In the bad-risk patient it is safer to insert a Riches catheter under local analgesia and to await improvement in the general condition or in the renal function before undertaking prostatectomy.

REFERENCES

- Chapman, T. L. (1949). *Lancet*, 2, 684.
 Riches, E. W. (1943). *Brit. J. Surg.*, 31, 135.
 Ross, J. Cosbie (1936). *British Medical Journal*, 2, 1297.
 Wallace, D. M. (1951). *Proc. roy. Soc. Med.*, 44, 434.

IDIOPATHIC HYPOPARATHYROIDISM PRESENTING AS DEMENTIA

BY

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Idiopathic hypoparathyroidism is a rare condition which presents in a variety of ways and which, once diagnosed, is very amenable to treatment. The subject has recently been fully reviewed (de Mowbray *et al.*, 1954), and we shall therefore confine ourselves to a few comments.

The criteria for diagnosis were discussed by Drake *et al.* (1939). They include chronic tetany, low serum calcium, and high serum inorganic phosphate with normal renal function. Other features often found in these cases are epileptiform attacks; mental changes; cataracts; defects in other ectodermal structures such as dryness and coarseness of the skin, cutaneous calcium deposits, thinness and scanty growth of hair, trophic changes in the nails, and poor dental development, with failure of eruption, faulty enamel, and acalcification of the dentine (experimentally produced in rats by Erdheim in 1911, quoted by Albright and Strock, 1933). Radiographic changes include symmetrical calcification of the basal ganglia and thickening of the cortical layers of the long bones. The aetiology of the condition is still obscure.

The pathology was discussed by Drake *et al.* (1939), who found that in one case at necropsy all four parathyroid glands were present and appeared macroscopically normal, but consisted histologically of an excessive number of fat cells, replacing the normal epithelial cells. In 1946 Leonard (quoted by Albright and Reifstein, 1948) described a case in which the parathyroid glands were absent. Occasionally idiopathic hypoparathyroidism was found to be associated with other endocrine disorders such as Addison's disease or hypothyroidism. More often, monilia infection of the nails and mucosae was observed.

Cases occur in any age group, but the majority appear to have been found either in childhood or between 38 and 52 years. Our case is of interest because of the late onset of the symptoms and the mode of presentation.

Case Report

The patient, a widow aged 61, was admitted to the Whittington Hospital on February 11, 1954, with a diagnosis by her doctor of status epilepticus. No reliable history could be obtained either from her or from her relatives, but it seemed that her symptoms had begun seven years before admission, shortly after the death of her husband,

with depression and gradually increasing loss of interest in her surroundings and her appearance. Some time after the onset bilateral cataract operations had been performed. For several years she had occasionally complained of numbness and tingling in the legs. In 1951 she had attended the out-patient department of the National Hospital, Queen Square, with giddiness and tremors of the hands; mild anxiety symptoms were observed, and an x-ray film of the skull showed calcification of the basal ganglia. Although asked to attend again, she had failed to do so and was not seen again at any hospital until her present admission. Since 1952 there had been episodes of urinary incontinence, and "fainting spells" had occurred in the past six months; twitching of the limbs had been observed in the past three months. For the past five weeks there had been considerable mental deterioration, with confusion and loss of memory. The first epileptiform attack had occurred two days before admission. From the description given by the relatives this had consisted of generalized tonic and clonic contractions preceded by a cry and followed by two hours' sleep. Several similar attacks had occurred in the next forty-eight hours.

On arrival in hospital the patient was conscious but disorientated, apathetic, and unable to concentrate. She attempted to co-operate in the examination, but could not give any details of her illness or carry on a rational conversation. Her general condition was one of extreme neglect; she was thin and pale, and doubly incontinent. The heart, lungs, and abdomen were normal and the blood pressure was 150/95. The only teeth present were the upper four incisors, which were misshapen and carious. She had a slurring dysarthria and a fine lateral nystagmus. Bilateral iridectomies had been performed and both lenses had been removed. The fundi showed choroido-retinal degeneration; there was no papilloedema. All tendon reflexes were diminished and neither ankle-jerk could be obtained; both plantar responses were feebly extensor. Otherwise the central nervous system was normal.

During the first few days in hospital atypical epileptiform attacks were witnessed, in which a hoarse cry was followed by tonic contractions involving the whole body. Between these the patient lay in bed showing no kind of initiative and had to be fed and washed. It was observed that she frequently adopted a position of flexion at the large joints of the arms and legs, with occasional writhing movements of the arms. No improvement was obtained with phenobarbitone. On the twelfth day of her stay in hospital definite attacks of tetany with carpal spasms were first seen. Chvostek's sign was positive; biochemistry at this time was as follows: serum calcium, 6.5 mg. per 100 ml., repeated the following day, 5.1 mg.; serum inorganic phosphate, 10.4 mg. per 100 ml.; and serum alkaline phosphatase, 4.5 King-Armstrong units. Renal investigations, estimation of faecal fats, and alkali reserve were normal.

