# Correspondence

EDITED BY LOUISE HOWARD

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## Anti-androgenic agent cyproterone acetate cured a woman of severe sexual obsessions

**Sir:** A previously healthy married woman, then 49 years of age, with two children, sought my advice at my psychiatric practice concerning a history of more than three years of obsessive sexual thoughts which occupied her mind every hour of the day. In her everyday thoughts she imagined herself engaged in a very violent sexual act with a male acquaintance of the family. These obsessive thoughts caused her a lot of pain and she was quite disabled by them. She did not dare to leave the house for fear of meeting the man. She could not go to her job as a teacher at a nursery school and had been on sick-leave for more than two years. She had been subjected to a complete hormonal investigation as an in-patient at a clinic for internal medicine without any pathological findings. She had had psychotherapy twice a week for more than two years without any improvement in her condition.

As sexual thoughts in men can be diminished by anti-androgenic agents (Cooper, 1981) and female sexuality is enhanced by the androgenic hormone testosterone (Sherwin *et al*, 1985), I started treatment with cyproterone acetate 100 mg every morning. After three days of treatment all the symptoms had disappeared and the patient was totally recovered. After a few weeks she returned to work. The treatment was prolonged for two years without any relapse and today, one year after withdrawal of the medication, she is still totally free from the illness.

The nature of this psychiatric disorder is of obvious interest. Though the symptoms were of a mainly sexual nature the patient fulfilled all the criteria stipulated in DSM–IV (American Psychiatric Association, 1994) for obsessive–compulsive disorder (OCD). Thus, this diagnosis seems to be the most likely. This assumption raises the question of whether anti-androgenic treatment might be effective in other cases of OCD, perhaps even in cases not involving any sexual symptoms. Treatment of patients suffering from OCD with anti-androgenic agents has previously been suggested by Casas *et al* (1986) who based their suggestion on a pilot study of four women suffering from OCD who all improved after treatment with cyproterone acetate. No study confirming these results has, to my knowledge, been published. Nevertheless, the possibility that anti-androgenic agents could be effective in at least some forms of OCD cannot be ruled out.

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## Pisa syndrome during treatment with sertindole

**Sir:** The Pisa syndrome or pleurothotonus originally described by Ekbom *et al* (1972) is a rare side-effect of classical neuroleptic medication. We report the case of a patient who developed Pisa syndrome after 10 weeks' treatment with sertindole, a novel antipsychotic agent with high selectivity for the mesolimbic dopaminergic pathway and for dopamine $D_2$, serotonin 5-$HT_2$ and $\alpha_1$ noradrenaline receptors (Zimbroff *et al*, 1997).

Mrs R., an 85-year-old pensioner who had no history of psychiatric disorder, was admitted to our old age psychiatry department with acoustic hallucinations followed by symptoms of anxiety and agitation. The patient had a history of hypertonia and the cranial computed tomography scan revealed a cerebral atrophy with extensive confluent periventricular hypodensities without lacunar lesions. We diagnosed a subcortical arteriosclerotic encephalopathy and started symptomatic treatment with haloperidol 5 mg four times daily for six weeks. Because of extrapyramidal side-effects psychopharmacotherapy was changed to sertindole 4–8 mg four times daily, leading to a complete recovery from psychiatric symptoms and a marked improvement in the neuroleptic-induced Parkinsonism. The clinical course was complicated by pneumonia and a stroke with right occipital and cerebellar infarction. During this event sertindole treatment was not discontinued, but the dose was reduced from 8 to 6 mg at week 6. After 10 weeks of sertindole treatment (five weeks after the stroke) Mrs R. was suddenly found leaning backwards and to the right side. This posture was maintained independently of body position. There were no other new neurological signs. The patient was diagnosed with Pisa syndrome and the sertindole was reduced to 2 mg four times daily. Four days after dose reduction all signs of dystonia had fully resolved.

Our patient met all major features previously described with Pisa syndrome: an axial dystonia with flexion of the trunk; a remarkable indifference to her grossly abnormal posture; current antipsychotic treatment; and the absence of other causes or family history of dystonia. The case is complicated in its interpretation by the patient's stroke that in itself could have produced motor abnormalities. The sequence of events, however, with the onset of Pisa syndrome five weeks after the stroke during continuous rehabilitation, together with the cessation of Pisa syndrome after dose reduction of sertindole, suggests a drug-induced effect. We conclude that Pisa syndrome may occur with sertindole treatment as it has previously been reported with clozapine (Kurtz *et al*, 1993). Even atypical neuroleptics should be used with caution in elderly patients at risk of developing motor side-effects.

