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MENTAL ILLNESS, EPILEPSY AND HYPOTHYROIDISM IN XXYY SYNDROME

DEAR SIR,

I am reporting a case of XXYY syndrome, associated with psychotic episodes, epilepsy and hypothyroidism. The patient is a nineteen year old male with the clinical features of XXYY syndrome, confirmed by cytological investigations.

His height is 194 cms. His intellectual assessment showed him to be in the low average range of intelligence (IQ varied from 72 to 88). There is no familial or prenatal history available, as the patient was adopted at five years of age. There is a history of epilepsy following a febrile illness at six months and a further convulsion at the age of five years following a smallpox vaccination. An EEG confirmed that he suffered from temporal lobe epilepsy with both grand mal and partial attacks.

The patient first came to the attention of psychiatric services when he was ten years old. The reasons for referral were slow learning, marked fantasy flights, behaviour difficulties and a very high degree of anxiety and tension within the family. At the age of fourteen years his problems increased: his behaviour became worse, he developed transvestite fantasies and started stealing ladies underwear. After being admitted to a special school, he started to steal money from other residents, became argumentative, at times depressed, and attempted suicide by trying to cut his wrists.

Since his admission to a mental hospital in 1983 his mental state is gradually improving and he is now placed in a small hostel and attending the hospital occupational therapy department. His epilepsy is fairly well controlled. Psychotic disorder and EEG abnormalities in the XXYY Syndrome have been described by Jancar (1968).

In addition to abnormal mental functioning and epilepsy, it was noted that the patient tended to be sleepy. His pulse rate was 56 per minute, total thyroxine was 64 mol/l and his free thyroxine index was 61 units. Treatment for hypothyroidism started with L-thyroxine 0.1 mg daily to which the patient responded well.

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PSYCHOSIS AND ANTIDIURETIC HORMONE

DEAR SIR,

We were interested in the letter from Emsley and Gledhill (*Journal*, March 1984, **144**, 331–2) and would like to make the following observations.

The possibility of a *common* neurological/hypothalamic disturbance was considered in our paper but discounted in the absence of abnormal drinking behaviour and fluctuation in body weight, with an appropriate ACTH response to hypocortisolaemia. However our patient's prolactin level was normal (370 mU/L n.r. <450 m U/L). This is of interest since in acutely adrenalectomised rats serum prolactin levels are elevated (Ben-David *et al*, 1971) suggesting lack of dopaminergic inhibition. Pituitary dopamine and dopamine metabolite concentrations have, however, been found to be normal in adrenalectomised rats (Leung *et al*, 1980). The mechanism of hyperprolactinaemia is unexplained, but our patient's prolactin level possibly implies an isolated disturbance of hypothalamic function.

While we agree that both present and past phenothiazine therapy may be misleading in interpreting antidiuretic hormone (ADH) levels it seems unnecessary to postulate a drug effect in the presence of such profound hypoadrenalism, with salt and water loss. Moreover Raskind *et al* (1978) whom they cite as evidence for serum ADH elevation in psychosis only discontinued drugs for 4 weeks before the ADH levels were taken.

We would suggest that further studies to investigate the relationship between ADH *per se* and psychosis will need to demonstrate that (a) variations in ADH levels correspond to the course of the psychosis, (b) CSF ADH levels as well as serum ADH levels are raised during psychosis, (c) these disturbances are