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case report

A case of male anorexia with Klinefelter's syndrome, 22 years later

Christopher Paul Szabo

In 1986 the *British Journal of Psychiatry* published a report 'A case of anorexia nervosa with Klinefelter's syndrome' (Hindler CG, Norris, DL. *Br J Psychiatry*; **149**: 659–660). The case was of interest as it was the first documented case of anorexia nervosa in a male associated with Klinefelter's syndrome. The patient had initially been diagnosed as having 'atypical anorexia nervosa' and the Klinefelter's syndrome was cytogenetically proven. No other associated organic pathology was diagnosed at that time. Specifically, computed tomography (CT) scans were reported within normal limits.

Clinically, the patient had presented (in 1985) with significant weight loss, from 50 kg to 39.9 kg over the preceding 2 years. He had a history of prior admission to the unit (7 years earlier, at the age of 13) with a diagnosis of anorexia nervosa. In addition to the weight loss, he reported vomiting five times per week in the 2 months before the admission, but not as a consequence of binge eating or guilt following perceived overeating. The vomiting was not self-induced. The patient denied any fear of fatness, reporting a loss of appetite, excessive fluid intake and persistently feeling cold (features not inconsistent with anorexia nervosa). His limb length, sexual characteristics and delayed puberty suggested he had Klinefelter's syndrome.

Following the admission in 1985, a magnetic resonance image (MRI) scan was undertaken due to the onset of an ataxic gait, tremor and noticeable nystagmus. A cerebellar tumour was diagnosed, identified as an 'atypical teratoma' during a subsequent neurosurgery. Because of local infiltration and incomplete tumour excision, a course of radiotherapy followed the surgery. Thereafter, the patient gained 5.5 kg over the first 3 months and a further 3.5 kg in the next 6 months to weigh 51 kg. His recovery was generally satisfactory. However, in January 1988 his speech began to slur and he became uncoordinated. A CT scan revealed a tumour in the right frontal lobe. Following surgery (with adjuvant radiotherapy), a germinoma was diagnosed. Review of the pathology findings concluded that both tumours had indeed been germinomas.

Between 1988 and 2000 a number of MRI scans did not reveal any recurrent tumour growth. The patient's last gadolinium-enhanced MRI scan (2005) showed no evidence of tumour recurrence in either the right frontal lobe or posterior fossa, and generalised cerebral and cerebellar involutional changes, possibly secondary to previous radiotherapy. At the most recent consultation (2006), the patient reported no eating-related concerns and he had no symptoms of an eating disorder.

Given that both tumours (frontal and cerebellar) were diagnosed as germinomas, it may be that although the cerebellar tumour had been the one initially diagnosed, the one in the frontal lobe had been there all along. With hindsight it appears possible that the initial clinical features were more harbingers of the pathology that was ultimately diagnosed, rather than of an eating disorder. Features of a developing neurological disturbance were noted during the various admissions to the eating disorders unit. However, they were not as prominent as the weight loss and vomiting which, together with the diagnosis of Klinefelter's syndrome and the initial negative CT scans, may have deflected attention. The reported absence of the more complex features of psychopathology usually associated with an eating disorder further suggests a more likely primarily neurological pathology in evolution.

The case highlights a specific clinical issue – the clinical interpretation of disordered eating and the distinction of such behaviour from an eating disorder.

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The British Journal of Psychiatry (2008) 193, 388. doi: 10.1192/bjp.193.5.388

https://doi.org/10.1192/bjp.193.5.388 Published online by Cambridge University Press