was normotensive and non proteinuric throughout her antenatal period. At 37 weeks gestational age, 2 to 3 days before delivery she developed symptoms similar to severe features of preeclampsia. Her laboratory test were within normal limits but her symptoms persisted intraoperatoratively also with ultimately development of complication of preeclampsia i.e. postpartum eclampsia, HELLP syndrome, new onset cerebral involvement with acute kidney failure and pulmonary oedema. She was diagnosed to be a case of atypical preeclampsia with severe complications. It is not very clear and established, whether this patient would not have developed eclampsia and HELLP syndrome if her physician had diagnosed this case beforehand as non classical form of preeclampsia and started the treatment according to the guidelines. This case elaborates the need for future research and revision of classification of hypertensive disorder related to pregnancy. Vigilant monitoring, awareness of the symptoms and active approach towards the management may help preventing development of serious complications and decreasing maternal morbidity and mortality.

In women with postpartum eclampsia and HELLP syndrome, risk of renal failure, pulmonary oedema, new onset cerebral involvement is more. Early postpartum administration of high dose corticosteroids might accelerate recovery in these patients. Due to already vasoconstricted intravascular volume, too little fluid administration during perioperative period can lead to acute renal injury in these patients. Therefore, adequate fluid management also plays a vital role in recovery of these patients.

PIH patients remains in a vasoconstricted state and are highly sensitive to vasopressor agents. As our patient was also an atypical preeclampsia case, the use of vasopressor in immediate postpartum period lead to sudden rise of blood pressure. Therefore, in these type of patients there should be careful monitoring of vitals and judicious use of drugs to prevent complications.

Women with postpartum eclampsia and HELLP syndrome who deteriorate progressively with rise of bilirubin and creatinine after delivery may benefit from plasma exchange with fresh frozen plasma. In cases with persistent haemolysis, thrombocytopenia, hypoalbuminemia post partum blood product substitution, as well as albumin supplementation are standard regimen.

These patients with HELLP syndrome are also at higher risk of developing DIC. Therefore, there comes the role of platelet transfusion, plasma transfusion and use of antithrombotic agents can be explained here. Vigorous and early treatment of eclampsia with MgSO4 and control of blood pressure and emergency management of these cases in intensive care unit also play a major role in further treatment of patients with HELLP syndrome associated with eclampsia with new onset cerebral involvement.

CONCLUSION
In summary, we have described an atypical case of preeclampsia which developed complications during immediate postpartum period. Because of its occult type of presentation there was a delay in diagnosis and increase in complication with increase in maternal morbidity and mortality. We therefore, recommend taking clinical symptoms also into consideration for diagnosis of preeclampsia as a major factor to prevent complication and to decrease morbidity and mortality related to this by early suspicion, vigilant monitoring, detailed laboratory work up and awareness among general physicians and also among general public about this type of presentation for better management and recovery.

CONFLICTS OF INTEREST
There are no conflicts of interest.

REFERENCES
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