FIG. 1.—Radiograph showing calcification of basal ganglia.

X-ray examination of the skeleton showed slight cortical thickening of the long bones, some periosteal new bone formation in the fibulae, and calcification of the basal ganglia (Fig. 1). The two upper eighth and one upper seventh molar teeth were unerupted. An electrocardiogram (Fig. 2) showed the prolonged Q-T_c interval found in hypocalcaemia, and low T waves in the standard leads. There was no evidence of

FIG. 2.—Electrocardiogram on February 26, showing Q-T_c prolonged (0.45 second). Serum calcium, 5.8 mg. per 100 ml.

previous thyroid operation, and the diagnosis of primary or idiopathic hypoparathyroidism was made.

Treatment was begun on the same day with an intravenous injection of 10 ml. of 10% calcium gluconate, with no apparent immediate effect on the muscular spasms. In addition, oral treatment with 5 ml. of dihydrotachysterol (A.T.10), and 60 gr. (4 g.) of calcium lactate daily, was started. On the next day no further generalized spasms occurred, but the carpal spasms continued at short intervals, and the dose of dihydrotachysterol was increased to 20 ml. daily. The patient's general condition began to improve, and on the following day the carpal spasms ceased.

Chvostek's sign remained positive to a diminishing degree for more than four weeks. By that time she had ceased to be incontinent, was able to walk normally, and spent most of the day out of bed. Her mental state had improved

FIG. 3.—Results of modified Ellsworth-Howard test.

FIG. 4.—Response of serum calcium and phosphates to treatment.

to a remarkable degree. She was normally oriented and took an intelligent interest in her surroundings and in her own progress.

In the first two weeks of treatment the improvement in the biochemical changes did not keep pace with the clinical recovery. The initial rise of serum calcium was slow, despite high dosage with more than one batch of freshly obtained dihydrotachysterol. The serum calcium levels remained below 6 mg. per 100 ml. during the first and below 7 mg. during the second week. We considered the possibility of pseudo-hypoparathyroidism (Albright *et al.*, 1942; Reynolds *et al.*, 1952). After discontinuing dihydrotachysterol for three days, a modified Ellsworth-Howard test was carried out (Fig. 3). This showed that an intramuscular injection of 40 units of "para-thor-mone" was followed by a normal rise in urinary phosphate excretion, and there was therefore no support for this diagnosis. Resumed treatment with dihydrotachysterol eventually resulted in a slow restoration of the serum calcium to near-normal levels, aided possibly by a high protein intake of approximately 130 g. daily (Jordan and Kelsall, 1951). The serum inorganic phosphate had fallen, but remained somewhat above normal levels (Fig. 4).

In the fourth week of treatment it became possible to reduce the dose of dihydrotachysterol to 3 ml. daily; later calciferol-forte, 1 tablet daily (=50,000 units=1 ml. dihydrotachysterol) was substituted for reasons of convenience for the patient, and she was eventually discharged from hospital on this maintenance dose on April 20, 1954.

FIG. 5.—Electrocardiogram on March 22, showing Q-T_c normal (0.35 second). Serum calcium, 9.2 mg. per 100 ml.

because tests for urinary calcium excretion with Sulkowitch's reagent often gave misleading results. On several occasions the tests indicated normal serum calcium levels when in fact these were below 7 mg. per 100 ml.

Four weeks after the beginning of treatment the electrocardiogram had become normal (Fig. 5).

An electroencephalogram at that time showed a slow dominant activity of about 6 cycles per second, and the record was not of epileptic significance (Dr. Douglas Gordon). Of the various electroencephalographic changes found with hypoparathyroidism, slow waves, of about 2-5 c/s, have been associated with low serum calcium. They occur singly or in series, with occasional spiking, and remain unchanged when a transient rise of the serum calcium level is produced by intravenous injection of a calcium salt; they tend to disappear when the serum calcium is permanently maintained at normal levels (Gotta and Odoriz, 1948). In

our case the electroencephalogram was done after the elevation of the serum calcium level, and the abnormal waves were not seen.

Some months after treatment had begun horizontal ridges appeared across the finger- and thumb-nails (Fig. 6).

Discussion

It has been emphasized previously that idiopathic hypoparathyroidism may simulate idiopathic epilepsy or cerebral tumour. In our case this condition presented as dementia. The common feature of these three modes of presentation is the incidence of epileptiform attacks.

