isolated from the stools or urine. Biochemistry, chest *x*-ray picture, and E.C.G. were normal.

Epidemiology.—The patient was infected with *Salm. typhi* with an untypable Vi strain. Her landlady was shown to be a carrier of the disease and two phage types of *Salm. typhi* were isolated from her. She had an untypable Vi strain and also a type A. The landlady's brother had an attack of typhoid some years previously in Sweden, and this was reported at the time to be a *Salm. typhi*, phage type A.

Comment

Until 1923 there had been only 30 cases of breast abscess in typhoid in the world literature, and this led Madelung (1923) to comment on its extreme rarity. Klose and Sebening (1930) subsequently suggested that 0·3% of typhoid patients develop mastitis, but it was not until Pezinski (1937) studied 1,196 cases of typhoid over a period of two years that the relevant statistics became available.

In Pezinski's series, in fact, 0·5% of the patients developed a breast abscess, and among the females the incidence was over 0·9%.

Erbslöh (1954) defined a typhoid breast abscess as one which arose in a case of the generalized disease and emphasized the difficulty in isolating the organism, the constant finding of a W.B.C. below 13,000/mm^3, and that it is a late complication, being predisposed to by physiological breast activity. He also reaffirmed Madelung's finding that the skin over the abscess is not reddened and that in the pus one usually finds a mixed growth and not pure *Salm. typhi.*

In the present case the patient was nulliparous, and apart from a three-week history of fever had no other manifestations of typhoid. The skin over the abscess was reddened, and after the drainage procedure a pure growth of *Salm. typhi* was grown from the wound. The relatively low W.B.C. agreed with the findings of Erbslöh.

In conclusion, in cases of a breast abscess occurring where there are no predisposing factors (breast feeding, pregnancy, etc.) it follows that an underlying general disease should be considered. Clearly, such a diagnosis could easily be missed if the possibility of such a condition was not entertained.

We wish to express our gratitude to Professor H. E. de Wardener and Mr. B. P. Bliss for permission to report this case, Dr. Bubna-Kasteliz for help with the translation, Dr. Hilary J. Andrews and the Department of Bacteriology, Fulham Hospital, and Dr. E. S. Anderson, Enteric Reference Laboratory, Colindale.

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Anorexia Nervosa Associated with Hypothalamic Tumour

K. LEWIN, D. MATTINGLY, R. R. MILLIS

British Medical Journal, 1972, **2**, 629–630

The well-recognized signs and symptoms associated with hypothalamic lesions include both cachexia and obesity. So far as we know, however, true anorexia nervosa has not been described. The present report describes a case presenting clinically as anorexia nervosa, where at necropsy a small neoplasm of the hypothalamus was found.

Case Report

A 25-year-old New Zealand nurse was admitted to the Royal Devon and Exeter Hospital with a history of secondary amenorrhoea, anorexia, and weight loss. Eighteen months earlier she had left New Zealand for a working holiday in Europe knowing that her father had an inoperable bronchial carcinoma. Her periods, previously regular, stopped shortly thereafter but her weight remained steady at about 118 lb (53·5 kg). Six months before admission her father died, after which she developed an aversion to any form of carbohydrate and lost weight. Despite this she remained active and continued to work. During this time she noticed an increased growth of hair on her face and trunk, developed pitting oedema of the feet, and became severely constipated. One month before admission she was admitted to another hospital with a left basal pneumonia, which was successfully treated with antibiotics and physiotherapy. Until this illness she had been in good health apart from an attack of glandular fever three years previously.

Clinically she was bright and cheerful despite her emaciated appearance (68 lb, 30·8 kg) and seemed unaware of the gravity of her illness. Her skin was dry and scaling, with increased growth of fine downy hair on her face and back. She had a sinus bradycardia, a blood pressure of 100/80 mm Hg, and minimal ankle oedema. No abnormal neurological signs were found. Routine investigations showed no abnormality apart from a serum potassium of 3·3 mEq/l. and a borderline serum protein-bound iodine of 3·8 μg/100 ml. Her fasting blood sugar was 64 mg/100 ml. There was evidence of increased adrenocortical activity; an overnight urine specimen contained 940 μg of 11-hydroxycorticoids (normal range 80-320 μg/24 hr), and a morning plasma 11-hydroxycorticoid level was 77·8 μg/100 ml (normal range 6-24 μg/100 ml). The chest *x*-ray picture was normal and a barium-meal examination showed a slowly emptying atonic stomach. A diagnosis of anorexia nervosa was made, thought to have been precipitated by her father's death.

