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Case Report

Whenever Parkinsonism and Muscle Hyperexcitability, Think Calcium

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Abstract

Early-onset symmetric parkinsonism that is unresponsive to therapy should prompt exclusion of treatable causes. A 55-year-old patient presented with one-year-long akinetic parkinsonism and depression. Levodopa-carbidopa trials were unsuccessful. Later aggravation with weight loss, muscle spasms and chorea prompted secondary imaging and laboratory studies, which showed bilateral basal ganglia calcification and primary hypoparathyroidism with low plasmatic calcium. Their extrapyramidal symptoms and muscle hyperexcitability were successfully treated with a parathyroid hormone receptor agonist.

Keywords: *Secondary Parkinsonism, Spasms, Fahr, Hypoparathyroidism, Hypocalcemia*

Introduction

Extrapyramidal symptoms usually present progressively and are attributed to neurodegenerative disorders or medical iatrogenesis. However, more acute-onset parkinsonisms are sometimes seen in clinical practice, and their origin may be harder to pinpoint in certain ages or population groups. This has implications on treatment and prognosis, as some patients may benefit largely from an earlier diagnosis not only in symptomatic control but also even clinical resolution, if a secondary treatable cause is found. We present the illustrative diagnostic challenge of a 55-year-old male patient. [1]

Case Presentation

He was referred to a Neurology consultation after one-year-long lasting complaints of a perception of more laborious daily tasks, loss of balance, slowed walking and more recently involuntary limb movements upon standing up. He had a history of hypertension and mildly high blood cholesterol, denying frequent consumption of traditional toxic or other substances. These symptoms were aggravating his mood and promoting higher levels of anxiety at work. When asked, he also mentioned urinating more often than before, but denied increased thirst, changes in urine coloration or other urinary symptoms. He denied fever, weight loss or other systemic or neurologic symptoms. Laboratory tests remotely ordered by a general practitioner consisting of serum sodium, potassium and basic blood, kidney, liver, thyroid and urinary parameters were mostly ordinary, revealing only augmented lactate dehydrogenase (700, reference range <280 UI/L) and creatinine kinase (1200, reference range <200 UI/L) levels.

Erythrocyte sedimentation rate and C-reactive protein levels were normal. Vital signs were stable. Observation revealed mild psychomotor slowing and dysarthria but pretty intelligible speech and otherwise preserved attention and remaining higher cognitive abilities. He had generalized brisk nonpathological deep tendon reflexes and, mostly left, bilateral limb bradykinesia and irregularly increased cogwheel muscle tonus (grade 2 of 4 within the Unified Parkinson's Disease Rating Scale[1]). Upper extremity mild action postural tremor was also noted, and the gait was narrow-based but slowed, on small steps, and with reduced upper limb bobbing. He had slight camptocormia. Touch, moving from a sitting to standing position or actively doing ampler movements elicited spasms and hyperextension of the limb muscles. He denied exposure to any suspicious environmental agents. There had been no introduction or change of medication. Levodopa-carbidopa titrated to 400/100 mg/daily and duloxetine to 60 mg/daily dose produced no significant response, so brain MRI was ordered due to apparent early-onset resistant parkinsonism. Images showed mixed signal on T1, and T2 and susceptibility-weighted hypointensities along the caudate nuclei, bilateral globus pallidus, posterior thalamus, and also more mildly alongside centromedullary, subcortical parts of the cerebellum and corpus callosum. There was no restriction to water molecule diffusion or contrast enhancement.

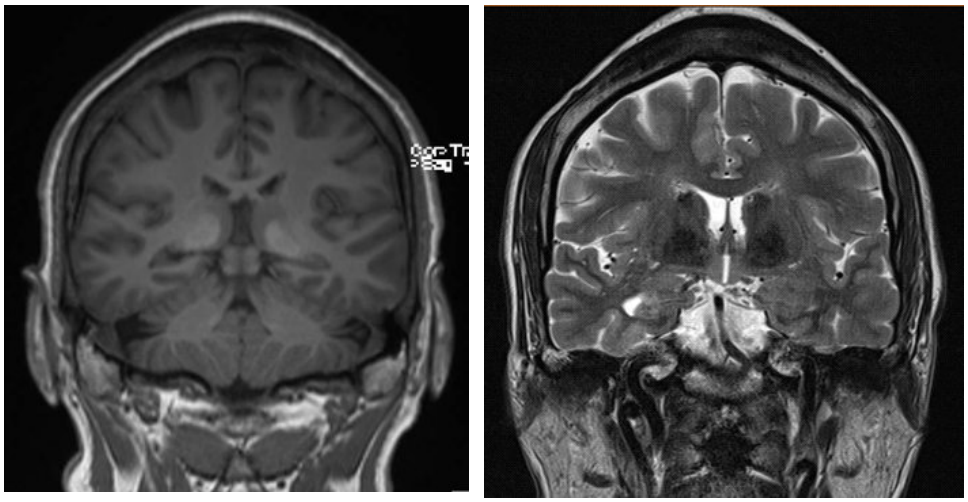

Figure 1. Coronal T1 and T2 brain MRI sequences.

