Seizure as a Presenting Manifestation of Idiopathic Hypoparathyroidism

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Abstract:
A 31-year-old male presented with generalized new-onset tonic-clonic seizure related to severe hypocalcaemia (serum calcium 1.2 mmol/l) caused by idiopathic hypoparathyroidism (parathyroid hormone concentration 6 pg/ml; normal 15-65 pg/ml).

Computerized tomographic scan showed bilateral, symmetrical, intracranial calcifications involving the basal ganglia and the cerebellum.

There are few reports in clinical literature describing new-onset seizure caused by primary hypoparathyroidism in adulthood and hypoparathyroidism needs to be considered in the differential diagnosis of adult-onset seizure.

Key words: seizure, hypocalcaemia, hypoparathyroidism

Introduction:
The most common cause of hypoparathyroidism is injury to the parathyroid glands during thyroid surgery,(1) while hypoparathyroidism not related to surgery is usually an autoimmune disease.(2)

Clinical manifestations of hypoparathyroidism include: tetany, painful muscle spasms of hands and feet, and facial muscle spasms. Hypoparathyroidism is also associated with psychiatric symptoms mainly anxiety, depression, psychosis, and delirium.(3)

Treatment of severe symptomatic hypocalcaemia requires prompt administration of parenteral calcium. In contrast asymptomatic hypocalcaemia may be treated with oral calcium and vitamin D supplements.(4)

Case Report:
A 31 year old man presented to the medical emergency room, Hamad General Hospital, with new-onset, generalized tonic-clonic seizure related to severe hypocalcaemia, serum calcium 1.2 mmol/l (normal 2.1-2.6 mmol/l), ionized calcium 0.61 mmol/l (normal 1.18-1.32 mmol/l), with low parathyroid hormone concentration 6 pg/ml (normal 15-65 pg/ml), and hyperphosphateemia, serum phosphate 2.34 mmol/l (normal 0.87-1.45 mmol/l). Renal function tests, serum magnesium, serum sodium, serum albumin, alkaline phosphatase, vitamin D level, thyroid function test, and serum cortisol were all normal but his serum potassium was low, 2.8 mmol/l (normal 3.6-5.1). He had a history of bilateral cataract extraction five years previously. Computerized tomographic scan showed bilateral, symmetrical, intracranial calcifications involving the basal ganglia and cerebellum (Figures 1 and 2). He was admitted and treated with intravenous calcium gluconate, oral calcium carbonate and vitamin D supplements.

Discussion:
Patients with hypocalcaemia usually present with paresthesia, cramps, myalgia, and muscular weakness. Severe hypocalcaemia manifests as spontaneous tetany,
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Figure 2: Head CT scan showing bilateral cerebellar calcifications which may appear in the form of carpopedal spasms. (5) There are only few reports in clinical literature which describe new onset-seizure caused by hypocalcaemia in adulthood. (6) Mrowka, et al. (7) and Lehmann, et al. (8) described patients presenting with new onset tonic-clonic seizures as the presenting manifestation of post-operative hypoparathyroidism, while Ameneiros-Lago, et al. (9) described new-onset seizure secondary to primary hypoparathyroidism.

Diagnosis of hypoparathyroidism is based on low serum calcium, high serum phosphate, normal serum magnesium, and alkaline phosphatase. (10) Computerized tomography scan allows earlier diagnosis by detecting intracranial calcifications with high sensitivity and specificity, while magnetic resonance imaging is not useful as the signal intensity of calcified lesions varies widely. (3)

Intracranial calcifications can be physiological or pathological. Physiological intracranial calcifications are detected in about 0.3-1.5% of the routine radiological examinations. Pathological basal ganglia calcifications are due to various causes: metabolic disorders, infectious, and genetic. Hypoparathyroidism and pseudohypoparathyroidism are the most common causes of pathological basal ganglia calcification. (11, 12) The mechanism of intracranial calcifications in hypoparathyroidism is not clear; it may be related to the duration of hypocalcemia and hyperphosphatemia as hyperphosphatemia increases calcium deposition in the brain tissue. (3)

Conclusion:

Seizures related to marked hypocalcaemia might be the presenting feature in adult patients with idiopathic hypoparathyroidism, so this needs to be considered in the differential diagnosis of new-onset generalized tonic-clonic seizure.

References: