Section: Critical Care

Case Report

LATE POSTPARTUM DENOVO PRE-ECLAMPSIA COMPLICATED WITH ATYPICAL POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN IVF PREGNANCY: AN INTERESTING CASE REPORT

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ABSTRACT

Background: Atypical posterior reversible encephalopathy syndrome (PRES) is an uncommon but significant neurological condition. Its association with late postpartum pre-eclampsia occurring without prior signs of hypertension (Denovo) in in vitro fertilization (IVF) pregnancies is extremely rare and awareness of this entity shall help clinician in early diagnosis and management of this life-threatening illness.

Case Presentation: A 32-year-old woman, three days postpartum following a twin delivery through IVF, presented with generalized tonic-clonic seizures and altered mental status. She had no history of hypertension or pre-eclampsia during pregnancy or the immediate postpartum period. On admission, her blood pressure was 200/110 mmHg. Magnetic resonance imaging revealed symmetrical vasogenic edema in the bilateral parieto-occipital lobes, consistent with PRES. Laboratory investigations showed mild elevation of lactate dehydrogenase and trace proteinuria, with otherwise normal liver, renal, and coagulation profiles. The patient was diagnosed with late postpartum de novo pre-eclampsia complicated by PRES. The patient received intravenous antihypertensives, magnesium sulphate, and supportive care. Her condition improved within three days, with complete resolution of neurological symptoms and radiological findings. She was discharged on oral antihypertensives and advised regular monitoring.

Conclusion: This case highlights the importance of recognizing atypical presentations of pre-eclampsia and PRES, especially in IVF pregnancies. Early diagnosis and prompt management are critical for preventing severe complications and achieving favorable outcomes. This is a rare report of PRES in the context of late postpartum de novo pre-eclampsia following IVF, underscoring the need for heightened clinical awareness.

Keywords: Posterior reversible encephalopathy syndrome (PRES), Late postpartum preeclampsia and eclampsia; In vitro fertilisation (IVF), magnetic resonance imaging (MRI), Generalized tonic-clonic convulsions (GTCS), Hypertensive emergency.

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a characterised by a combination of clinical and radiological features. Patients typically present with a range of nonspecific symptoms, such as headaches, nausea, visual impairments, changes in mental status, seizures, and, in severe cases, loss

of consciousness. Imaging studies frequently reveal sub-cortical vasogenic oedema, particularly affecting the parietal and occipital lobes bilaterally.[1] Pre-eclampsia and eclampsia are recognised as one of the leading causes of PRES.[2]

Pre-eclampsia and eclampsia most commonly occur between 20 weeks of gestation and the first 48 hours after delivery. However, a smaller proportion of cases develop between 48 hours and four weeks postpartum, referred to as late postpartum pre-eclampsia or eclampsia. This late-onset form represents less than 16% of all postpartum eclampsia cases, and may occur without any evidence of hypertension in antenatal, intrapartum and in immediate post-partum period after 48 hours (de-novo pre-eclampsia).[3] The exact cause of this complex and variable syndrome remains unclear; however, several risk factors have been associated with its development. These include advanced maternal age, multiple pregnancies, polycystic ovary syndrome (PCOS), systemic lupus erythematosus (SLE), obesity, pre-existing hypertension, and diabetes.[4] Many of these risk factors are frequently observed in infertile patients undergoing in-vitro fertilisation (IVF) treatment, which partially explains the increased prevalence of pre-eclampsia in pregnancies conceived through IVF.[5,6]

The impact of initiating low-dose aspirin therapy during the second trimester of pregnancy on preventing pre-eclampsia has been widely researched. While large randomised controlled trials and meta-analyses have generally demonstrated minimal preventive benefit in all pregnant individuals, a study by Lambers et al. highlighted that in patients undergoing IVF starting low-dose aspirin treatment significantly reduced the incidence of hypertensive complications during pregnancy.[7] The American College of Obstetrics and Gynaecology also recommends low-dose aspirin initiated between 12 weeks and 28 weeks of gestation and continued daily until delivery for IVF pregnancies.[8]