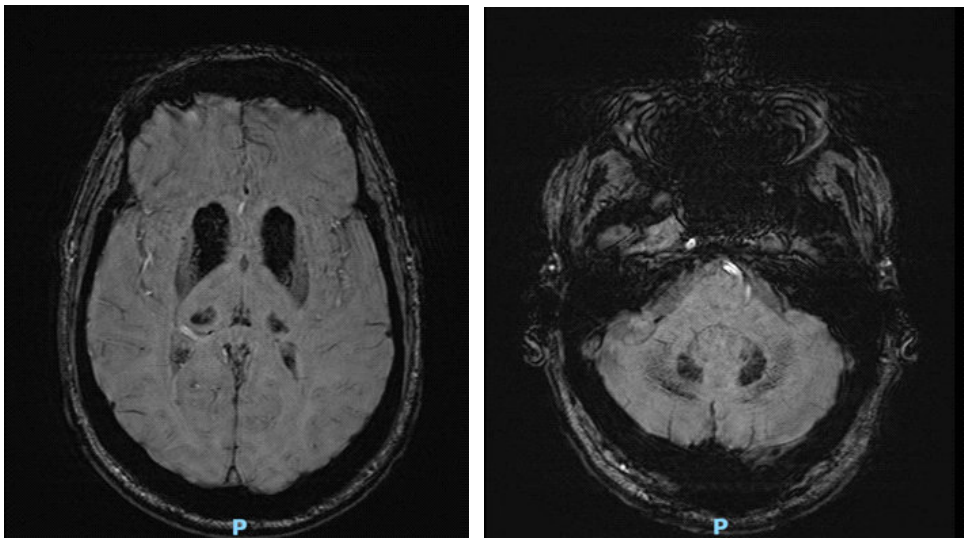

Figure 2. Axial SWI brain MRI sequences.

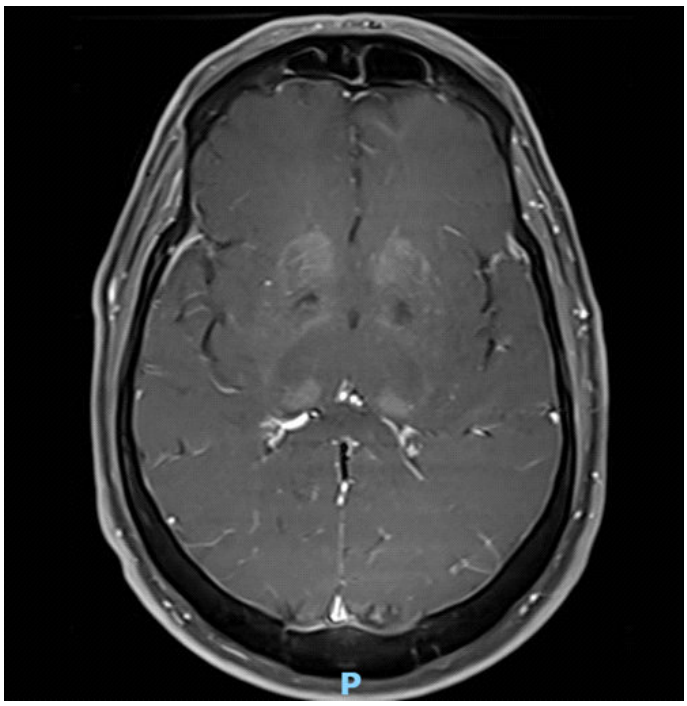

Figure 3. Axial gadolinium-enhanced brain MRI sequence.

Otherwise mild small vessel white matter disease, these signal changes were compatible with calcium deposition mostly in the basal ganglia. Routine 40-minute electroencephalography with sleep and activation tests showed no slowed or epileptiform activity. Secondary laboratory work-up revealed reduced total (4.1, reference range 8.5-10.3 mg/dL) and ionized (0.5, reference range 4.5-5.3 mg/dL) plasmatic calcium, 25-hydroxivitamin D (10.3, reference range 20-50 ng/mL) and parathyroid hormone (14, reference range 15-85 pg/mL) levels, with normal albumin (4.5, reference range 3.4-5.4 g/dL). Phosphate levels were increased (6.4, reference range 2.8-4.5 mg/dL). These findings were compatible with hypocalcemia secondary to primary hypoparathyroidism. Lyme disease (IgM/IgG) and HIV (antigen p24, anti-HIV1/2 IgM/IgG) serologies were negative, alongside VDRL tests. Electrocardiography revealed a long QT interval (490 ms), with normal remaining intervals and wave morphology. Renal ultrasonography revealed no calcium stone deposits. In between consultations (1-2 months) he lost 7 kilos and developed choreic movements of the lower extremities and more extreme gait slowing with movement decomposition and postural instability. Second observation was remarkable for more vigorous hyperreflexia, longstanding tetany-evoking touch-elicited muscle spasms and upper limb Chvostek sign. He was immediately admitted in the hospital for I.V. fluids, calcium gluconate and calcitriol, with remarkable improvement, and discharged one week after on daily calcium carbonate, calcitriol, recombinant parathyroid hormone, clonazepam and sertraline oral formulations. Whole body CT scan did not reveal signs of malignancy. There was no clinical or laboratorial evidence of dysfunction of other glands. Specific genetic testing for primary familial brain calcification (SCL20A2, PDGFRB, PDGFB) and hereditary hypoparathyroidism (CaSR, PTH) was negative, classifying his disease as a Fahr syndrome due to primary idiopathic hypoparathyroidism. He was eventually included in a Phase III parathyroid hormone receptor agonist clinical trial (with the active agent eneboparatide – AZP-3601, against placebo) that showed promise in hypoparathyroidism relative to conventional therapy by elevating serum calcium levels without overtly inducing hypercalciuria or need for concomitant oral vitamin D or calcium supplementations. This therapy showed a successful and lasting response. Dopaminergic therapy was eventually discontinued and repeated brain MRI 12 months later showed near-total remission of the signal changes.

