

Calcification of Basal Ganglia in Chronic Hypoparathyroidism

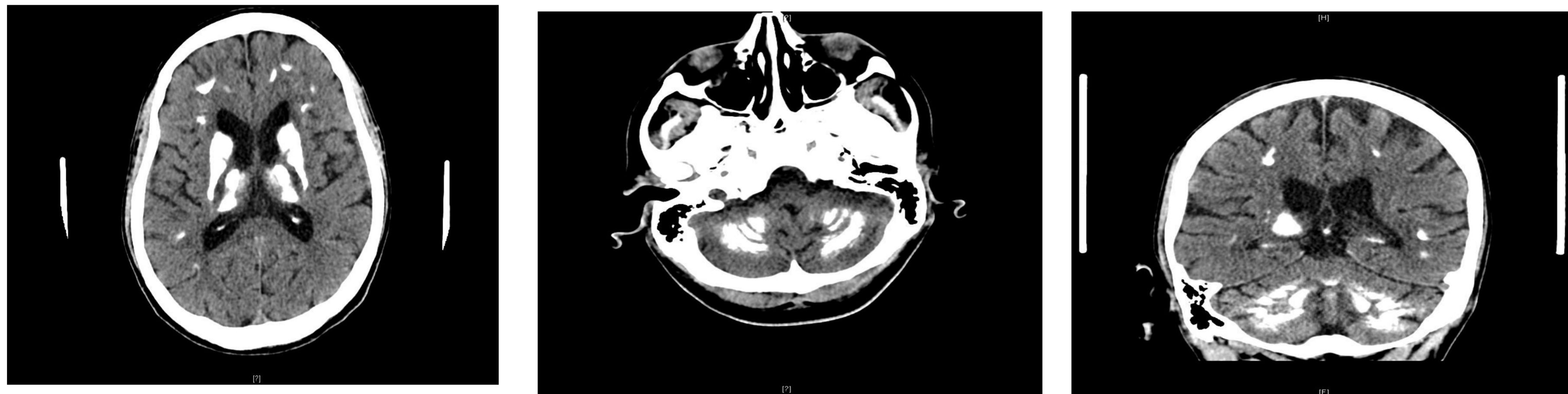
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Case Presentation

- A 39 years old lady with Type 1 Diabetes was repeatedly hospitalised with seizures thought to be related to hypoglycaemia though capillary glucose was never documented in these episodes
 - She also had primary hypoparathyroidism for the last ten years and was on calcium and vitamin D supplements
 - She had cataract removal from right eye and also had early cataract in left eye
 - Her family also confirmed her declining memory over past 2 years. She would take long pauses trying to recall things
 - On examination she had slow speech, serpentine gaze and shuffling gait. Chvostek's sign and Trousseau's signs were negative.
- Rest of the systemic examination was normal
- Her calcium was reasonable with adjusted calcium of 2.-2.5mmol/L on treatment

Progress and Management

She was sent for neuropsychological assessment to ascertain the cognitive impairment and whether she would be capable of looking after her diabetes by herself. She was found to have marked cognitive impairment in everyday functioning, particularly planning & organisation, abstract reasoning, attending to verbal information and processing new information, recalling and decision making. Thus had CT brain which showed wide spread calcification in the brain. She was seen by a neurologist and started on anticonvulsant treatment. Her seizures improved with no improvement in her cognition and gait abnormalities



CT Brain - showing calcification in basal ganglia involving caudate nucleus, lentiform nucleus and thalamus, in the centrum semiovale bilaterally & also subcortical dense curvilinear calcification bilaterally

Discussion

- Hypoparathyroidism is the common cause of pathological calcification in the brain though 0.3- 1.5 % cases are physiological
- Seizures occur in up to 70% of patients with symptomatic hypoparathyroidism^{1,2,3}
- Parkinsonism, dystonia, hemiballismus, choreoathetosis, and oculogyric crises occur in 5-10% of patients with idiopathic hypoparathyroidism⁴ but are less common in patients with surgical hypoparathyroidism or other causes of hypocalcemia⁴
- Parkinsonism seems to be resistant to levodopa but in some cases improvement occurs with calcium and vitamin D replacement^{5,6,7}
- The mechanism of intracranial calcification is not completely understood⁸. It may be related to the duration of hypocalcaemia and hyperphosphataemia than parathyroid hormone itself
- The goal of therapy in hypoparathyroidism is to control symptoms and minimise complications. Use of PTH is not recommended⁹
- In our case, initially hypoglycaemia was thought to be the cause of patient's seizures and neuropsychiatric symptoms as serum calcium was stable (2.2-2.5mmol/L). However, her visit to the neurologist and subsequent CT brain findings and improvement in seizures (after anticonvulsant treatment) showed that her symptoms were due to hypoparathyroidism related brain calcification

Conclusion

This case emphasizes the importance of thinking about the whole spectrum of the disease even though the biochemical markers are stable on treatment.

References

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