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Brief Reports

Paedophilia and Hyperprolactinaemia

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The case of a man presenting with paedophilia who has found to be hyperprolactinaemic is described. There is possibly a link between paedophilia and endocrine disorders.

Men with hyperprolactinaemia have a high incidence of sexual problems, with loss of libido, erectile dysfunction, and ejaculatory failure occurring in 50–90% (Carter *et al*, 1978; Franks *et al*, 1978; Bouloux & Grossman, 1987). Moreover, raised prolactin was discovered in 6% of men attending a clinic for sexual dysfunction (Schwartz *et al*, 1982), indicating an important but poorly understood role for prolactin in male sexual behaviour (Drago, 1984; Bancroft, 1988). Separate work suggests that hypothalamo–pituitary–gonadal axis (HPGA) dysfunction may be a feature of some paedophiles, who exhibit an elevated leutinising hormone (LH) response to leutinising-hormone releasing hormone (LHRH) when compared with non-paedophile paraphilics and controls (Gaffney & Berlin, 1984). Furthermore, men with Klinefelter's syndrome have similar LH release profiles (Smals *et al*, 1976), raised prolactin (Schiavi *et al*, 1978), and may also have an increased incidence of paedophilia (Mosier *et al*, 1960; Neilsen, 1972; Wakeling, 1972), although this is controversial (*Lancet*, 1988).

Case report

The patient, a 29-year-old storeman, was referred for a psychiatric court report after admitting to indecent assault over a four-year period against a girl who was six when the offences began in 1983. These took the form of kissing and fondling, after which he would masturbate, although with impaired potency and ejaculation. Sexual interest was only partly directed towards this girl, in that he also reported sexual fantasies involving adult women; he therefore satisfies the DSM–III–R criteria (American Psychiatric Association, 1987) for non-exclusive paedophilia.

He was educated in a remedial class, leaving school without qualifications and having a number of unskilled jobs. He is a shy, solitary man whose main interest is making models. His one adult sexual relationship, which started in 1981, lasted two years, ending before his offending began. Sexual difficulties were partly responsible for the end of the relationship, as reduced libido, erectile dysfunction, and ejaculatory failure had prevented successful intercourse. Previously, he had masturbated with full potency and ejaculation. In 1983 he also required circumcision for balanitis, at which time diabetes mellitus was diagnosed, and oral hypoglycaemics commenced. Past medical and psychiatric history, and family history, are unremarkable apart from maturity-onset diabetes in both parents.

At assessment he was noted to be obese with a somewhat high-pitched voice. There were no abnormal neurological signs or evidence of hypogonadism. Chromosomal analysis showed a normal male karyotype. An endocrine screen revealed a raised prolactin level (2398 mU/l, $n = 0–450$) and low testosterone level (10 nmol/l, $n = 14–42$). LH, follicle-stimulating hormone (FSH), thyroid function tests, liver function tests, electrolytes, and full blood count were all normal. He was not on any medication known to raise prolactin levels. Blood glucose averaged 6–9 mmol/l with a glycosylated haemoglobin of 9%. Investigation of hyperprolactinaemia included skull and pituitary X-rays, computerised tomography of the cranium, dexamethasone suppression test, and plasma and urinary cortisol, which were all normal. Neuropsychological testing demonstrated an IQ of 80 with a verbal IQ of 77 and a performance IQ of 91.

Although the time of onset of hyperprolactinaemia was unknown, it was considered that the raised prolactin was likely to have contributed to the offence in some way (see below), and that his prognosis in terms of reoffending would be good if his prolactin were normalised. After a short time on remand awaiting trial, he was sentenced to three years' probation with a condition of treatment. A period of psychiatric and endocrine in-patient care has been followed by out-patient management. Bromocriptine (2.5 mg t.d.s.) returned prolactin to normal within six weeks. On follow-up, prolactin levels have remained normal, although testosterone remains slightly low (11 nmol/l). Over this period there was a return of libido, erectile response, and ejaculation, and sexual fantasies were claimed to be solely towards adult women. However, this period also coincided with release from custody, and his reporting of sexual matters may have been influenced by this, as well as by a wish to be seen to respond to treatment. On follow-up, he continues to deny paedophile interests and is in full-time employment, but there has been little progress in terms of establishing adult sexual relationships.

Discussion

To our knowledge this is the first report of hyperprolactinaemia presenting in association with paedophilia, and as such complements the above findings relating this sexual disorder to other endocrine abnormalities. Clearly, no causal

connection can be inferred from a single case, but several issues are raised.