At 3.30 a.m. five days after admission she suddenly became unconscious. She was pale and sweating, incontinent of urine, and had generalized convulsions. Her blood sugar was less than 40 mg/100 ml and she was given glucose intravenously. The convulsions stopped but she remained in coma. Although her blood sugar was maintained at normal levels by intravenous dextrose infusions she never regained consciousness and died two weeks later from a salmonella infection.

Necropsy Findings.—The patient was severely emaciated, with complete absence of subcutaneous fat. The lungs showed severe purulent bronchitis and a confluent bronchopneumonia. Culture of sputum grew *Salmonella typhimurium.* The heart and liver showed brown atrophy. The pituitary was normal on gross examination. Microscopically the cells of the anterior pituitary were mostly of the sparsely granulated type and eosinophils were scant. There were a moderate number of Crooke's hyaline mucoid cells. The adrenals showed cortical lipid depletion. The thyroid, parathyroids, and islets of Langerhans were normal. Except for the brain the viscera appeared normal. The brain contained a small circumscribed nodule measuring 0·5 cm in diameter on the inferior surface of the hypothalamus posterior to the optic chiasma and immediately posterior and to the right of the tuber cinereum (Fig. 1). Microscopical examination showed a well-demarcated but non-encapsulated tumour consisting of a dense fibrillary network of glial fibres among which were many

Department of Pathology, Stanford University Medical Center, Stanford, California, U.S.A.

K. LEWIN, M.D., M.R.C.PATH., Assistant Professor (Formerly Senior Registrar, Area Laboratory, Royal Devon and Exeter Hospital)

R. R. MILLIS, M.B., B.S., M.R.C.PATH., Visiting Assistant Professor

Postgraduate Medical Institute, University of Exeter and Royal Devon and Exeter Hospital

D. MATTINGLY, M.B., B.S., F.R.C.P., Director and Consultant Physician

mature astrocytes. The glial fibres merged with the hypothalamus at the margin of the tumour (Figs. 2 and 3). The material was sent to Professor P. M. Daniel, who confirmed the diagnosis of a well-differentiated astrocytoma surrounded by a normal hypothalamic bed. The rest of the brain showed no abnormality.

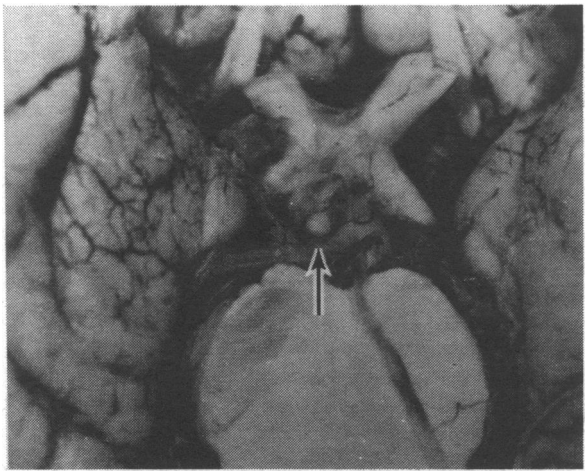

FIG. 1—Under surface of brain showing hypothalamic tumour (arrowed) lying between optic chiasma and midbrain. Above optic chiasma are inferior surfaces of frontal lobes and, laterally, temporal lobes. (× 7.)

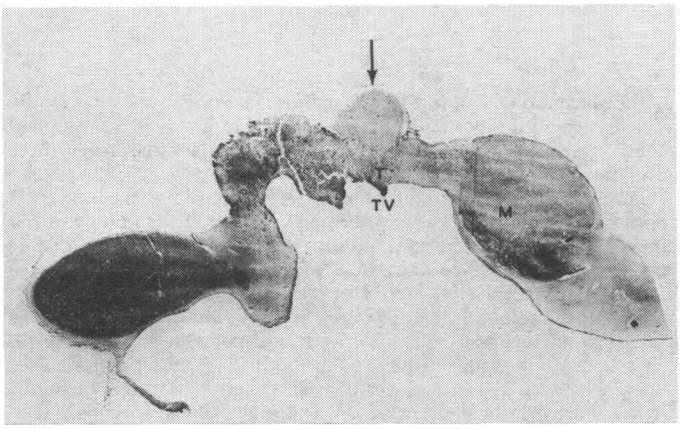

FIG. 2—Histological section of hypothalamus cut in saggital plane showing relation of tumour (arrowed) to other structures of hypothalamus. M = Mammillary body. T = Tuber cinereum. O = Optic chiasma. TV = Third ventricle. (Haematoxylin and eosin. × 5.)

