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HYPOCALCEMIC SEIZURES DUE TO HYPOPARATHYROIDISM IN TRANSFUSION-DEPENDENT -THALASSEMIA: A CASE REPORT

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ABSTRACT Transfusion-dependent -thalassemia is linked to chronic iron overload, which can cause various endocrine problems. Iron buildup in the parathyroid glands may lead to hypoparathyroidism, resulting in severe hypocalcemia that can cause neuromuscular irritability, tetany, or seizures. However, seizures caused by hypocalcemia as the first sign in these patients are relatively rare and can be missed in emergencies. This case report aims to highlight hypocalcemia caused by hypoparathyroidism as a rare but reversible cause of seizures in patients with transfusion-dependent thalassemia and to stress the importance of early metabolic assessment. We report a 27-year-old male with -thalassemia major on regular blood transfusions who presented with newly onset generalized tonic–clonic seizures. Clinical examination, biochemical tests, electrocardiography, and neuroimaging were performed to identify the underlying cause. Investigations showed severe hypocalcemia with significantly low parathyroid hormone levels and high ferritin, indicating iron overload. Electrocardiography revealed QTc prolongation. CT and MRI of the brain displayed basal ganglia calcifications and hemosiderin buildup. The patient improved after receiving intravenous calcium and supportive care. This case emphasizes the importance of considering endocrine and metabolic complications in thalassemia patients presenting with seizures. Early identification and swift correction of hypocalcemia can prevent serious neurological and cardiac issues.

KEYWORDS : Hypocalcemia, Seizures, Hypoparathyroidism, Thalassemia, Blood transfusion, Iron overload

INTRODUCTION

Beta thalassemia is an inherited hemoglobin disorder characterized by reduced or absent production of beta-globin chains (Cappellini et al., 2014). Patients with transfusion-dependent beta thalassemia need lifelong regular blood transfusions to survive (Cappellini et al., 2014).

Although transfusion therapy prolongs life expectancy, repeated transfusions can cause iron overload. Excess iron in endocrine glands may lead to disorders like diabetes mellitus, hypothyroidism, hypogonadism, and hypoparathyroidism (De Sanctis et al., 2018).

Hypoparathyroidism decreases parathyroid hormone (PTH) secretion, resulting in hypocalcemia and hyperphosphatemia (Bilezikian et al., 2011). Hypocalcemia can cause neuromuscular irritability, tetany, seizures, and cardiac conduction issues (Bilezikian et al., 2011).

Seizures caused by hypocalcemia are rare but significant, as they represent a reversible neurological emergency (Bilezikian et al., 2011). This case illustrates hypocalcemia-induced seizures in a patient with transfusion-dependent thalassemia.

CASE STUDY

Case Description

A 27-year-old male with transfusion-dependent beta thalassemia presented to the emergency department with an acute onset of generalized tonic-clonic seizures and subsequent altered sensorium. He was drowsy and post-ictal. Initial stabilization was performed in accordance with standard emergency protocols. Vital parameters were monitored and airway protection ensured.

The patient was a known case of beta thalassemia major since one month of age and was on regular blood transfusions and oral iron chelation therapy (deferiprone). Past history included a cholecystectomy performed one year prior.

Clinical examination revealed neuromuscular irritability and a positive Chvostek's sign, indicating hypocalcemia. Electrocardiography demonstrated QTc prolongation with T-wave alternans. Laboratory investigations revealed elevated serum ferritin (1000 ng/mL), markedly reduced serum calcium (4.0 mg/dL) and magnesium, and low parathyroid hormone levels (1.99 pg/mL), with normal vitamin D levels (42 ng/mL).

A diagnosis of hypocalcemia-induced seizures caused by hypoparathyroidism from chronic iron overload was made, based on clinical features, ECG findings, and laboratory results.

Management included intravenous calcium supplementation, antiepileptic therapy, and iron chelation. Additionally, close monitoring of cardiac and neurological functions was essential throughout the patient's hospital course.

