Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Postpartum Eclampsia Complicated by Meningeal Haemorrhage: Case Report

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DOI: https://doi.org/10.36347/sjmcr.2025.v13i01.016 | **Received:** 02.12.2024 | **Accepted:** 10.01.2025 | **Published:** 13.01.2025

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Abstract **Case Report**

Eclampsia is a fatal complication of pregnancy that is rare in developed countries, whereas its incidence is more marked in developing countries and constitutes a public health problem. Although a number of risk factors have been identified, the best prevention to date is pregnancy monitoring and screening of patients with gestational hypertension. The pathophysiology of an eclampsia seizure is not well understood to date and remains the subject of hypothesis; however, disruption of the blood-brain barrier in the cerebral parenchyma remains the most widespread theory, hence the interest in close monitoring throughout pregnancy and the postpartum period when complications may also arise. We discuss a clinical case of this type.

Keywords: Hypertension, eclampsia, post partum, meningeal haemorrhage.

INTRODUCTION

Eclampsia is defined as the occurrence of one or more seizures unrelated to a neurological cause during pregnancy or postpartum [1, 2]. Although 10% of pregnancies are complicated by hypertensive disorders, eclampsia continues to occur in 0.8% of women with hypertensive disorders [3]. In recent years, the rate of eclampsia has fallen in developed countries thanks to good pregnancy monitoring and multidisciplinary involvement. This was the case in our patient, who neglected to measure her blood pressure (BP) in the face of excruciating headaches, since her BP was normal throughout her pregnancy and even during the three days postpartum until tonic-clonic seizures set in, prompting a visit to the emergency department. Most post-partum eclampsia occurs in the first 48 hours after delivery [4-6].

CASE REPORT

The patient was 35 years old, had no previous pathological history and had given birth to 3 healthy children during 3 pregnancies. During the last pregnancy, the follow-up was unremarkable and the delivery was by a programmed caesarean section with the birth of a female newborn in good health. On the 7th post-partum day of this last pregnancy, the sudden onset of atrocious thunderclap headaches in the afternoon, which were resistant to analgesic treatment. At around 5 a.m. on the 2nd day, a generalised tonic-clonic epileptic seizure occurred during sleep. The patient was taken by her family to emergency at our hospital, and on admission she had a second epileptic seizure similar to the previous one, lasting 1 minute. The 2nd seizure occurred 30 minutes after the first seizure, in an apyretic state with no visual disturbances or vomiting. Clinical examination revealed an obnubilated, confused patient with a post-critical Glasgow Coma Scale of 13/15. There was no fever, no skin lesions and no signs of respiratory distress. Blood pressure was 180/100 mmHg, heart rate 100 beats per minute (bpm). Post-critical neurological examination revealed no motor or sensory deficits, with pupils that were equal and reactive. The patient was initially treated with diazepam (5 mg IVL), paracetamol (1g/8h IV infusion) and nicardipine (IV autopulsed syringe), followed by nimodipine (30 mg/4h) after control of hypertension. Clinically, the patient became conscious with good temporospatial orientation but still had mild headaches. A cerebral computed tomography (CT) scan was performed and showed spontaneous hyperdensity in the cortical sulci on the right side. Cerebral magnetic resonance imaging (MRI) with angio sequences and Gado injection (Figure 1) confirmed

right-sided subarachnoid haemorrhage in the territory of the distal branches of the right middle cerebral artery, with no presence of cerebral thrombophlebitis, arteriovenous malformations or aneurysms.