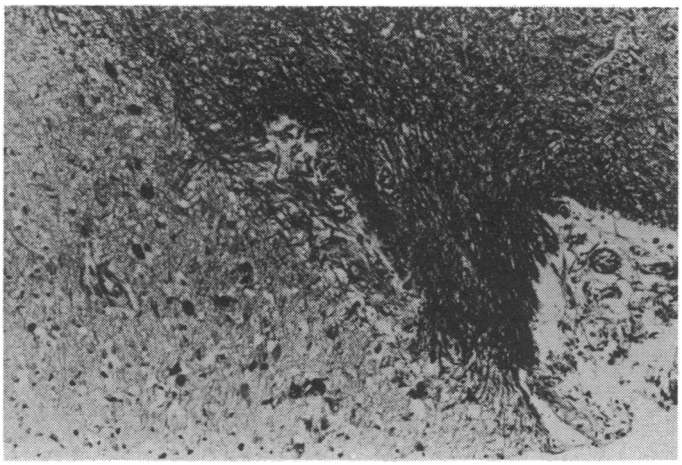

FIG. 3—High-power magnification of junction of tumour and normal tuber cinereum showing dense fibrillary network in tumour in which are embedded numerous astrocytes. (Phosphotungstic acid haematoxylin. × 110.)

Comment

In the few necropsy reports on anorexia nervosa in the literature the predominant findings were those of inanition and overwhelming infection, although in some instances death was due to cardiovascular disorders or suicide (Kay and Leigh, 1954; Mosli, 1967). In no case was the state of the hypothalamus mentioned, and so far as we are aware there have been no detailed neuropathological studies. In view of the close similarity of some cases of hypopituitarism to anorexia nervosa (Escamilla and Lisser, 1942) many reports have been devoted to discussion of the possible relation of the two entities (Sheldon, 1939; Wilson, 1954). Many conflicting opinions have been expressed about the changes in the pituitary and whether these are primary or secondary to inanition. Consistent changes have not been shown, however, and in some cases the changes seen suggest adaption of the endocrine system to the stress of inanition. This is well shown in the case described by Siebenmann (1955) of a 27-year-old woman in whom there was amenorrhoea due to atrophy of the ovaries, hypofunction of the thyroid, and increased secretory activity of the adrenals. With the exception of a slight increase of acidophils the pituitary was normal. The present case is almost identical to that one in that the patient was amenorrhoeic and had a low serum protein-bound iodine level and a greatly increased adrenocortical function, as shown by raised plasma and urinary hydroxycorticoid levels and the state of the adrenals at necropsy. Further support for this was the finding of Crooke's hyaline change in the pituitary, a change associated with conditions of adrenocortical hyperactivity.

Although it can be debated whether functional lesions of the hypothalamus produce anorexia nervosa it is well recognized that tumours of the hypothalamus can cause emaciation and may simulate anorexia nervosa. Sooner or later, however, features of the tumour become apparent. Thus, White and Hain (1959) reported the case of a 62-year-old woman diagnosed clinically as anorexia nervosa who was found at necropsy to have a destructive lesion of the hypothalamus. There were, however, several atypical features. The age of the patient was wrong, she had a normal menstrual history, and she was an alcoholic with psychotic symptoms long antedating her anorexia. Brain tumours are a rare cause of anorexia in childhood but the picture does not really resemble anorexia nervosa.

The hypothalamic lesions probably produce this symptomatology by acting on the feeding centre, which has been shown by animal experiments to lie in the lateral hypothalamus (Anand and Brobeck, 1951). The mechanism by which the lesion in the present case might have produced anorexia is obscure. The tumour was fairly close to the midline and did not directly involve the feeding centre, but it may have affected its function indirectly. However, we cannot rule out that the lesion was merely a coincidental finding. Further careful necropsy examination of the hypothalamus in cases of anorexia nervosa may be rewarding as small lesions may well have been overlooked in the past.

We thank Professor P. M. Daniel, Dr. G. F. M. Russell, and Professor L. J. Rubinstein for their helpful suggestions and discussion of the case.

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