Generalized muscular contractions in hypocalcaemia have been thought to originate in one of three ways. (1) The low serum calcium level may precipitate epileptic attacks in latent coexisting epilepsy with electroencephalographic changes (Jordan and Kelsall, 1951). (2) Cerebral oedema due to low serum calcium may provoke epileptiform attacks which subside with adequate treatment of the hypocalcaemia but are not abolished by phenobarbitone. There is experimental and necropsy evidence for the production of such oedema. Perfusion of a frog's leg with a solution containing a low concentration of calcium chloride caused oedema, which did not appear with higher concentrations (Hamburg, quoted by Albrecht, 1923). Cerebral oedema was found at necropsy in children who had died of infantile tetany (Grant, 1953). The epileptiform attacks in our case may have been of this type; the electroencephalogram after elevation of the serum calcium was not of epileptic significance, and there was no improvement with sedatives, but prompt relief with dihydrotachysterol. (3) Increased peripheral nervous irritability may cause widespread tetanic spasms, which may resemble attacks of Jacksonian epilepsy.

Occasionally epileptiform fits in hypoparathyroidism have been associated with papilloedema and raised pressure of the cerebrospinal fluid, and have then simulated cerebral tumour. In our case there was no papilloedema, and the pressure of the cerebrospinal fluid was normal.

Like many other endocrine diseases, hypoparathyroidism may be complicated by mental changes, but in the present case these were predominant, and the initial differential diagnosis seemed to lie between the organic dementias, with epileptiform attacks amongst the associated features. After exclusion of the commoner types, the provisional diagnosis of pre-senile dementia of the type of Pick or Alzheimer was made. Hypocalcaemia was not at first suspected because the usual appearance of tetany with carpo-pedal spasm was absent; later carpal spasm was observed, but even then true main d'accoucheur did not develop.

The diagnosis of idiopathic hypoparathyroidism in the absence of frank tetany may be suggested by calcification of the basal ganglia (Camp, 1947), which, although not confined to this condition, is found in a high proportion of cases. Confirmation is afforded by the unequivocal biochemical changes.

Summary

A case of idiopathic hypoparathyroidism of late onset is described, and its mode of presentation as dementia is emphasized. Other cerebral manifestations of this condition, as well as some diagnostic features, are mentioned. Treatment—started with dihydrotachysterol, continued with calciferol, and controlled with serum calcium estimations—resulted in a striking improvement in the mental state.

We would like to thank Dr. A. L. Jacobs for his help and permission to report the case; and Dr. N. Gordon for information about the patient's first attendance at hospital.

ADDENDUM.—The patient has been seen in the out-patient department since this paper was written, and she has remained well. Her mental state is normal, there have been no epileptiform attacks and no recurrence of tetany, and she has gained weight. The serum calcium has been kept

FIG. 6.—Finger-nails four months after starting treatment, showing a transverse ridge halfway along each nail.

between 7.5 and 9 mg. per 100 mL. However, towards the end of May, 1954, about three months after the beginning of treatment, she began to exhibit involuntary movements of the left arm and leg. They were choreiform, and, apart from sudden facial grimaces, strictly unilateral. Dr. Michael Ashby, neurologist to the Whittington Hospital, stated that they were virtually identical with those seen bilaterally in Huntington's chorea, especially in regard to the fleeting grimaces. These involuntary movements must be regarded as being due to a lesion in the basal ganglia. Although this is the site of the calcification in hypoparathyroidism, such movements have not, so far as we are aware, been previously recorded in this condition. It is possible that the arteriosclerosis in our patient, who is older than most of those whose cases have been reported, is a factor in producing this interesting new feature.

REFERENCES

- Albrecht, K. (1923). *Msschr. Psychiat. Neurol.*, **55**, 55.
 Albright, F., Burnett, C. H., Smith, P. H., and Parson, W. (1942). *Endocrinology*, **30**, 922.
 — and Reifenstein, E. C. (1948). *The Parathyroid Glands and Metabolic Bone Disease*. Williams and Wilkins Company, Baltimore.
 — and Strock, M. S. (1933). *J. clin. Invest.*, **12**, 974.
 Camp, J. D. (1947). *Radiology*, **49**, 568.
 Drake, T. G., Albright, F., Bauer, W., and Castleman, B. (1939). *Ann. intern. Med.*, **12**, 1751.
 Gotta, H., and Odoriz, J. B. (1948). *J. clin. Endocr.*, **8**, 674.
 Grant, D. K. (1953). *Quart. J. Med.*, **22**, 243.
 Jordan, A., and Kelsall, A. R. (1951). *Arch. intern. Med.*, **87**, 242.
 de Mowbray, R. R., Smith, S. H. L., and Symonds, W. J. C. (1954). *British Medical Journal*, **1**, 903.
 Reynolds, T. B., Jacobson, G., Edmondson, H. A., Martin, H. E., and Nelson, C. H. (1952). *J. clin. Endocr.*, **12**, 560.