In over 90% of cases involving delayed postpartum eclampsia seizures, there is typically at least one early symptom indicative of pre-eclampsia such as headache, blurring of vision or vague neurological symptoms. However, most patients fail to report these symptoms to healthcare providers. Consequently, fewer than 22% of these patients are accurately diagnosed with pre-eclampsia.[9,10] This oversight leads to missed opportunities for eclampsia prevention and significantly increases the likelihood of developing PRES. We report a rare instance of late postpartum de novo preeclampsia complicated by atypical PRES in an IVF pregnancy, highlighting the need for vigilance in postpartum care, even in patients without prior hypertension or preeclampsia.

Case Description

A 32-year-old postpartum female presented to the emergency department three days after delivering twin babies via lower segment caesarean section (LSCS) at a nursing home. Her pregnancy was achieved through IVF, and the surgical course was uneventful. She had no history of hypertension, diabetes, neurological disorders, or psychiatric illness before or during pregnancy. The patient was asymptomatic and discharged two days post-surgery. On the third day post-partum, she developed headache, blurred vision, and generalised uneasiness accompanied by mild breathing difficulty. She sought medical advice and was prescribed paracetamol with instructions to follow up in the outpatient department. Within hours, she experienced generalised tonic-clonic seizures (GTCS) followed by altered sensorium and was rushed to the emergency department of our hospital. On detailed history, it was found that she did not receive aspirin prophylaxis.

At admission, her vital signs were notable for a blood pressure of 200/110 mmHg, pulse rate of 108 beats/min, respiratory rate of 26 breaths/min, temperature of 39°C, and oxygen saturation (SpO_2) of 88% on room air. The Glasgow Coma Scale (GCS) score was E2M5V3, with bilaterally reactive pupils and flexor plantar responses. Auscultation revealed bilateral basal crepitations. She was positioned upright, and oxygen supplementation was initiated at 5 L/min, improving SpO_2 to 98%. The patient received a loading dose of intravenous (IV) levetiracetam (1 g) and magnesium sulphate (5 g), followed by continuous magnesium infusion and IV levetiracetam.

Laboratory findings revealed haemoglobin of 10 g/dL, a platelet count of 2.2 lakh/mm^3, and a total leucocyte count of 10,790/mm^3 with 80% neutrophils. Lactate dehydrogenase was mildly elevated at 410 U/L, and urinalysis showed trace protein and 3–4 pus cells per high-power field. Capillary blood glucose was 130 mg/dL. Liver and renal function tests, as well as coagulation studies, were within normal limits. (Table1) Fundus examination showed no papilledema.

Magnetic resonance imaging (MRI) of the brain revealed symmetrical, patchy, and confluent hyperintensities on T2-weighted and FLAIR sequences (Fig. 1.a, 1.b, 1.c, 1.d) involving the cortical/subcortical regions and deep white matter of the bilateral fronto-parieto-occipital lobes, including the centrum semiovale, corpus callosum, and left basal ganglia. There was no evidence of acute infarction or intraparenchymal haemorrhage (Fig. 1.e). Diffusion-weighted imaging showed no diffusion restriction, ruling out cerebral infarction. Magnetic resonance venography was normal (Fig. 1.f), excluding cerebral venous thrombosis. These findings were consistent with atypical PRES. Based on her clinical presentation and above MRI findings, a diagnosis of late postpartum preeclampsia complicated by atypical PRES was considered, and aggressive blood pressure management was initiated.

Blood pressure was gradually reduced to 150/90 mmHg within 24 hours using IV labetalol, followed

by oral antihypertensives for maintenance. Pulmonary oedema was managed with diuretics, and IV dexamethasone was administered to reduce intracranial pressure. The patient showed significant clinical improvement by the third day, with the resolution of headache and visual symptoms and no recurrence of seizures. Respiratory distress also resolved, and she maintained normal oxygen saturation on room air. Blood pressure was well-controlled with oral antihypertensives, allowing discontinuation of IV labetalol.

