Medical Memoranda

Panhypopituitarism after Cured Tuberculous Meningitis

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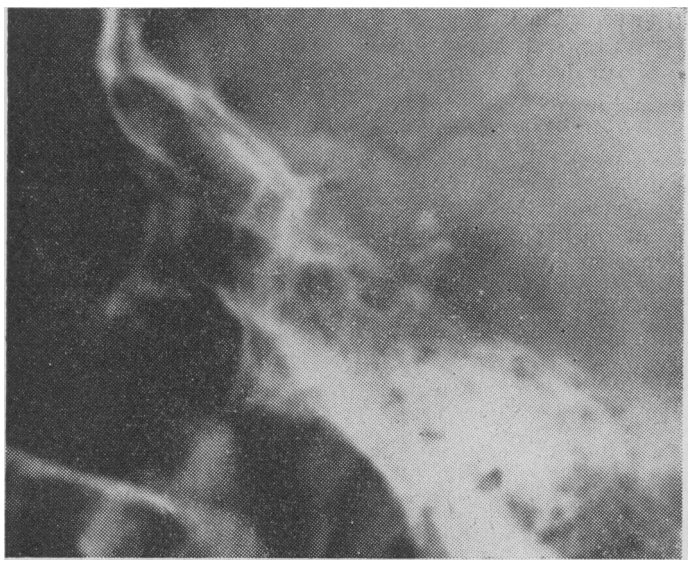
Though tuberculous granulomata of the pituitary or its stalk are often included in the lists of causes of hypopituitarism, most of the cases have in fact been diagnosed only at necropsy. Chronic clinical hypopituitarism due to proved tuberculosis has rarely been reported.

Case Report

The patient had developed tuberculosis of the lungs, spine, and meninges when she was aged 6. Streptomycin therapy was given for two years and she was discharged from hospital after a further year. The menarche occurred at age 10; the periods lasted for only four cycles, though lower abdominal pain at monthly intervals continued for a further six months. Thereafter there was no cyclical pain or bleeding.

At age 20 she was investigated elsewhere for amenorrhoea; she was short of stature (145 cm.) and no signs of active tuberculosis were obtained. A vaginal smear showed no evidence of oestrogenic activity. Curettage was attempted but no endometrium could be obtained from the small uterus. Cyclical oestrogen therapy was started and resulted in enlargement of the breasts and uterus and withdrawal bleedings. On discontinuing the therapy no spontaneous periods occurred.



X-ray film of skull to show suprasellar calcification, presumably in the meninges.

At age 27 she developed a sore throat, pyrexia, and hypotension (B.P. 90/60) and rapidly lapsed into a stuporous condition. No evidence of raised intracranial pressure was found. In view of the history and the finding of very scanty pubic and axillary hair she was treated with intramuscular hydrocortisone as well as ampicillin. A rapid recovery ensued and therapy was temporarily withheld while laboratory tests were performed.

Investigations.—The plasma cortisol was 3.4 and 2.4 μg./100 ml. at 10 a.m. on two occasions. The urinary daily excretion of 17-hydroxycorticosteroids and 17-oxosteroids was 2.3 and 1.5 mg. respectively. On the day 3 g. of metyrapone was administered orally the 17-hydroxycorticosteroid excretion was 2.8 mg. and on the following day it was 2.0 mg. Injection of A.C.T.H. gel (20 units b.d. for three days) increased the 17-hydroxycorticosteroid excretion to 20.5 mg./24 hours. The serum protein-bound iodine was 2.9 μg./100 ml. and the serum thyroxine 4.3 μg./100 ml. The four-hour ^{132}I uptake was 11.0%. This was increased to 29.2% after 10 units of thyroid stimulating hormone. The urinary excretion of gonadotrophins was less than 3 i.u./24 hours. The total urinary oestrogen excretion was 16 μg./24 hours. There was no polyuria and serum electrolytes were normal. *X*-ray examination of the skull showed suprasellar calcification (see Fig.).

After treatment with cortisone and thyroxine the patient felt much more alert and active and less cold-sensitive.

Comment

The laboratory investigations confirm the diagnosis of hypopituitarism. The lack of evidence suggestive of a space-occupying lesion and the presence of suprasellar calcification make it almost certain that the hypopituitarism followed the tuberculous meningitis.

Tuberculosis has been reported to affect both the parenchyma of the anterior lobe and the meninges around the hypothalamus and stalk. Since the menarche in the present case occurred some years after the active stage of the meningitis, it seems unlikely that parenchymatous tuberculosis was responsible, and the slow development of the hypopituitarism would rather suggest chronic scarring and calcification in the region of the stalk, the *x*-ray appearances being consistent with this pathogenesis.

Post-mortem cases of tuberculosis of the pituitary region are reviewed by Sheehan and Summers (1949) and by Rickards and Harvey (1954). A common cause of death in this group was hypopituitary coma. Reports of clinical cases of hypopituitarism which followed tuberculosis and which were confirmed by adequate laboratory tests are rare. Durlach *et al.* (1959) described the case of a man aged 29 with pituitary insufficiency in the course of pulmonary tuberculosis. He was treated with streptomycin but died in hyperthermia. Permission for necropsy was not obtained. Courvoisier *et al.* (1960) reported the case of a man aged 45 who had suffered for 28 years from tuberculosis at many sites. Diabetes insipidus and testicular atrophy developed and was followed later by panhypopituitarism.

We have been unable to find in the literature any description of a case exactly comparable to the present one, though a case of chronic hypopituitarism occurring after purulent meningitis was described by Wetherbee and Turner (1963).

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