Multiple Manifestations of Hypoparathyroidism

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It is well known that hypoparathyroidism may present signs and symptoms of mental deficiency, neuroses or psychoses. Diverse neurological manifestations including extrapyramidal symptomatology have been noted as the only presenting signs in the absence of tetany (Fourman, et al[1]; Kaye, et al[2]; Greene and Swanson[3]; Wise and Hart[4]; Dimich, et al[5]; Fonseca and Calverley[6]; Sugar[7]; Lachmann[8]). Convulsions and increased sensitivity to dystonic reactions after the administration of phenothiazines to hypoparathyroid patients have been reported (Schaaf and Payne[9]).

The occurrence of many of these manifestations in a single patient has rarely been reported. The following case typifies such an unusual finding:

A 34-year-old white, nulliparous housewife, developed symptoms of ideas of reference, auditory and somatic hallucinations, flattened affect, depression and irritability, two years prior to being seen by us. The symptoms became more marked, and after one year without treatment she was seen by a psychiatrist and placed on chlorpromazine (CPZ). She developed a dermatitis while on CPZ and medication was changed to perphenazine. On perphenazine the patient had one grand-mal convulsion, and at that point was referred to one of us (J.A.Y.-T.).

There was no family history of endocrinological imbalance, mental retardation or psychosis. Since all hospital records were not available, the historical data, up to the age of eight, was obtained from the patient's mother.

The patient was the product of a full-term pregnancy complicated by toxemia. At the age of 14 days she had several convulsions described as grand-mal in type. She was admitted to a hospital where a diagnosis of rickets was made and she was treated with calcium lactate and vitamin D. At the age of four, a diagnosis of rheumatic fever was made. There was no cardiac sequelae. She had a history of pneumonia at two, six, and eight years of age. Following the last episode of pneumonia, she was given intelligence tests because of poor school performance. On the Stanford-Binet (Form L), she achieved an IQ of 46 (CA. = 8.4, M.A. = 3.8) and was diagnosed as mentally retarded.

The patient had no other major illnesses, operations, injuries or hospitalizations until she was 25 years old. At that time she was hospitalized at a metropolitan teaching hospital which provided us with the following summary:

"Patient complained of dysphonia, dysphagia, paresthesia and spasm of her fingers. Other symptoms and signs were nocturia, polyuria, constipation and a series of major motor convulsions.

"A clinical examination revealed dry and coarse skin, loss of hair and metabolic lenticular cataracts. Gait was described as shuffling, deep tendon reflexes were hypoactive, and Chvostek and Trousseau signs were negative. Electroen-cephalographic dysfunction compatible with seizure disorder, serum calcium of 6.8 mg%, serum phosphorus of 5.0 mg%, a negative Sulkowitch test, low urine calcium and high urine phosphorus values were obtained. The Ellsworth-Howard test was negative. Chest X-ray, IVP, bone survey, small bone series, carotid arteriogram, gastrointestinal series, and lumbar puncture were all within normal limits. Skull X-ray revealed cerebral calcification at the base of the sella turcica.

"A diagnosis of idiopathic hypoparathyroidism was established and she was placed on 30 gm. of calcium and 50,000 units of vitamin D daily. During her treatment at that time, calcium values consistently ran low. Upon discharge, serum calcium was 7.6 mg% and phosphorus 4.1 mg%. She apparently did well on this medication regimen as clinic follow-up notes indicated. At the age of 31 she was married. There is no other significant history until the development of psychotic symptoms at the age of 32."

When examined by us, her diagnosis was idiopathic hypoparathyroidism, mental retardation and schizophrenia; paranoid type. Mental status examination and intelligence testing confirmed the diagnoses. She showed the primary and secondary Bleulerian signs of schizophrenia and on the Wechs-ler-Adult Intelligence Scale, she obtained a full scale IQ of 61. Her scaled subtest scores ranged from two on picture arrangement to seven on digit symbol. A repeated electroencephalogram did not reveal any seizure patterns but did reveal some borderline asymmetries of alpha rhythm. Re-peated laboratory examination at that time revealed a serum calcium of 9.3 mg%, and a serum phosphorus of 3.3 mg%. The remainder of the laboratory evaluation including SGPT, FBS, electrolytes, urinalysis and complete blood count were all within normal limits.