The patient was discharged on day six with advice for regular blood pressure monitoring, recognition of warning symptoms, and a follow-up MRI to confirm radiological recovery. A repeat MRI demonstrated complete resolution of the lesions, further supporting the diagnosis of PRES.

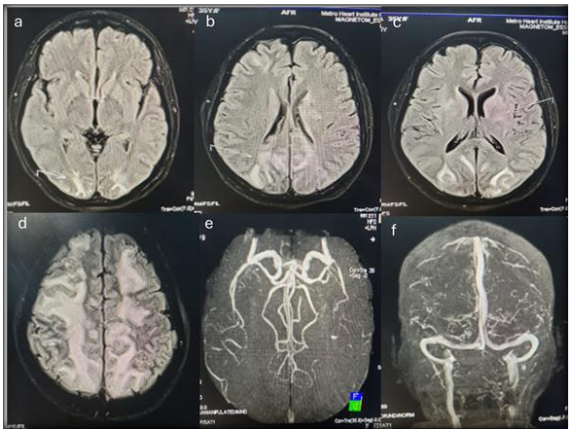

Figure 1: Imaging findings suggestive of Posterior reversible encephalopathy syndrome (PRES) in this case: Axial FLAIR weighted MRI image (a, b, c) and T2 weighted MRI image (d) showing high signal intensity of the cortical/subcortical location and deep white matter of bilateral frontal-parietooccipital lobes with involvement of the centrum semiovale, corpus callosum and patchy involvement of the left basal ganglia; MR angiography (d) showing no acute infarcts; MR venography (e) not showing any venous thrombus.

Table 1: Laboratory parameters on admission of our patient

Parameter	Reference range	DAY 1
Hb (gm/dl)	13.0-17.0	10
WBC Total	4000-10000	10790
Neutrophils		80%
Platelet Count (mm^3)	1.5-4.1	2.21
PCV %	40-50	31.2
MCV fl	83-101	81.9
Urea	19.2-42.8	29
S. Creatinine mg/dl	0.66-1.25	0.7
S.Na*mmol/L	135-155	137
S.K*mmol/L	3.5-5.5	3.7
PT (INR)	12.4-14.8/0.89-1.13	0.82
S. BIL Total mg/dl	0.2-1.3	0.6
S. BIL Direct mg/dl	0.0-0.4	0.2
S. BIL In Direct mg/dl	0.0-1.1	0.4
SGOT		
SGPT U/L	9 - 52	
Urine Glucose	Negative	Negative
Urine Ketones(mg/ dl)	0-<5	Negative
Urine Glucose	Negative	Negative
Urine Ketones(mg/ dl)	0-<5	Negative
LDH		410
Urine protein		Trace
PH %	7.35-7.45	7.483
PaCO2 MMHg	35-45	26
HCO3 - (mmHg)	22-26	19
Anion gap (mmol/L)	8-12	18
Blood glucose (mg/dl)	70-100	130
Sodium (mmol/L)	135-155	137
Serum lactate (mmol/L)	0.50-1.50	1.14

DISCUSSION

PRES typically manifests with a range of non-specific symptoms, including headache, vomiting, visual disturbances, altered mental status, seizures, and loss of consciousness, with seizures and headaches being the most frequent presentations. Seizures are often generalised. Visual impairments are also common and may include blurred vision, hemianopia, visual neglect, or cortical blindness.[1] These symptoms may develop acutely or progress gradually over several days.[11] Some patients also exhibit reduced or asymmetrical muscle strength in the limbs, although tendon reflexes generally remain active.[12]