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## Olanzapine-induced thrombocytopenia in association with idiopathic thrombocytopenic purpura

**Sir:** Thombocytopenia is one of the common side-effects of pharmacological therapy, but it is rarely induced by psychotropic drugs. We would like to draw attention to a case where the conditions of both an idiopathic and a neuroleptic-induced thrombocytopenia occurred.

A 30-year-old woman had a four-year history of paranoid schizophrenia. She was hospitalised because of a grand-mal seizure and persistent delusions while receiving clozapine. She presented with psychomotor retardation, affective blunting and delusions of reference. Routine examinations were unremarkable, except a platelet count of 137/nl. Medical records on previous hospitalisations elsewhere report low platelet counts of 107/nl and 117/nl prior to receiving medication; and she had a history of petechia. However, 11 years ago platelets had been within normal range. She made a good recovery on benperidol. To meet psychomotor side-effects, we then prescribed olanzapine (20 mg). While checking laboratory values regularly we discovered a decrease in platelets to 10/nl on the 17th day of treatment with olanzapine. This decrease had occurred in three days. The patient was transferred to a general medical ward. Platelets returned to normal after discontinuation of olanzapine and administration of human gamma globulins and prednisolone. The patient was discharged on 300 mg sulpiride and a maintenance dose of prednisolone.

We assumed that olanzapine had worsened a pre-existing idiopathic thrombocytopenic purpura (ITP) on an autoimmune basis. To confirm this diagnosis, we repeatedly attempted to document the presence of autoantibodies directed against the complex formed by the drug binding to the thrombocyte membrane. This is possible in about 40% of all presumed cases of drug-induced thrombocytopenia (Greinacher *et al*, 1994). Since we failed, the attribution of thrombocytopenia to autoimmune drug-dependent destruction was not proved definitively, and relies on the time-dependence of the abnormal blood count on the administration of olanzapine. Thrombocytopenia usually occurs 5–15 days after starting a drug therapy (Handin, 1998).

Previous reports support the hypothesis that psychiatric medication can worsen the condition of a pre-existing ITP (König *et al*, 1995) or induce an immune-mediated thrombocytopenia (Balon *et al*, 1987; Durst *et al*, 1993; Mahmood *et al*, 1996). Therefore, this case raises the issue of a coincidence or a possible interdependence between an idiopathic tendency to thrombocytopenia as in ITP, and drug effects of olanzapine.

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## Salinophagia in anorexia nervosa

**Sir:** We report a case of pathological salt ingestion as a feature of anorexia nervosa. The patient is a single woman in her thirties with a 15-year history of anorexia nervosa (World Health Organization, 1992), of sufficient severity to necessitate in-patient treatment on a specialist unit. While engaged in our standardised treatment programme, combining weight gain with psychotherapy, she admitted to intermittent pathological ingestion of table salt over the preceding two years in the form of up to 20 packets (approximately 80 g) of salt per day, which she would consume with bread or potatoes. Her impulses towards salt ingestion existed in negative reciprocity with her body mass index, and came to light as her weight reached the mean matched population weight. Despite this history her electrolyte levels were normal, with adequate renal compensation of hypernatraemia.

The phenomenology of her behaviour appeared to be a form of deliberate self-harm, ego-syntonic but self-punative in nature. In particular, her salt ingestion lacked salient features of an obsessive–compulsive disorder or pica. Notably, another patient on the unit appeared to adopt similar behaviour in imitation, which reflects the tendency for some symptoms of anorexia nervosa to run in trends.

We addressed her salt ingestion as a form of learned maladaptive behaviour, combining both cognitive–behavioural and psychodynamic techniques, and the patient remains in treatment.

Compulsive eating of unusual substances has been described in a variety of psychiatric disorders, including schizophrenia, learning disability (Jawed *et al*, 1993) and anorexia nervosa (McLoughlin & Hassanyeh, 1990). The latter description linked pagophagia (the compulsive eating of ice) with iron and zinc deficiency. However, our patient's behaviour was not compulsive in nature and, to our knowledge, is the first published description of pathological salt ingestion, or 'salinophagia', as a symptom of anorexia nervosa. Although rare, we feel it should be added to the list of maladaptive behaviours associated with anorexia nervosa and bulimia nervosa. In addition, physicians should consider salinophagia among their differential diagnoses when faced with unexplained compensated or uncompensated hypernatraemia.

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