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Parkinson's Disease Associated With Hypoparathyroidism

Hugh A. Clarke

Boy Frame

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EXTRA-PYRAMIDAL signs such as tremor, rigidity and choreiform movements have been reported to be associated with hypoparathyroidism.\textsuperscript{1} In the review of Bronsky and associates in 1958,\textsuperscript{2} seven of 90 cases of idiopathic and pseudohypoparathyroidism were reported to have features of Parkinson's disease. All of these cases had basal ganglia calcification.

The present authors have recently had the opportunity to observe a patient who developed classic features of parkinsonism many years following thyroid surgery. Metabolic studies were compatible with hypoparathyroidism. Unilateral chemothalamolysis was carried out before the underlying metabolic disorder was detected and resulted in elimination of the tremor and marked reduction of rigidity on the contralateral side. Detection of the underlying hypoparathyroidism was then accomplished and treatment with vitamin D and calcium resulted in disappearance of the tremor and decrease in rigidity of the unoperated side.

CASE REPORT — The patient, M.B., Case No. 102 88 36, was a 72-year-old white housewife who was referred to the hospital for chemothalamolysis because of progressive parkinsonism. Six years before admission a tremor and increasing rigidity began in the right upper extremity and later progressed to the left upper extremity. Gradually a typical parkinsonian masked facies and gait became apparent. The patient experienced increasing difficulty in carrying out simple daily activities such as dressing and eating. Artane to tolerance was given without significant improvement.

The past history was pertinent in that a partial thyroidectomy for goiter had been performed 35 years before admission. Several days following that operation the patient developed symptoms of muscle stiffness and twitching. She was placed on three calcium tablets daily which she took intermittently to the time of admission. There was no history of encephalitis.

The general examination was not remarkable. The skin was oily and the hair soft. There was a well healed thyroidectomy scar. Early cataracts were present. Blood pressure was 120/75. No abnormalities were noted in the examination of the heart, lungs and abdomen.

A mask-like facies and propulsive gait were present. There was moderate cogwheel rigidity and a marked pill rolling tremor in both upper extremities. There was moderate rigidity in the lower extremities, but no tremor. The reflexes were brisk throughout, but no pathological reflexes were present. Sensory examination was normal.

Laboratory studies demonstrated normal hemoglobin, white blood count and urinalysis. Serological test for syphilis (VDRL) was non-reactive. Protein bound iodine was 6.7 mcg/100 ml. Chest x-ray was normal. Skull x-rays, including laminograms, were normal without evidence of intracranial calcification. Electroencephalogram was considered to be

\textsuperscript{*}Division of Neurological Surgery.

\textsuperscript{**}Department of Medicine.
within normal limits. A preoperative pneumoencephalogram showed no evidence of ventricular dilatation or cortical atrophy.

In an attempt to improve the Parkinson’s disease, 0.5 cc. absolute alcohol was injected into the left ventrolateral nucleus of the thalamus following which there was marked improvement in the rigidity and tremor of the right upper extremity.

While convalescing from this injection, the diagnosis of hypoparathyroidism was confirmed. Chvostek was 4 plus active. Serum calcium was 6 and inorganic phosphorus was 5 mg/100 ml. Alkaline phosphatase was 3 Bodansky units. Serum protein electrophoresis was normal. A 24 hour urine sample contained 21 mg/ml of calcium. Therapy with vitamin D 100,000 units and calcium lactate 4 grams daily was initiated. Over a period of three weeks the serum calcium rose to 8.6 mg. and serum inorganic phosphorus fell to 3 mg/100 ml. During this time there was a complete disappearance of the tremor of the left (non-operated side) hand and a reduction in the rigidity. The masked facies and festinating gait remained unchanged. There has been continuing ambulation and patient is now able to dress herself — a task which she was previously unable to do.

DISCUSSION

The available clinical evidence suggests that in certain cases there is a relationship between hypoparathyroidism and the development of basal ganglia symptoms. In Bronsky’s review, five cases with pseudo- and two cases with idiopathic hypoparathyroidism had clinical Parkinson’s disease. All of these cases had intracerebral calcification in the area of the basal and/or in the parietal and occipital areas of the brain. Why hypoparathyroidism in certain instances leads to cerebral calcification and extra-pyramidal syndromes is not known. Presumably, the damage to adjacent brain tissue by the metastatic calcification in the region of the basal ganglia is the causative factor for the symptoms. It is possible that basal ganglia calcification may be present and not detected on skull x-rays. Support for this has been supplied by McMahon and associates who demonstrated perivascular calcification microscopically in an autopsied case of idiopathic hypoparathyroidism which was not visible on routine skull x-rays.

The incidence of basal ganglia calcification and parkinsonism in postoperative hypoparathyroidism is not known. Bennett in a review of 88 cases of basal ganglia calcification reported only two cases which presumably occurred following thyroidectomy with apparent removal of parathyroid tissue. Siglin and associates reported a case of a 52-year-old man who had a thyroidectomy and postoperative hypoparathyroidism. He was later noted to have basal ganglia calcification on skull x-ray. However, there were no symptoms of parkinsonism in this patient.

Danowski and associates are of the opinion that basal ganglia calcification is rare in patients who develop postoperative hypoparathyroidism. The patients who develop postoperative hypoparathyroidism are, in general, diagnosed and treated earlier than patients with idiopathic hypoparathyroidism. Hence, there may be less opportunity for basal ganglia involvement in the postoperative cases. In addition, patients with pseudo-hypoparathyroidism are especially prone to develop metastatic calcification not only in the basal ganglia, but in other soft tissues of the body including muscle and subcutaneous areas.

In the present case there was a 30 year lapse between the thyroid operation (when the parathyroid glands were presumably damaged) and the onset of the parkinsonism. This length of time raises legitimate doubt as to the relationship of
the two diseases in the patient under discussion. It is possible that the hypoparathyroidism might have aggravated an existing idiopathic parkinsonism. The absence of basal ganglia calcification, demonstrable roentgenographically, may also be used as argument against damage to these areas as a result of the hypoparathyroidism. However, as pointed out previously, microscopic calcification of the basal ganglia possibly leading to adjacent nervous tissue damage has been demonstrated microscopically despite its absence on skull x-rays. It is conceivable that poorly treated hypoparathyroidism as in the present case over a period of many years may lead to damage of the basal ganglia and result in parkinsonism.

The strongest evidence that the hypoparathyroidism may have been related to the parkinsonism in the present instance was the gradual disappearance of the tremor and the diminution of the rigidity on the non-operated side following treatment of the parathyroid hypofunction. Other features of the parkinsonism including the masked facies and festinating gait persisted. Perhaps it is not to be expected that biochemical correction of the hypoparathyroidism alone would correct all manifestations of basal ganglia involvement. Permanent histopathologic changes that might be present in these areas would not be altered by correction of the biochemical movement.

It is not suggested that any of the forms of hypoparathyroidism will very often be found in association with parkinsonism. However, from the evidence available, it would seem that occasionally basal ganglia dysfunction may be due to underlying hypoparathyroidism. At a time when operative treatment for Parkinson’s disease is advocated, it is prudent to remember the possible relationship between the two diseases. The presence of basal ganglia calcification would make this association especially worthy of consideration.

**SUMMARY**

The occasional association between Parkinson’s disease and underlying hypoparathyroidism is reviewed. A case is reported in which a parkinsonian syndrome occurred in a patient with long-standing hypoparathyroidism. Evidence is presented which suggests that treatment of the hypoparathyroidism improved certain features of the parkinsonism.

**REFERENCES**