RESULTS

The patient received intravenous calcium supplementation, antiepileptic therapy, electrolyte correction, and supportive care with close cardiac and neurological monitoring. Laboratory tests showed markedly low serum calcium, very low parathyroid hormone levels, and normal vitamin D levels, indicating hypoparathyroidism. Serum ferritin was elevated, suggesting chronic iron overload. An electrocardiogram showed QTc prolongation with T-wave alternans, a typical sign of hypocalcemia. Computed tomography of the brain demonstrated multiple symmetrical calcifications involving bilateral basal ganglia and thalami as well as calcifications at the grey–white matter junction in the cerebrum and cerebellum.

Magnetic resonance imaging of the brain revealed altered signal intensity involving bilateral caudate nuclei, lentiform nuclei, and dorsolateral thalami, suggestive of hemosiderin deposition secondary to chronic iron overload. After correcting the calcium levels, the patient improved clinically, and the seizures resolved.

DISCUSSION

Repeated transfusions in beta thalassemia major cause secondary iron overload, which can harm multiple organs, including endocrine glands. Iron buildup in the parathyroid glands can lead to hypoparathyroidism, resulting in impaired calcium regulation and severe hypocalcemia.

Hypocalcemia increases neuronal excitability and can cause neuromuscular irritability, tetany, and seizures (Bilezikian et al., 2011). Electrocardiographic findings, such as QT interval prolongation, may also occur (Bilezikian et al., 2011). Quick recognition and treatment with intravenous calcium are vital to prevent serious complications, including cardiac arrhythmias (Bilezikian et al., 2011).

In this case, hypoparathyroidism caused by iron overload was confirmed by the presence of severe hypocalcemia, extremely low parathyroid hormone levels, and normal vitamin D levels. The patient responded well to calcium supplementation, emphasizing the importance of early diagnosis and treatment. Hypocalcemia should be considered in patients with transfusion-dependent thalassemia presenting with seizures or neuromuscular irritability. Early identification and timely management can prevent recurrent seizures and improve patient outcomes.

Hypocalcemia is an uncommon but important reversible cause of seizures. Calcium is essential for synaptic transmission and neuronal excitability. Reduced extracellular calcium lowers the threshold for neuronal depolarization, increasing seizure risk.

Patients with transfusion-dependent thalassemia are at risk of endocrine complications due to iron deposition in endocrine glands (De Sanctis et al., 2018). Hypoparathyroidism is one such complication that can cause chronic hypocalcemia (De Sanctis et al., 2018).

Massive or repeated blood transfusions may also contribute to hypocalcemia because citrate in stored blood binds calcium.

Figure 1: Electrocardiogram showing QTc prolongation with T-wave alternans

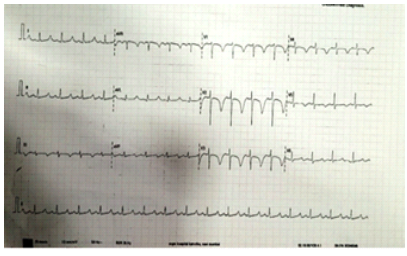
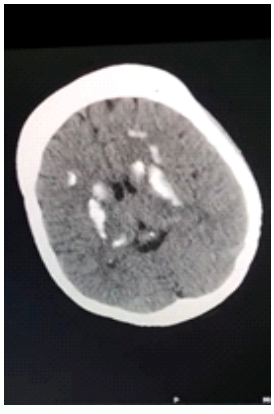


Figure 2: Computed tomography of the brain demonstrated multiple symmetrical calcifications involving bilateral basal ganglia and thalami



CONCLUSION

As patient survival improves, endocrine complications related to transfusion-dependent -thalassemia are becoming more evident. Chronic iron overload can cause hypoparathyroidism, leading to severe hypocalcemia that may manifest as seizures and neuromuscular irritability. This case underscores the importance of considering metabolic causes when diagnosing new seizures, particularly in patients with extensive transfusion histories. Prompt diagnosis and appropriate treatment can significantly lower morbidity and prevent future neurological issues.

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