

Letters to the editor

The abuse of thyroxine in a psychiatric setting

D Sloan, M Hannon, J O'Boyle

St Vincent's Psychiatric Hospital, Dublin 3, Ireland

A 35-year-old woman with a history of bipolar affective disorder was admitted to our acute psychiatric unit with typical features of mania. She was irritable, overtalkative, and sexually disinhibited. Of note, she had been diagnosed as having lithium induced hypothyroidism and had been started on thyroxine replacement 50 µg tds, 4 months previously. Physical examination was normal apart from a resting tachycardia of (100 beats/min). Biochemical testing revealed a T₄ of 180 nmol/L (70–145) and thyroid stimulating hormone (TSH) of 0.2 mIU/L (0.3–5.0); a pattern consistent with hyperthyroidism. It was discovered that she had been abusing her thyroxine medication and had in fact consumed forty × 50 µgm tablets in the 48 hours before her admission. She required treatment with intramuscular droperidol, and oral haloperidol, and thyroxine was discontinued. Her manic symptoms subsided within 5 days and she was discharged. Thyroxine was started again and the patient returned as an outpatient.

The association between lithium therapy and hypothyroidism is well documented (Persad et al, 1993), as is the complex relationship between disturbance of thyroid hormone homeostasis and affective disorders (Gadde et al, 1994). Rapid administration of thyroxine can cause an abrupt augmentation in catecholamine sensitivity, and this combined with an augmented response of beta adrenergic receptors is postulated to be the neurochemical basis for mania in thyrotoxicosis (Lee et al, 1991). Although it has been previously shown that thyroxine replacement therapy for hypothyroidism can precipitate mania in patients with bipolar affective disorder (Whybrow, 1994), the abuse of thyroxine in this setting has not previously been reported to our knowledge. Although this phenomena may be very rare, astute clinical observation and appropriate laboratory tests are called for to detect its existence and provide appropriate intervention.

REFERENCES

- Gadde KM, Krishnan K, Ranga R. Endocrine factors in depression. *Psychiatr Annals* 1994;24:521–4
Lee S, Chow CC, Wing YK et al. Mania secondary to thyrotoxicosis. *Br J Psychiatry* 1991;159:712–3
Persad E, Forbath N, Merskey H. Hyperthyroidism after treatment with lithium. *Can J Psychiatry* 1993;38:599–602

Whybrow PC. The therapeutic use of triiodothyronine and high dose thyroxine in psychiatric disorder (Review). *Acta Medica Austriaca* 1994;21:47–52

Unpredictable neuroleptics induced priapism: a case report

M Razali Salleh¹, H Mohamad², J Zainol²

¹Department of Psychiatry, ²Department of Surgery, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia

Priapism is defined as a pathologically prolonged painful erection of the penis involving the corpora cavernosa, while the corpus spongiosum and gland of penis remain flaccid. The erection is usually unrelated to sexual excitement or stimulation, although some cases have occurred after prolonged sexual activity. In review of the literature, Thompson et al (1990) found that 54 of 261 reported cases of priapism or prolonged erection associated with psychotropic medication were related to antipsychotics. Among the antipsychotics, chlorpromazine which has prominent α-blocking side effects was commonly associated with priapism.

Psychiatrists should be alerted to the infrequent side effects of psychotropic medication. Priapism is no longer considered an extremely rare side effect of psychotropic drugs. Patients prescribed potent α-blocking neuroleptics should be warned of the potential side effect of the drug. Once priapism develops, it should be considered a urologic emergency.

CASE REPORT

A 22-year-old single Malay man, a known case of schizophrenia for three years was seen at a follow-up in a district hospital. He complained of persistent painful penile erection for one week. He was on regular medication for his schizophrenia with chlorpromazine 100 mg tds and haloperidol 5 mg nocte since his last discharge from the psychiatric ward about 6 months ago. There was no history of prolonged erection in the past.

The patient was calm and cooperative. There was no significant finding in mental status examination. Clinical examination revealed an erect penis of normal skin colour. The diagnosis of schizophrenia in remission with priapism was made. Medication was stopped and the patient was sent back home because the family refused surgical intervention.

The next day the patient presented to the Accident and Emergency (A&E) department of the University Sains Malaysia (USM) Hospital because he could not tolerate the pain. He was then admitted for irrigation and drainage of the carvenosa under general anaesthesia. Detumescence was achieved, however on the second post-operative day re-evacuation of clot by aspiration was necessary due to recurrence of priapism. The patient was well on discharge and found to have normal erection on follow up.

The exact mechanism of priapism remains uncertain. The final pathway in the development of priapism regardless of the offending agent or disease process is the obstruction of the venous drainage from the corpora cavernosa of the penis. One of the mechanisms responsible for this action is increased parasympathetic tone in relation to sympathetic tone via direct α -blockade (Kogeorgos and de Always, 1986). The onset of priapism in our patient was difficult to predict. He had no history of prolonged erection which occurs in 50% of the cases before the actual onset of priapism. The dosage of anti-

psychotic and length of treatment did not have a significant relationship with the onset of priapism (Thompson et al, 1990). In contrast, trazodone-related priapism appears to be much more predictable in regard to dosage and length of treatment (Warner et al, 1987).

Further research on the mechanism of penile tumescence and detumescence, and the effect of neuroleptics on these mechanisms is necessary to detect high-risk cases. It is hoped that priapism associated with neuroleptics may become a totally preventable complication in future.

REFERENCES

- Kogeorgos J, de Always C. Priapism and psychotropic medication. *Br J Psychiatry* 1986;149:241-3
- Thompson JW, Ware MR, Blashfield RK. Psychotropic medication and priapism: a comprehensive review. *J Clin Psychiatry* 1990;51:430-3
- Warner MD, Peabody CA, Whiteford HA. Trazodone and priapism. *J Clin Psychiatry* 1987;48:244-5