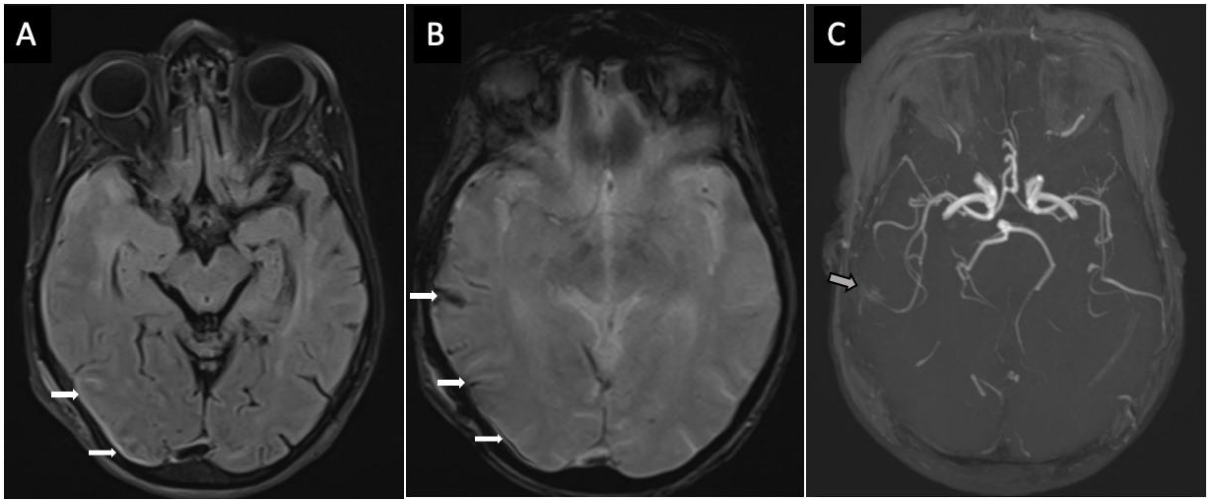

Figure 1: Brain MRI showing signal abnormalities in the right peri-temporal subarachnoid space with hyper-intensity on FLAIR sequence (A) and hypo-intensity on T2*-weighted gradient-echo (B). The TOF sequence (C) shows evidence of haemorrhage without aneurysmal lesions

General paraclinical tests were carried out and returned without abnormalities, including: complete blood count, prothrombotic tests, liver and kidney tests, haemoglobin electrophoresis, tests for systemic vasculitis and autoimmune diseases, syphilis serology, HIV, viral hepatitis B and C. The electrocardiogram (ECG), Doppler ultrasound of the cervical arteries and echocardiography were unremarkable. A conventional arteriogram was performed one month after the incident and showed no arterial abnormality in the brain. The patient was initially controlled on nimodipine, which was subsequently replaced by amlodipine 5 mg/day. For anti-epileptic drugs, she was put on sodium valproate 500 mg twice daily for 6 months. The course of treatment was favourable, with no recurrence of seizures and stabilisation of haemodynamic status.

DISCUSSION

Eclampsia is a major cause of pregnancy-related maternal mortality, in addition to the complications of abortion, dystocia and haemorrhage [7]. Eclampsia is a major public health issue worldwide, and many prevention programmes are aimed at improving the prognosis of pregnancies in both industrialised and developing countries [8, 9], hence the discussion of our clinical case, which represents a particular case of eclampsia on the 7th day post partum. The patient presented with excruciating headaches with a sudden onset beginning in the middle of the day, and in the evening the patient presented with a tonic-clonic seizure during her sleep, leading to consultation of the emergency department. Throughout the pregnancy, blood pressure was around 100/70 mmHg, even during the 3 days of postoperative hospitalisation for the planned caesarean section. On admission to A&E, she suffered a second tonic-clonic seizure, with an admission blood pressure of 180/110 mmHg. This is why it is important to inform women of the need to avoid trivialising headaches, visual disturbances or residual abdominal pain, and of the importance of consulting a doctor if any of these symptoms appear [10]. This clinical presentation accounts for 44% and 28% of cases of eclampsia in the UK and US respectively [1, 11, 12]. The majority of post-partum eclampsia occurs within the first 48 hours after delivery, and there have been reports of eclampsia up to 23 days post-partum [4, 13, 6]. At present, the most likely pathophysiological hypothesis for eclampsia is that of a posterior reversible encephalopathy (PRES) [14]. The hypertensive crisis is thought to be at the origin of this PRES syndrome due to vasogenic cerebral oedema caused by dysfunction of the cerebral autoregulatory mechanisms with disruption of the blood-brain barrier. This hypothesis helps to explain why some eclampsia occurs when systolic and mean BP are relatively low [15]. In the work by Brewer *et al*., 98% of the eclampsia patients studied had PRES identified by MRI, cerebral CT or MRI angiography, with cerebral oedema located mainly in the subcortical white matter of the occipital territories, where perivascular sympathetic innervation is less important and therefore more vulnerable. The result is increased perfusion with endothelial rupture and pericapillary suffusion in these areas [16]. Ischaemic or haemorrhagic lesions may also appear secondarily, worsening the prognosis. The latter was the case in our patient complicated by a meningeal haemorrhage detected on cerebral CT and MRI.

CONCLUSION

Eclampsia is a dreadful complication of pregnancy, directly affecting the maternal-foetal prognosis. Screening for gestational hypertension is the best way to prevent eclampsia and its complications.

This screening must be maintained in the postpartum period, with education for patients and nursing staff.

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