The etiologies of PRES are commonly classified into two categories: toxic PRES, associated with substances such as antineoplastic and illicit drugs,

and PRES linked to clinical conditions, including acute hypertension and autoimmune disorders. While the exact pathophysiology of PRES remains unclear, two primary hypotheses have been proposed: cerebral hyperperfusion resulting from acute hypertension and cerebral hypoperfusion caused by endothelial dysfunction.[13]

Typical imaging findings of PRES include reversible vasogenic subcortical oedema, most commonly affecting the bilateral parietal and occipital lobes, with involvement of the frontal and temporal lobes, as well as the cerebellar hemispheres. Atypical sites, such as the basal ganglia, brainstem, and deep white matter, may also be involved. Oedema can occasionally present asymmetrically or unilaterally.[14] The posterior cerebrum is more prone to oedema due to the scarcity of sympathetic nerve fibres in the vertebrobasilar system compared to the internal carotid system, leading to reduced blood pressure regulation in this region.[15] In addition to the typical lesion locations, the most frequent atypical sites involve the brainstem, including the midbrain, pons, and medulla. Atypical involvement has also been observed in the basal ganglia, such as the thalamus and caudate nucleus, and, in rare cases, the spinal cord without cerebral hemispheric involvement.[16] Our patient had atypical involvement of the cortical/subcortical regions and deep white matter of the bilateral frontal-parietal-occipital lobes, including the centrum semiovale, corpus callosum, and left basal ganglia and hence diagnosed as atypical PRES.

In a study of 124 patients with PRES, McKinney et al. reported that 4% exhibited imaging findings consistent with a "central variant" of PRES, characterised by brainstem or deep grey nuclei involvement without cerebral hemispheric involvement.[17] In another series by McKinney et al. involving 76 patients, the findings included the involvement of the thalamus (30.3%), cerebellum (34.2%), brainstem (18.4%), and basal ganglia (11.8%), with unilateral involvement observed in 2.6% of cases.[18]

Similarly, Liman et al. analysed a cohort of 96 PRES patients, noting deep grey nuclei involvement in approximately 25% of cases and infratentorial involvement, predominantly in the cerebellum and pons, in over 50% of cases. Their study identified a parieto-occipital pattern in 53%, a superior frontal sulcus pattern in 17%, a holohemispheric watershed pattern in 17%, and a central pattern in 14% of patients.[19]

Radiological diagnosis of PRES relies primarily on brain CT and MRI. While these imaging modalities localise cerebral oedema, they cannot distinguish its type, necessitating continuous clinical observation. Misdiagnoses, such as cerebral infarction, may delay appropriate treatment.[20] On CT, brain oedema appears as low-density regions, while MRI shows low or iso-signals on T1-weighted images and high signals on T2-weighted images.[20] FLAIR sequences are particularly sensitive for detecting mild oedema, suppressing cerebrospinal fluid interference and highlighting high signal areas of surrounding tissue oedema. However, FLAIR, like T2-weighted imaging, cannot differentiate oedema types.[21] Unusual MRI findings may include restricted diffusion on diffusion-weighted imaging (DWI), post-gadolinium contrast enhancement on T1-weighted images, and evidence of haemorrhage.

With accurate diagnosis and timely, effective treatment, the prognosis for PRES is generally favourable. Neurological symptoms, clinical signs, and radiographic abnormalities typically resolve within 1–2 weeks. However, delayed or incorrect treatment can result in irreversible neurological damage or even death.[20] Management focuses on treating the underlying condition, controlling neurological symptoms, and managing hypertension.[16]

In cases of pre-eclampsia/eclampsia-induced PRES, the primary goal is blood pressure control, seizure prevention or management, and, when necessary, termination of pregnancy. Antihypertensive medications, such as labetalol, hydralazine, nicardipine, or short-acting oral nifedipine, are recommended if systolic blood pressure exceeds 160 mmHg or diastolic pressure exceeds 105–110 mmHg. Magnesium sulphate remains the first-line antispasmodic, with an initial intravenous loading dose followed by maintenance infusion. Other options for seizure control include magnesium sulphate in combination with diazepam, phenytoin, or a lytic cocktail. Additional therapies, such as diuretics and corticosteroids (e.g., dexamethasone or betamethasone), may be used as supportive treatments.[2,22]

Our case of PRES in late denovo eclampsia is particularly unique as it occurred in a woman during the late postpartum period without prior evidence of gestational hypertension, preeclampsia, or eclampsia until the onset of seizures. Additionally, to the best of our knowledge, this represents the first reported instance of PRES associated with de novo preeclampsia in an IVF pregnancy.

