Case Report

Ferric carboxymaltose-induced posterior reversible encephalopathy syndrome: A rare case report

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic condition characterized by acute neurological symptoms and distinctive imaging findings. It is commonly linked to hypertension, immunosuppressants, or chemotherapy, but rarely to intravenous (IV) iron infusion. We describe a 40-year-old female who developed PRES following IV ferric carboxymaltose (1 g) for severe anemia. Soon after infusion, she experienced anaphylactic shock with hypotension, bradycardia, facial swelling, vision loss, headache, and vomiting. Magnetic resonance imaging brain revealed bilateral parieto-occipital hyperintensities on T2/fluid-attenuated inversion recovery sequences, consistent with PRES. Labs showed worsening anemia (hemoglobin 7.4 → 6.3 g/dL), thrombocytopenia, leukocytosis, and a sharp rise in serum iron (362 µg/dL). She was managed with mannitol, levetiracetam, antibiotics, and supportive care. Gradual neurological recovery was observed, with vision improving over 1 week. This report highlights a rare instance of PRES triggered by ferric carboxymaltose. Timely recognition and appropriate treatment are essential to prevent long-term complications. Clinicians should remain vigilant for PRES in patients presenting with acute neurological symptoms after IV Iron therapy.

Key words: Case report, Ferric carboxymaltose, Intravenous iron, Posterior reversible encephalopathy syndrome

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic state characterized on the basis of neurological symptoms and brain imaging findings. It is associated with impaired cerebral autoregulation and discretion of the endothelium [1,2]. The common symptoms of PRES are visual loss, headache, altered brain function, and sensory disturbance [1-3]. The prevalence of PRES is highly variable. Although it is rare in the general population, the prevalence is higher (1–25%) in populations with predisposing conditions, such as eclampsia, transplant, or renal failure [4-7]. Finding of PRES is commonly associated with hypertension, immunosuppressive agents, and chemotherapy [1,8-10].

PRES following intravenous (IV) iron therapy, particularly ferric carboxymaltose, is extremely rare and poorly documented [11]. This case highlights a very unique presentation of PRES, triggered by ferric carboxymaltose-induced impaired cerebral autoregulation and acute anaphylaxis. Recognizing such atypical triggers is very crucial, by which, early diagnosis and management of PRES is possible. In addition, early detection can prevent permanent neurological damage [7].

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CASE PRESENTATION

A 40-year-old female presented to our hospital emergency department with generalized weakness and blurred vision for 1 day. The reports of the magnetic resonance imaging (MRI) brain done outside the hospital suggested PRES.

On arrival at the emergency department, the patient's vital parameters were as follows: Blood pressure 86/56 mmHg, pulse 45/min, respiratory rate 18/min, SpO_2 96% on room air, and temperature 36.8°C.

On detailed history, it was found that 1 day before admission to our hospital, the patient was administered an injection of iron 1 g (Ferric carboxymaltose) from outside the hospital and upon administration, the patient had low blood pressure- 86/56 mmhg, low pulse rate 45/min swelling on face, lips, hands, loss of vision, headache, nausea, vomiting, and the anaphylactic shock. The investigations done in another hospital showed that the patient had severe microcytic hypochromic anemia with hemoglobin of 7.4 g/dL, mean corpuscular volume 61.5 fL, mean corpuscular hemoglobin 18.8 pg, and mean

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corpuscular hemoglobin concentration 30.6 g/dL. Serum iron was 14.9 μg/dL, and erythrocyte sedimentation rate was 20 mm/h. Platelet count was normal at 321 × 10^9/L, and the total leukocyte count (TLC) was 5.8 × 10^9/L. The patient was treated with injection pantoprazole 40 mg IV once daily, injection paracetamol 1 g IV SOS, injection dexamethasone 8 mg IV STAT, injection mannitol 100 mL, and inj. piperacillin tazobactam 4.5 g IV 8 h at outside hospital.

The patient was admitted to the intensive care unit for management, and then, the patient was catheterized due to decreased urine output. On investigations, MRI showed multiple areas of T2 and fluid-attenuated inversion recovery hyperintensity, which involved all areas of the brain, suggestive of PRES. Non-contrast computed tomography brain was suggestive of III-defined hypodense areas seen involving the left cerebellum, vermis, and bilateral parieto-occipital regions, suggesting PRES.

On admission, hemoglobin remained low (7.8 g/dL) with a sharp rise in TLC (21.8 × 10^9/L) and a fall in platelets (284 × 10^9/L). Procalcitonin (PCT) on the 2nd day of admission was 1.020. In the following days, anemia worsened, with hemoglobin reaching its lowest value of 6.3 g/dL and hematocrit dropping to 26.1%. Platelets also decreased to 135 × 10^9/L by day 6. At the same time, serum iron increased significantly to 362 μg/dL on day 2, indicating iron overload after therapy.

The patient was treated with injection piperacillin tazobactam 4.5 g IV thrice daily in view of increased PCT and TLC. Also started with injection levipil 500 mg IV twice daily, injection vitamin K 10 mg IV once daily for 3 days, injection paracetamol 1g IV 6 h, injection mannitol 100 mL IV 8 h.