The patient was placed on daily doses of 300 mg. of chlorprothixene, 30 gm. Of calcium daily and 50,000 units of vitamin D four times a week. On this medication she developed a mild form of drug-induced Parkinsonism consisting primarily of some rigidity and tremor, which was treated with trihexyphenidyl. Considerable improvement of her psychiatric symptoms was noted on this regimen. It is important to report that on two occasions where a recurrence of auditory hallucinations and Parkinsonism was manifested, a serum calcium of 7.6 mg% was determined. After increasing her daily calcium intake to 35 gm., the patient stopped hallucinating, and her Parkinsonism disappeared, without decreasing the administration of chlorprothixene.

Discussion

It was unfortunate that no medical history was available in reference to the diagnosis of infantile rickets. Therefore, it is difficult to assert that these convulsions

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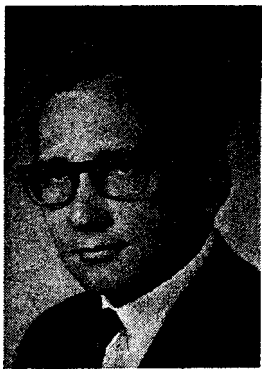

were caused by an underlying hypoparathyroidism that manifested itself first with tetany and later with mental retardation until its definitive diagnosis at the age of 25.

Our observations led us to believe that the multiplicity of symptoms has conducted each previous physician to diagnose the patient according to the outstanding symptom present.

She was treated for rickets, mental retardation, idiopathic hypoparathyroidism with Parkinsonian features, schizophrenia, drug-induced Parkinsonism and convulsions. All of these were probably related to the same underlying dysfunction; that of a calcium disorder.

Some psychological aspects of this case also are important for reviewing. In the past it was believed that mental retardation resulting from hypoparathyroidism was irreversible. Most recent findings suggest that increasing calcium levels will produce some increase in intellectual functioning even after long standing retardation due to hypoparathyroidism (Ehrhardt and Money[10]).

However our patient showed an improvement in IQ in the absence of adequate treatment until age 25, thus suggesting that there can be a spontaneous change in intellectual function without treatment. The adequate social functioning of the patient and her marriage after treatment, confirm the impression reported in the literature, that mental retardation associated with idiopathic hypoparathyroidism is generally not severe (Bronsky, et al.[11]).

One question we ask ourselves: did this patient have the combined syndrome of mental retardation and schizophrenia during her childhood? This syndrome is well known and was described by Bleuler as "pfropfschizophrenia." In our patient, except for psychological testing at the age of eight, a lack of previous psychiatric examination did not permit further elaboration to determine whether a schizophrenic process was grafted on her mental retardation or was an independent event.

Another important aspect is the therapeutic management of this case. Extrapyramidal symptoms and convulsive episodes which are not rare after the administration of phenothiazines (Shaw, et al[12]; Christian and Paulson[13]) were manifested at various points of the patient's life. These symptoms appeared under the presence of low calcemia or after the administration of phenothiazines and as it has been reported that an antagonistic or competitive action between calcium and phenothiazines could exist (Bucci and Johnson[14]), our clinical observation may offer some suggestions in this area. It is known that extrapyramidal symptoms are present in hypoparathyroidism. Therefore the role of calcium metabolism is probably intimately related to

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disturbances of the extrapyramidal system, although results are contradictory (Yaryura-Tobias, et al.[15]).

Previous reports have also indicated that a dystonic reaction to major tranquilizers should suggest the possibility of an underlying hypoparathyroidism (Schaaf and Payne[9]). In our patient an increase of her calcium therapy resulted in a discontinuation of the anti-Parkinson treatment without reducing chlorprothixene intake. However, a single case does not speak for others and further collection of data would be mandatory in order to reach a conclusion.

Finally, it seems imperative to emphasize the need for a complete physical workup in mental patients with the various medical specialities working closer for better diagnosis, care and treatment of patients.

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