Absent oculocephalic reflex and brain death in a patient of pre-eclampsia: The latent annotation

Dear Editor,

Pre-eclampsia is a common obstetric emergency with neurological manifestations including convulsions, infarcts, haemorrhage and cerebral oedema.[1] Brain death without evidence of these neurological manifestations is not usually seen.

A 31-year-old female with 33 weeks of gestation was scheduled for emergency caesarean delivery, given severe pre-eclampsia. In the immediate preoperative period, the patient was drowsy with a Glasgow Coma Scale (GCS) score of 13/15, the chest was clear upon auscultation, and arterial blood gas analysis was

normal with serum magnesium of 0.32 mmol/L. An uneventful caesarean delivery was conducted under standard general anaesthesia, including rapid sequence induction and intubation. Residual neuromuscular blockade was not reversed, and the patient was shifted to the intensive care unit (ICU), where intravenous (IV) 10 mg amlodipine, 20 mg labetalol boluses and 5 μg/min nitroglycerin infusion were required for blood pressure control. IV magnesium sulphate (1 g/h) was continued as well. After 18 h, the patient remained unresponsive with a GCS score of 2/10 ($E_1V_TM_1$) despite no additional sedatives or neuromuscular agents; pupils were normal sized but non-reactive to light. A computerised tomography (CT) scan of the head was unremarkable, and the fundus showed grade II hypertensive changes. Detailed neurological examination showed negative oculocephalic reflex, absence of respiratory efforts, cough reflex, and plantar and deep tendon reflexes, all suggesting brain death. Fresh investigations showed raised serum creatinine (2.4 mg/dL), but the urine output was preserved. A 12-lead electrocardiogram revealed tall T-waves from leads V_2 to V_6 with prolonged QTc interval (500 msec). Suspecting hypermagnesaemia, serum levels were checked, which confirmed the suspicion (8 mg/dL). Magnesium sulphate infusion was discontinued, and IV 10% calcium gluconate therapy and furosemide were initiated. After 23 h, GCS score improved to 3/10 ($E_2V_TM_1$) and weak gag reflex was present, although oculocephalic reflex was still absent with persistent muscle paralysis (train-of-four [TOF] ratio- 0.7). By 48 h, the patient was completely responsive with normal muscle strength ($E_4V_TM_6$); the magnesium level declined to 5.1 mg/dL and normalised to 2.4 mg/dL after another two days of medical management and two sessions of haemodialysis. The trachea was extubated, and the patient was discharged from ICU on the sixth postoperative day after complete recovery.

Magnesium toxicity is known to result in coma, cardiovascular and neuromuscular manifestations.[2,3] While these were evident in our patient, the worrisome presentation was the absence of oculocephalic reflex and the unresponsiveness suggestive of brain death. There had been no critical event, all vital parameters had been within normal limits, the head CT scan was unremarkable, and there was a lack of localising signs, making pre-eclampsia–associated neurological damage with brain death seem less likely. The present case focuses on hypermagnesaemia's contribution to mimicking brain death. Testing oculocephalic reflex is an essential component of neurological assessment in comatose patients.[4] Although there is no direct relationship between hypermagnesaemia and absent oculocephalic reflex, neuro-ophthalmologic effects can include bilateral symmetric ptosis, convergence insufficiency and abnormal pupillary testing.[5] The abnormalities suggest the involvement of a supranuclear pathway and peripheral dysfunction. Neuromuscular blockers can result in the absence of oculocephalic reflexes, lack of gag/cough reflexes and motor paralysis. However, despite recovery of neuromuscular junction activity (TOF = 0.7), the patient continued to exhibit muscle paralysis, suggesting greater contribution of hypermagnesaemia. The prolonged contributory effect of neuromuscular blockade was secondary to magnesium toxicity.

We are reporting this case to highlight that the commonly understood complication of magnesium toxicity in patients with severe pre-eclampsia may rarely mimic brain death, which is distinct from the previously documented cerebral function suppression associated with toxicity-induced coma.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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REFERENCES

1. Lucia M, Viviana M, Alba C, Giulia D, Carlo DR, Grazia PM, *et al*. Neurological complications in pregnancy and the puerperium: Methodology for a clinical diagnosis. J Clin Med 2023;12:2994. doi: 10.3390/jcm12082994.
2. Lu JF, Nightingale CH. Magnesium sulfate in eclampsia and pre-eclampsia: Pharmacokinetic principles. Clin Pharmacokinet 2000;38:305-14.
3. Brodie H, Malinow AM. Anesthetic management of preeclampsia/eclampsia. Int J Obstet Anesth 1999;8:110-24.
4. Simakurthy S, Tripathy K. Oculovestibular Reflex. StatPearls. Treasure Island (FL): StatPearls Publishing; 2022. Available from: http://www.ncbi.nlm.nih.gov/books/NBK542281/. [Last accessed on 2022 Feb 27].
5. Digre KB, Varner MW, Schiffman JS. Neuroophthalmologic effects of intravenous magnesium sulfate. Am J Obstet Gynecol 1990;163:1848-52.

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