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Lesson of the Week

Occult intracranial tumours masquerading as early onset anorexia nervosa

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Any boy with apparent anorexia should have careful anthropometric and endocrine assessment as well as cranial imaging, which may need to be repeated

Childhood onset anorexia nervosa may be difficult to diagnose because of the lack of clear diagnostic criteria for prepubertal children and because of the difficulty of differentiating psychogenic from organic disease.¹ Psychological disturbance and symptoms of anorexia without neurological manifestation may also be presenting features of an early intracranial lesion, usually affecting the diencephalon—as shown by the following three cases.

Case reports

CASE 1

An 8½ year old boy presented with a six month history of intermittent headaches and vomiting. Clinical examination was unremarkable. A provisional diagnosis of migraine was made after unenhanced computed tomography of the brain showed nothing abnormal. Seven months after presentation the initial symptoms had settled but the patient had developed anorexia. Onset of physical symptoms coincided with the patient becoming introverted, emotionally labile, and periodically morose. His mother had died from a hepatocellular carcinoma when he was 3 years. The combination of unresolved grief, his father being admitted to hospital for a routine operation, and his eldest sister leaving home were thought to have precipitated this change in personality. He could not be coaxed into eating and was referred to a child psychiatrist, who diagnosed childhood onset anorexia nervosa with depression. Over the next nine months the patient continued solely under psychiatric follow up but with negligible improvement in weight gain or stature. His home environment was thought to be contributory, and, in view of these concerns, he was placed on the child protection register.

The patient was subsequently admitted to a second psychiatric unit for re-evaluation. Although clinically depressed, he was unresponsive to three months of psychotherapy. An endocrine opinion confirmed growth arrest both in terms of stature and weight. Despite having prolonged "anorexia," he had normal skinfold thicknesses. Formal pituitary function tests showed isolated growth hormone deficiency and a high serum prolactin concentration of 841 mU/l (normal ≤ 350 mU/l). Magnetic resonance imaging of the brain showed a craniopharyngioma. This was successfully resected, and the patient made an excellent recovery with immediate improvement in his mental state and resolution of his anorexia.

CASE 2

A 13 year old boy presented with a six month history of anorexia, nausea, difficulty in swallowing, and

associated weight loss. Clinical examination and investigations, including barium studies, were normal. Both the patient's mother and maternal aunt had a history of anorexia nervosa in their adolescent and early adult years. The patient's mother had had symptoms for four years but eventually recovered when she met her future spouse. Although the boy had no apparent fear of fatness or distortion of body image, childhood onset anorexia nervosa was diagnosed after he had been referred to a child psychiatrist. He then underwent a stringent behaviour modification programme in hospital. During this period he was noted to have persistently dilated pupils, but visual acuity and peripheral fields were normal. There was, however, transient swelling of the optic discs. Unenhanced computed tomography of the brain showed only calcification of the pineal gland.

Nine months later the patient's condition deteriorated: he was extremely lethargic; he refused to eat, resulting in further weight loss; and he would vomit effortlessly, usually around meal times. He had also developed a right divergent squint but was not complaining of any other visual symptoms.

Psychiatric re-evaluation 10 months after presentation showed no evidence of a primary psychiatric disorder. Visual assessment confirmed a variable right divergent squint due to blindness in that eye and associated optic atrophy. Endocrine evaluation showed statural growth arrest, cortisol insufficiency, biochemical diabetes insipidus, and a high serum prolactin concentration of 7800 mU/l. Magnetic resonance imaging showed extensive enhancement of the optic nerves and chiasm, the hypothalamic area, the lower medulla, and upper spinal cord, as well as periventricularly. There was also considerable enhancement of the pineal gland. Diagnosis of a disseminating pineal germinoma was confirmed by stereotactic brain biopsy. The patient had an excellent clinical and radiological response to craniospinal irradiation and gained 12 kg in the six months after treatment.

CASE 3

A 7 year old boy was referred to our hospital with a three year history of progressive anorexia, recurrent episodes of vomiting, coughing, and discomfort on swallowing, and subsequent failure to thrive. Onset of symptoms had coincided with his father leaving home and his stepfather moving in with the family. Extensive investigation showed no organic cause for his symptoms, and computed tomography showed no abnormality, although there was no imaging below the level of the foramen magnum. Although his symptoms were atypical for anorexia nervosa, the patient was thought to have a psychological eating disorder and received

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intensive psychiatric input, including a three month period as an inpatient. At the age of 9 years he developed vertical nystagmus. His gait had also become increasingly unsteady and school performance deteriorated. Tests of hypothalamopituitary function were normal. Magnetic resonance imaging showed a cervicomedullary tumour, which proved to be a low grade astrocytoma.

