

Pandora's Box: Neurological Manifestations of Hypoparathyroidism

Sir,

Was it God who had meant mere mortals to forget and ignore the parathyroid glands or was it Nature who thought that humans would never understand the importance of these glands and hence decided to conceal them behind a much larger endocrine organ, the thyroid? However, in this age of evidence-based medicine, can we still continue to ignore these little dot-like structures?

It is difficult to definitely pinpoint the prevalence of hypoparathyroidism in our country because of the lack of published research in this field, but data from Denmark put an incidence of 0.8 per 1 lakh person-years and a prevalence of 22 per 1 lakh person-years with a somewhat higher prevalence in the USA (37 per lakh).^[1] To manifest clinically, all the parathyroid glands (commonly four in number) need to stop functioning and hence hypoparathyroidism is quite uncommon. Even then, the myriad clinical manifestations range from renal calculi, posterior subcapsular cataract to seizures. The most fascinating manifestations are, however, the ones involving the nervous system. Imaging studies have noted the presence of calcifications in the brain as well as calculi in the kidneys. Unfortunately, however, there is not much correlation between the severities of the clinical manifestations with the radiological extent of the disease.^[2]

Neurological manifestations have been reported since as long back as 1967 where they documented patients manifesting with tetany, chorea, and seizures.^[3] Some very relevant publications from India have found carpopedal spasm as the most common clinical manifestation of hypoparathyroidism (about 70% cases), followed by paresthesia and seizures (54%).^[2] More specifically, the most common clinical manifestation of hypoparathyroidism seems to be neurological.^[3] We too have been seeing hypoparathyroidism manifest with different neurological manifestations. We have found patients to present with varied extrapyramidal symptoms such as choreoathetosis and status epilepticus.^[4] We have also noted patients to present with clinical manifestations of the cerebellar disease.^[5] As such, the primary interaction with the patient should focus on trying to elicit signs and symptoms of neuromuscular irritability,^[2] out of which the most common (as noted above) is paresthesias (involving fingertips, toes, perioral area), followed by others such as hyperirritability, fatigue, anxiety, mood swings and/or personality disturbances, seizures (especially in patients with epilepsy), hoarseness (due to laryngospasm), wheezing and dyspnea (due to bronchospasm), muscle cramps, diaphoresis, and biliary colic.^[6] Often this neuromuscular irritability may just manifest as cramps in the legs, feet, and back. It is only when the irritability is more that we get the clinical signs

of Chvostek and Trousseau. Choreoathetoid movements, as documented in our patient, have also been reported by other clinicians and are probably due to the basal ganglia calcifications that we see in primary hypoparathyroidism.^[7] Even features of Parkinsonism, dystonias, hemiballismus, and oculogyric crises have been noted by clinicians to the extent of up to 5% cases of idiopathic hypoparathyroidism.^[8] Ataxia, paraplegia, dysphagia, and dysarthria have all been documented in patients with hypoparathyroidism. Thankfully, there are some symptoms which have been seen to get reverted with correction of the hypocalcemia starting from Chvostek and Trousseau signs to papilledema. Many patients primarily present to psychiatrists with features of emotional instability, anxiety, depression, confusion, hallucinations, and psychosis. Thankfully, normocalcemia corrects these conditions.^[6] To make matters worse, any coexisting hypomagnesemia, hypokalemia, and alkalosis will worsen the signs and symptoms of hypocalcemia and hence needs to be excluded while treating a patient with hypoparathyroidism.

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Conflicts of interest

There are no conflicts of interest.

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