PSYCHIATRIC ILLNESS IN THE ELDERLY

A FOLLOW-UP STUDY

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The increasing expectation of life has important implications for medicine, and not least for psychiatry, since the incidence of mental illness increases with advancing years. Lewis (1946), dealing with this problem, concluded that mental disorders of the elderly would be responsible within the next thirty years for the bulk of patients admitted to mental hospitals. The present study is a further examination of this problem, and adds to those of Gallinek (1948) and of Roth and Morrissey (1952), who have pointed out that affective disorders are far commoner in old age than was previously supposed, are distinguishable from states due to organic senile change, are treatable, and carry a good prognosis. We are here concerned with the recognition of such states, the results of treatment, and the length of hospitalization required as compared with organic senile states.

Material and Method

In 1951, 119 patients aged 65 or over (see Table I) were admitted to Crichton Royal; 12 of these had had previous admissions. This is the material of the present study. Cases have been assigned to three major diagnostic groups (senile dementia, cerebral arteriosclerosis, and affective disorder) and three minor diagnostic groups (schizophrenia, delirious states, and mixed conditions). The diagnostic grouping of patients who had died or had been discharged when this study was begun was decided from perusal of the case records. All cases were followed up at six, twelve, and

twenty-four months after admission. Those still in hospital, or readmitted, were personally interviewed. Those discharged were followed up through correspondence with the family doctor and relatives, through hospital reports, or from a social worker's visit. The clinical state was classified under the following headings: (1) dead; (2) still in hospital; (3) home, but invalided; and (4) home, with social recovery—that is, resumption of previous life and occupation. Sex seemed to have no significant effect on the clinical outcome, and no sex differential is therefore made in the follow-up tables.

Diagnostic Findings

The most important finding was that differentiation of the affective disorders from the organic psychoses presented little difficulty on the whole.

Major Group

Affective Disorder.—In 32 out of the 33 cases this was a depressive syndrome, usually with agitation and presenting with delusions of guilt and unworthiness, paranoid ideas in tune with the mood, loss of appetite and sleep, with such hypochondriacal complaints as numbness and tension in the head. There had been no evidence of mental abnormality before the depression, and intellectual functions were retained. Although recent memory often appeared defective, this was attributable to morbid preoccupations which interfered with the attention, and memory became normal with clinical improvement. Orientation was correct, except in two patients who showed mild confusion. In only two cases had habits deteriorated. The onset of illness had been fairly definite, and was commonly associated with psychological or physical trauma. Its duration before admission had varied from two weeks to one year, with an average of eighteen weeks. A family history of depression was frequent, and five patients had attempted suicide before admission. Of the 33 cases, 25 were in their first admission, and 8 had been previously admitted before the age of 65.

Senile Dementia (51 cases).—These presented a different picture, characterized by intellectual deterioration and confusion, and disorganization of the personality, nocturnal restlessness and wandering, and some delirium at one time or another in most cases. The typical affect was a shallow euphoria occasionally disturbed by irritability or rage. Delusions were frequent but unrelated to the mood, and were transient and variable, in line with the confusion. Onset of illness could not be accurately defined: there had been a drift from senescence to psychosis; but, so far as duration could be gauged, this was much longer than in affective cases—averaging three years, varying from two months to six years. Intellectual functions were not retained, grasp and recent memory were impaired, while confabulation was frequent. On testing, Goldstein's (1952) catastrophic reaction was frequently seen—a rapid change from good humour and co-operation to opposition, agitation, and tears. Orientation was impaired, habits had often deteriorated. No suicidal attempts had occurred, and this finding is in line with those of Batchelor and Napier (1953), who concluded that in elderly people a clear-cut depressive state comprises the setting in which the majority of suicidal attempts occur (80% in their series).

Cerebral Arteriosclerosis (13 cases).—As these cases carry better prospects of remission than those of senile dementia and can often be differentiated therefrom on Rothschild's (1947) criteria, an attempt at differentiation was made, notwithstanding the view of Robertson and Browne (1953) that such differentiation is possible only on necropsy. To reduce the possible error, cases are diagnosed as arteriosclerosis only when there is also an unmistakable history of cerebro-vascular accident. The clinical picture resembles that of the senile dementia group in the prominence of intellectual deterioration, with shallow euphoria accompanied by emotional lability, but differs from it in its fluctuating course, relative preservation of the personality and insight, and a history of cerebro-vascular accident.