The existing literature presents mixed findings on whether IVF serves as a significant risk factor for preeclampsia. A 2004 meta-analysis by Jackson et al., which analysed eight studies, identified an increased risk of preeclampsia associated with IVF, reporting an odds ratio of 1.55 (95% CI: 1.23–1.95).[23] However, the studies included in the meta-analysis adjusted for only a limited range of confounding variables, such as age and parity, thereby limiting their ability to fully account for assignment bias in the results. Following this analysis, several other studies have also demonstrated a correlation between IVF and an elevated risk of preeclampsia.[24,25] In contrast, research by Watanabe et al. involving propensity score matching analysis indicated that the association between IVF and preeclampsia was not significant and the observed relationship between

IVF and preeclampsia may be influenced by residual unmeasured confounding factors.[26]

Preventive measures for preeclampsia often fall into two categories: primary prevention, which targets the general population and includes measures such as bed rest, activity restriction or regular exercise, reduced salt intake, and the use of antioxidants like vitamins C and E, garlic, and marine oils. Secondary prevention focuses on high-risk populations, such as IVF pregnancies, and involves interventions such as diuretics, progesterone, nitric oxide, calcium supplementation, and low-dose aspirin. Low-dose aspirin especially when initiated before 16 weeks in high-risk groups, show promise in preventing preeclampsia.[27]

A meta-analysis evaluating the impact of low-dose aspirin on women at moderate or high risk for pre-eclampsia included 27 studies involving 11,348 participants, with follow-up assessing pre-eclampsia outcomes. Initiating low-dose aspirin at or before 16 weeks of gestation was associated with a significant reduction in the incidence of pre-eclampsia (relative risk [RR] 0.47; 95% confidence interval [CI] 0.34–0.65; prevalence 9.3% in treated vs. 21.3% in control groups) and fetal growth restriction (FGR) (RR 0.44; 95% CI 0.30–0.65; prevalence 7.0% treated vs. 16.3% control). In contrast, starting aspirin after 16 weeks showed no significant effect (PE: RR 0.81; 95% CI 0.63–1.03; prevalence 7.3% treated vs. 8.1% control; FGR: RR 0.98; 95% CI 0.87–1.10; prevalence 10.3% treated vs. 10.5% control). Early initiation of aspirin (≤16 weeks) also led to reductions in severe pre-eclampsia (RR 0.09; 95% CI 0.02–0.37; 0.7% treated vs. 15.0% control), gestational hypertension (RR 0.62; 95% CI 0.45–0.84; 16.7% treated vs. 29.7% control), and preterm birth (RR 0.22; 95% CI 0.10–0.49; prevalence 3.5% treated vs. 16.9% control).[28]

The biological link between IVF and preeclampsia remains unclear, though abnormal placentation is central to preeclampsia's development.[29] A systematic review proposed mechanisms by which IVF might disrupt placentation. These include the impact of conceptus transfer, altered hormonal environments during IVF, and differences in placental development initiated in vitro. Additionally, inadequate uteroplacental circulation may further contribute to this association.[30]

CONCLUSION

This case underscores the importance of recognising atypical presentations of postpartum eclampsia and PRES, especially in IVF pregnancies. Early identification and prompt management of symptoms are crucial to prevent life-threatening complications. Starting aspirin prophylaxis before 16 weeks in high risk pregnancies and in IVF pregnancy might help in prevention of this disorder.

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