Hyperprolactinaemia affects all three components of male sexual performance (libido, arousal, and ejaculation), but it is unclear which of these is a primary effect and which may be non-specific sequelae. Bancroft *et al* (1984) suggest that loss of libido is caused directly by hyperprolactinaemia, whereas the erectile dysfunction is a psychological reaction to the reduced sexual interest. Drago (1984), reviewing animal data, concludes that ejaculatory failure is more important, noting, however, that the effects of raised prolactin on male sexual function are complex and variable.

One explanation for our patient's offences might be, therefore, that the onset of hyperprolactinaemia precipitated overt sexual dysfunction and failure to maintain adult sexual relationships through both direct and indirect mechanisms. Sexual dysfunction, combined with his pre-morbid personality (characterised by shyness and social isolation) and limited intelligence, would have led him to perceive a sexual relationship with a child as less threatening. Increased anxiety and somatisation have been reported in hyperprolactinaemic men (Fava *et al*, 1982) and could have contributed to the sexual dysfunction, but these symptoms were not observed here. A more specific link between hyperprolactinaemia and paedophilia involving alterations in HPGA function, as described by Gaffney & Berlin (1984), cannot be ruled out.

In view of this case, together with the evidence indicating a connection of some kind between pituitary dysfunction, sexual problems, and paedophilia, an endocrine screen including prolactin should be considered as part of the assessment of paedophilia. To what extent the finding of a raised prolactin level should constitute a mitigating circumstance in terms of sentencing remains unclear. The 'medicalisation' of child sexual abusers is a contentious area, and the delineation of subgroups who have demonstrable organic or psychological problems which can be related to the offence may help identify boundaries of medical involvement.

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References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

BANCROFT, J. (1988) Sexual desire and the brain. *Sexual and Marital Therapy*, 3, 11–27.

——, O'CARROLL, R., MCNEILLY, A., *et al* (1984) The effects of bromocriptine on the sexual behaviour of hyperprolactinaemic man: a controlled case study. *Clinical Endocrinology*, 21, 131–137.

BOULOUX, P. M. & GROSSMAN, A. (1987) Hyperprolactinaemia and sexual function in the male. *British Journal of Hospital Medicine*, 37, 503–511.

CARTER, J. N., TYSON, J. E., TOLIS, G., *et al* (1978) Prolactin-secreting tumours and hypogonadism in 22 men. *New England Journal of Medicine*, 299, 847–852.

DRAGO, F. (1984) Prolactin and sexual behaviour: a review. *Neuroscience and Biobehavioural Reviews*, 8, 433–439.

FAVA, M., FAVA, G. A., KELLNER, R., *et al* (1982) Psychological correlates of hyperprolactinaemia in males. *Psychotherapy and Psychosomatics*, 37, 214–217.

FRANKS, S., JACOBS, H. S., MARTIN, N., *et al* (1978) Hyperprolactinaemia and impotence. *Clinical Endocrinology*, 8, 277–287.

GAFFNEY, G. R. & BERLIN, F. S. (1984) Is there hypothalamo–pituitary–gonadal dysfunction in paedophilia? *British Journal of Psychiatry*, 145, 657–660.

LANCET (1988) Klinefelter's syndrome. *Lancet*, *i*, 1316–1317.

MOSIER, H. D., SCOTT, L. W. & DINGMAN, H. F. (1960) Sexually deviant behaviour in Klinefelter's syndrome. *Journal of Paediatrics*, 57, 479–483.

NIELSEN, J. (1970) Criminality among patients with Klinefelter's syndrome and the XYY syndrome. *British Journal of Psychiatry*, 117, 365–369.

SCHIAVI, R. C., OWEN, D., FOGEL, M., *et al* (1978) Pituitary gonadal function in XXY and XYY men identified in a population survey. *Clinical Endocrinology*, 9, 223–239.

SCHWARTZ, M. F., BAUMAN, J. E. & MASTERS, W. H. (1982) Hyperprolactinaemia and sexual disorders in men. *Biological Psychiatry*, 17, 861–876.

SMALS, A. G. H., KLOPPENBERG, P. W. C., LEQUIN, R. M., *et al* (1976) The effect of gonadotrophin releasing hormone on pituitary–gonadal function in Klinefelter's syndrome. *Acta Endocrinologica*, 83, 829–838.

WAKELING, A. (1972) Comparative study of psychiatric patients with Klinefelter's syndrome and hypogonalism. *Psychological Medicine*, 2, 139–154.

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