Treatment with vitamin E will make the resistance normal and even increase it (Tollerz and Lannek, 1964).

The possibility exists that patients with malabsorption syndrome have an increased susceptibility to the toxic effects of free iron, and this needs further study.

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Idiopathic Addison's Disease Presenting with Hypercalcaemia

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Though Addison's disease is occasionally cited as a rare cause of hypercalcaemia (Watson, 1966), the association is not mentioned in several of the most authoritative textbooks of medicine. This report concerns a case of idiopathic Addison's disease which presented with hypercalcaemia.

CASE REPORT

A childless 24-year-old housewife was admitted to hospital in May 1969. At Easter she had begun to develop generalized muscular pains and feelings of faintness. She then became increasingly depressed and subject to outbursts of tears, complained of lethargy, anorexia, and bouts of nausea and vomiting, and more recently had developed polyuria, thirst, and nocturia and a liking for salty food. She had noticed muscular pains in the thighs and lower abdomen and had experienced difficulty in sitting up in bed. In childhood she had undergone craniotomy for raised intracranial pressure. Her past history was otherwise normal and there was no relevant family history. On examination she was depressed. She was not anaemic and had neither buccal nor generalized pigmentation. Corneal calcification was absent. The blood pressure was 95/70 mm. Hg. Physical examination was otherwise negative.

Investigations.—E.S.R. 25 mm./hour, blood picture normal. Paul–Bunnell test negative. Urine, routine *x*-ray films, liver biopsy, and sternal marrow were all normal. Plasma urea 125 mg./100 ml.; sodium 128 mEq/l.; bicarbonate 19 mEq/l.; chloride 94 mEq/l.; potassium 5·2 mEq/l.; calcium 11·8 mg./100 ml. (plasma S.G. 1024); phosphorus 5·2 mg./100 ml.; alkaline phosphatase 6 K.A. units/100 ml.; serum uric acid 9·9 mg./100 ml.; and creatinine clearance 25 ml./min. Twenty-four hour collection of urine contained protein 100 mg., calcium 90 mg., urea 12 g., chloride 36 mEq., sodium 40 mEq., and potassium 37 mEq. Urinary S.G. after overnight fluid deprivation was 1024. Two-hour postprandial blood sugar was 56 mg./100 ml. Antibody studies showed positive cytoplasmic fluorescence for adrenal antibodies, a high titre of thyroglobulin and thyroid cytoplasmic antibodies, and also the presence of gastric parietal cell antibodies. Plasma protein-bound iodine was 7·8 μg./100 ml. and serum vitamin B_{12} 250 pg./ml.

Hydrocortisone 40 mg. t.d.s. was given by mouth. Ten days later plasma calcium, urea, and electrolytes were all restored to within normal limits—the calcium falling in five days, which was an unexpectedly rapid response—the creatinine clearance had risen to 67 ml./min., and her symptoms had disappeared. On discontinuing the hydrocortisone her original symptoms recurred within a week and the previous biochemical abnormalities returned. Plasma cortisol values at midnight and 9 a.m. were 3·9 and 4·5 μg./100 ml. respectively and failed to rise above these subnormal values in response to 50 units of corticotrophin given intramuscularly on three successive days.

Replacement therapy with cortisone acetate 37·5 mg. and fludrocortisone 0·1 mg. daily was instituted and since then she has remained in excellent health. Plasma calcium, urea, and electrolytes were repeatedly within normal limits after steroid therapy was begun.

COMMENT

Pedersen (1967) pointed out that the clinical pictures of adrenal insufficiency and hypercalcaemia may be identical. In the present case the alterations in plasma calcium paralleled closely those in the clinical state. The mechanism of production of hypercalcaemia is unknown. Hypercalcaemia is a frequent occurrence after adrenalectomy for Cushing's syndrome (Sprague *et al.*, 1953). On the other hand, cortisone will lower plasma calcium in certain hypercalcaemic states (Connor *et al.*, 1956).

It is stressed that in this patient the original symptoms mimicked those of hypercalcaemia so closely that Addison's disease was not initially considered as a possible diagnosis. Hence our use of the standard hydrocortisone test in the differential diagnosis. The patient improved so rapidly during the test that our suspicions were aroused and then strengthened when no common cause was found for the hydrocortisone-responsive hypercalcaemia. This situation is very rare. It is the first time that it has been noted at University College Hospital, where the differential diagnosis of patients with hypercalcaemia is a frequent problem.

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