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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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