Priapism is a Pathologically prolonged and painful penile erection, usually unaccompanied with sexual desire. Any painful erection lasting for more than twelve hours is considered priapism. Pathophysiology involves obstruction of the venous drainage from the corpora cavernosa, while that of corpus spongiosum and glans penis remains unaffected, (Kogergos and de Alwis 1986), with the result that only corpora cavernosa gets turgid, while corpus spongiosum and glans penis remain flacid (Wasmer et al 1981). Prompt diagnosis is crucial to safeguard the future sexual functioning of the individual. The intervention has to be surgical and immediate and in the hands of a competent urologist. It is essential to drain the blood clots from corpora cavernosa. Delay may cause permanent and irreversible erectile impotence (Pryor 1982).

In 50% cases of priapism cause is unknown (idiopathic). In the remaining, various causes have been attributed like blood dyscrasias, especially with increased blood viscosity, direct traumatic injury to penis, local pelvic Pathology and rarely drugs, which possibly alters the neurological mechanism of normal erection and detumescence.

The main drugs associated with Priapism are antihypertensives and anticoagulants. Recently there have been reports of priapism due to psychotropic medication. Two cases of priapism due to psychotropic medication with a short followup are being reported.

**CASE REPORTS**

**Case No. 1**

A 43 year old male, watch repairer by profession with history of paranoid schizophrenic episode three years back, was treated with a course of Five Electro Convulsive treatments and chlorpromazine (400 mg/day), which he took for four months and then discontinued on his own.

He presented this time with Priapism which developed 18 hours after he took chlorpromazine 100 mg (Nocte), on his own, for insomnia. He came with two days history of persistent painful erection. He was immediately referred to a Urologist. Surgery was done on the third day, which consisted of removing blood clots and the creation of a right carporo-saphenous and left bulbu-corporal shunt. Post-operative period was uneventful till the sixth day, when patient developed frank psychotic
symptoms with delusions of persecution, abusive and assaultive behaviour which necessitated transfer to psychiatric care. Patient was treated with Haloperidol 10 mg/tid. and was given a course of 5 ECTs. His psychotic symptoms cleared up and was discharged two weeks after admission. His Haemotological and biochemical investigations were within normal limits and except for Priapism, systemic physical examination was normal at the time of admission. During 1 year followup, patient continued to have erectile impotence, though he remained well as regards his Psychiatric status.

Case No. 2

A 22 year old unmarried male patient working as a Clerk in a Private industry, started having sleep disturbance, vague fear of being followed, anxiety of ten days duration. He took sleeping tablets directly from chemist for two days (Diazepam 5 mg HS and Nitrazepam 5 mg HS). After two days as there was no improvement, on the advice of another friend he took trifluoperazine 5 mg. He developed painful erection roughly 6 hours after taking the tablet and was seen by us (after 11 hours of the development of priapism). Patient was referred immediately to Urologist. However, in the transit, he had ejaculated sprouts of frank blood per urethra and there was some relief. He was admitted for observation. He had priapism within two hours of taking Trifluoperazine which lasted for 3 hours and reduced spontaneously following the discharge of sprouts of frank blood. Hematological investigations done at the time were normal. He had two similar episodes during the following 48 hours albeit short lasting and each time resolved spontaneously.

For two days no medication was given. Subsequently on mental state examination, patient was found to be depressed with anxiety but no persecutory ideas. His sleep was disturbed and he had loss of appetite. He was started on chlor Diazepoxide 10 mg 1 tid and Amytryptyline 25 mg 1 tid. His depression and anxiety improved over a 3 week period. There was no further episode of priapism. He on followup at the end of 9 months, continued to be euthymic with no disturbance of biological and social functioning. He has been getting nocturnal erections as usual.

DISCUSSION

In the two cases described above, the first patient developed priapism, following the intake of chlorpromazine and surgery had to be performed to relieve the condition. In the second case, priapism developed after intake of trifluoperazine and there was more or less spontaneous relief with the discharge of blood cloth per urethra.

Priapism as a complication of psychotropic medication has been reported initially with Chlorpromazine (Meiraz and Fischelovitch 1969), but subsequently a wide range of neuroleptics and anti depressants were associated with this rare kind of side effect. Mitchell and Popkin (1982) however could find only 15 cases of priapism where specific drug could be identified. Most frequently the drug has been phenothiazine (thiordazine and chlorpormazine being the commonest). Of the anti-depressant, Trazadone was the only drug reportedly incriminated, 11 cases being reported by Scher et al (1983) and few more cases have since appeared (Benkelliat et al 1986, Lansky and Selzer 1984).

The pathophysiology of Priapism is unclear. Normal erection, is a result of sustained increase in arterial supply to the
tissue and according to Wasmer et al (1981), is possibly achieved by relaxation of valves, allowing increased blood flow with corpora cavernosa. Wagner et al (1982) opine that in addition, the above action is combined with simultaneous regulated partial constriction of ‘Shunt’ arteries, which impede venous drainage. These vascular changes are under autonomic nervous control both sympathetic and para sympathetic. Preponderance of sympathetic over Para sympathetic activation is associated with flaccidity while the reverse results in erection.

Intracavernosal injection of alpha blocker Phenoxybenzamine to induce penile erection in men with erectile impotence which were subsequently resolved by similar injection of alpha stimulant metaraminal suggest that the alpha receptors may be involved (Brindley 1983, 1984 & 1985). Most psychotropic drugs being potent alpha adrenergic receptor blockers could induce priapism by directly inhibiting the sympathetically controlled detumescence, which probably is the result of peripheral action on those receptors lying within or close to cavernosal space.

However, other pharmacological properties of anti-psychotics, the anticholinergic or hypotensive actions have not been substantiated as causes of Priapism. (Meiraz and Fischelovitch 1969). The rarity of Priapism with Psychotropic medications, if alpha adrenergic blockade is the cause - as commented by Kogerogos and de Alwis (1986) may be because of proportion of cases may go undetected, unrecognised, unreported because of its short duration and spontaneous remission or more likely because Priapism might infact be a multifactional disorder.

Treatment of Priapism is immediate surgical intervention. Delayed surgery increases the risk of permanent impotence. As Psychotropic medication is associated with small but definite risk of Priapism, considerable caution is required in prescribing them to patients with known risk factors like blood dyscrasis or previous history of abnormal erections.

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