Discussion

Tumours affecting the hypothalamus (cases 1 and 2) and, rarely, those impinging on the brain stem (case 3) are recognised as causing both psychological dysfunction and anorexic symptoms without initial neurological signs.² Diagnostic confusion with early onset anorexia nervosa has also been reported.² In all three cases, there were coexistent psychosocial factors. Although intracranial disease was considered early on, this was thought to have been excluded by normal brain imaging. The computed tomography scans of all three patients were reviewed, and, even with hindsight, were considered to be normal. In particular, there was no suprasellar calcification to suggest a craniopharyngioma (case 1), and in case 2 the calcified pineal gland was no more extensive than that found as a normal variant in up to 30% of pineal glands by the age of 15.³ Although scans of all three patients were unenhanced, magnetic resonance imaging may have proved more discriminating as an initial imaging technique. A high index of suspicion needs to be maintained even with normal neuroradiology, particularly in the presence of atypical clinical features, failure of response to treatment (case 1), or the development of new symptoms or signs (cases 2 and 3).

Childhood onset anorexia nervosa is rare. It is particularly unusual in boys, although several studies suggest that males may comprise a higher proportion (19 to 30%)^{4,5} of cases in childhood than they do in adolescence and adulthood (5 to 10%).⁶ Over the four years that these three cases presented we have seen a total of 20 boys referred with an eating disorder. Children may present with a variety of somatic symptoms, including feelings of nausea or fullness and

difficulty in swallowing. Depressive symptoms may occur relatively early in children as weight loss tends to be more rapid and emaciation more profound.⁷

Careful anthropometric and endocrine assessment may give additional clues to the diagnosis. Although starvation states such as anorexia nervosa will stunt statural growth as well as weight gain, the finding of normal skinfold thicknesses (as in case 1), and thereby adequate subcutaneous fat stores, would be inconsistent with such a diagnosis.

In general, therefore, the diagnosis of childhood onset anorexia nervosa should not be entertained unless there is an overt and relentless pursuit of thinness, with an expressed aversion to putting on weight. Had these criteria been applied in the first two cases the diagnosis of anorexia nervosa could not have been made. Even when the diagnosis seems certain, however, a high index of suspicion should be maintained and re-evaluated as the presence of an eating disorder does not necessarily exclude a concurrent organic diagnosis. We recommend that any boy with anorexia should have careful anthropometric and endocrine assessment as well as neuroimaging, which may need to be repeated.

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Conflict of interest. None.

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A MEMORABLE PATIENT

Postmortem caesarean section

I was just about to start an antenatal clinic when I was bleeped by the casualty consultant. He said that a woman who was 28 weeks pregnant had been found at home having hanged herself. The ambulancemen had performed cardiopulmonary resuscitation on her all the way to hospital in an attempt to keep the baby alive. "Would you like to deliver the baby? The mother is dead."

My thoughts were a little difficult to organise as I ran the 400 yards to the casualty department but ranged from, "This is pointless" to "I've got nothing to lose." When I arrived the situation was much more poignant. In the room were the two ambulancemen, two anaesthetists, three casualty senior house officers with the consultant, and at least four nurses. All eyes turned to me in anticipation.

"We are only carrying on until you make a decision. The woman is dead. Can you deliver the baby?"

I asked for a Pinards stethoscope to listen to the fetal heart, though more as a mechanism to give me time rather than in the hope of hearing anything. Under the weight of expectation I did not feel that I could do anything but deliver the baby. I took off my white coat, rolled up my sleeves and removed my tie. I was given a pair of gloves and offered a knife with a 10 blade (a very small blade). I made an incision and then reflected the bladder before incising the uterus. I delivered a very small infant who was handed to the anaesthetist to resuscitate until a

paediatrician arrived. I asked for a suture to close the patient but at this point everything stopped from the maternal point of view. Resuscitation was stopped and I was told by the consultant that there was no reason to sew up and was gently taken away from the patient.

I later discovered that the patient had been in hospital four weeks before after an attempted suicide by poisoning that was severe enough to require ventilation in the intensive care unit. She had recovered from this and had been discharged by the duty psychiatrist. Her problems seemed to have stemmed from being declined a termination of pregnancy in the first trimester.

The baby was transferred to neonatal intensive care and did well until a brain scan on the second day showed no brain activity and after discussion with the father intensive care was discontinued. Whether it was the first or second insult that accounted for the baby we will never know.

Two consultants gave differing responses to the case. One was extremely supportive, and for that I am grateful, agreeing that I had had no choice but to make the delivery. He had arrived shortly after the event. A second consultant, although not directly known to me, let it be known that he felt that the outcome was always bad in such cases and that I should not have intervened. Who knows who was right?—DEREK J TUFFNELL is a consultant obstetrician and gynaecologist in Bradford