Discussion

Fahr syndrome, also known as bilateral striatopallidodentate calcinosis, is a very rare (<1:1.000.000 prevalence) condition resulting from subcortical deep grey matter nuclei and selective white matter calcium deposition [2]. This leads primarily to dopaminergic neuron dysfunction resulting in bilateral mostly symmetric negative (bradykinesia, rigidity) and sometimes positive (chorea, dystonia) motor manifestations.

They more often show in early-mid adulthood, reflecting progressive mineral deposition, but are frequently preceded by cognitive and psychiatric complaints. Later, if the disorder is left untreated, seizures and encephalopathy/dementia may ensue [3]. The absence of genetic causes (which are varied but mostly monogenic) differentiates it from Fahr disease, a primarily hereditary calcium cerebral accumulation disorder with varying patterns of transmission and penetrance [4]. In Fahr syndrome, secondary phosphorus and calcium sometimes severe metabolism disorders are at fault for the central nervous system manifestations. This may back the more diverse clinical phenotype that is seen and better prognosis when a treatable cause is identified [2,3], as Fahr disease does not currently have a modifying treatment.

Our patient had a subacute onset of some classically considered akinetic-rigid parkinsonian symptoms. Initial exams were negative, but evolution with aggravated and more productive motor (spasms, tetany, chorea) and systemic symptoms (frequent urination, weight loss), alongside treatment unresponsiveness, prompted further investigation. These revealed relevant brain imaging changes, diminished serum calcium and a remaining laboratorial profile compatible with primary hypoparathyroidism. The deficit of parathyroid hormone leads to reduced calcium intestinal absorption, decreasing the calcium/phosphorus ratio, which in turn favors calcium-phosphorus combination and ectopic (including brain) deposition [5]. Dysarthria has been described in Fahr's [6] and probably reflects either chief extrapyramidal [7] or combined dopaminergic-cerebellar dysfuction [8] (calcium deposition along subcortical parts of the cerebellum), as both of these can contribute to bulbar muscle dyssynergia. On the other hand, while chorea can be hypodopaminergic in nature, we find the spasms and tetany already hinted by the initial observation more reflective of neuromuscular plate hyperexcitability, which is not specifically seen in Fahr's, but more frequently and primarily in the context of rapidly evolving hypocalcemia (due to easier neuron sodium influx) [9]. This also manifested in the cardiac system [10]. The complex disease mechanisms of hypoparathyroidism may also explain some urinary symptoms, including increased urination (as seen in our patient), as has been recently studied [11].

We recognize brain CT scans are much more readily available and similarly sensitive in the detection of intracranial calcifications [12] when compared to susceptibility weighted imaging MRI sequences [13]. In this situation and specific case, however, the absence of neuroimaging investigation in the primary care setting and the more broaden diagnostic advantages of the MRI motivated that option in specialized follow-up, particularly when symptoms worsened.

Symmetric involvement of subcortical structures of the brain like the basal ganglia and sometimes white matter of the cerebellum is what is classically found in metabolic disorders with central nervous system involvement [14], reflecting the generalized distribution of these byproducts with latter collection on small vessel-rich structures.

Our patient was successfully treated with calcium reposition and calcitriol at first, then recombinant parathyroid hormone and eventually a novel parathyroid hormone receptor agonist.

Conclusion

This case report serves to sensitize medical teams to order larger laboratory panels and imaging studies whenever presented with acute-onset parkinsonism, parkinsonism plus other atypical symptoms (like more positive/hyperkinetic features, muscle hyperexcitability, systemic symptoms), a roughly symmetric presentation, onset in an early age or unresponsiveness to treatment. In these situations, treatable causes, namely metabolic or endocrinological disorders (i.e. hypoparathyroidism), may sometimes be identified.

Patient Consent

Patient provided consent for publication of this work.

Conflict of Interest

The authors have no conflict of interest to disclose.

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