From day 5 to day 8, gradual recovery was observed. Hemoglobin improved to 8.6 g/dL, hematocrit increased to 35%, and leukocyte counts were stable in the range of 8–10 × 10^9/L. Platelet counts also improved to 163 × 10^9/L. Erythrocyte sedimentation rate during admission fluctuated between 11 and 24 mm/h. The patient was slowly recovering; the patient's vision improved slowly over 1 week as cerebral pressure decreased with mannitol. The patient was not able to see 1 m apart after a week clearly. At 1-month follow-up, the patient's vision and overall neurological condition had further improved, with no new symptoms reported.

DISCUSSION

PRES is a neurotoxic state characterized by acute neurological symptoms such as headache, seizures, visual loss, and altered sensorium, with MRI changes typically in the parieto-occipital lobes [1,2]. It is most commonly associated with hypertension, renal disease, eclampsia, and exposure to immunosuppressants or chemotherapeutic drugs [8-12]. Drug-induced PRES has been increasingly recognized, with agents such as calcineurin inhibitors, corticosteroids, monoclonal antibodies, and chemotherapeutics implicated [3,9,13]. The occurrence of PRES following ferric carboxymaltose infusion, as in our case, is extremely rare and underscores the need for vigilance in patients receiving IV iron therapy.

The patient's presentation required careful consideration of differential diagnoses. Acute

Table 1: Review of literature

S. No	Authors name	Detail of event	Drug involved
1.	Abbas *et al*. 2013 [1]	PRES in a patient treated with bevacizumab, presenting with hypertension and tonic-clonic seizures	Bevacizumab
2.	Nakamura *et al*. 2018 [2]	PRES after blood transfusion, presenting with extensive cytotoxic edema	Blood transfusion
3.	Morrow *et al*. 2015 [3]	PRES associated with high-dose corticosteroids, presenting with insomnia, dizziness, generalized weakness, and occipital headache	Corticosteroids
4.	Puram *et al*. 2022 [8]	PRES induced by mepolizumab, presenting with altered awareness, slurred, and incoherent speech	Mepolizumab
5.	Song *et al*. 2016 [9]	PRES in patients receiving calcineurin-inhibitor or sirolimus therapy presented with headache, blurred vision, and seizures	Calcineurin inhibitors, Sirolimus
6.	Wu *et al*. 2010 [10]	Tacrolimus-associated PRES in organ transplant patients, presenting with confusion and seizures	Tacrolimus
7.	Bagrin *et al*. 2024 [11]	Neurological impact of severe iron deficiency anemia, presenting with stroke and IV iron therapy	Iron deficiency anemia, IV iron therapy
8.	Gheith *et al*. 2017 [12]	Sirolimus-induced PRES in renal transplant recipients, presenting with visual disturbances and altered mental status	Sirolimus
9.	Tabot Tabot *et al*. 2023 [13]	Cyclophosphamide-induced PRES in a patient with lupus nephritis, presenting with restlessness, confusion, hallucinations, and altered mentation	Cyclophosphamide
10.	Truong *et al*. 2012 [14]	PRES associated with gemcitabine, presenting with hypertension and altered mental status	Gemcitabine
11.	Liu *et al*. 2024 [15]	Sepsis-associated encephalopathy, presenting with mild delirium to deep coma	Iron metabolism

PRES: Posterior reversible encephalopathy syndrome, IV: Intravenous

posterior circulation stroke can mimic PRES but usually demonstrates restricted diffusion on MRI. Demyelinating disorders such as multiple sclerosis and acute disseminated encephalomyelitis may also present with multifocal lesions but differ in chronicity and Cerebrospinal fluid findings. Infectious and metabolic encephalopathies, including hypoglycemia and uremia, can present similarly but are usually clarified by systemic investigations. Hypertensive encephalopathy was excluded due to the absence of persistent elevated blood pressure. After ruling out these conditions, PRES was the most consistent diagnosis [1,2,6,8].

The pathophysiology of PRES is not fully understood but involves endothelial dysfunction, impaired autoregulation, and disruption of the blood–brain barrier [6,7]. In this case, an acute hypersensitivity reaction with hypotension likely triggered cerebral endothelial injury, leading to vasogenic edema. Both hypertension and hypotension have been implicated as precipitating factors for PRES [4,5]. Literature shows cases linked to blood transfusion [2], sirolimus [12], calcineurin inhibitors [9], and high-dose corticosteroids [3]. However, its association with IV iron therapy is scarcely documented, making this report clinically relevant. Table 1 shows a review of the literature on the cases of PRES [1-3,8-15].

Early recognition and treatment are crucial, as PRES is potentially reversible when managed promptly [16,17]. Our patient improved with mannitol, anticonvulsants, and supportive therapy, with gradual recovery of vision within 1 week. This case highlights the importance of considering PRES in patients who develop acute neurological symptoms after IV iron infusion and contributes to the limited data on iron-induced PRES.

CONCLUSION

PRES is a potentially reversible condition if diagnosed and treated appropriately. This case is needed to create awareness among clinicians of rare drug-induced triggers of PRES and the need for further research into mechanisms and preventive strategies. Follow-up demonstrated sustained clinical recovery, underscoring the importance of early recognition and treatment of PRES.

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