Letters to the Editor

Non-epileptic attack disorder, psychoseizures and schizophrenia

Sir,

I read with great interest the recent paper on 'non-epileptic attack disorder' by Binnie.¹ Over the past years I have been interested in patients presenting with acute psychosis and labelled as schizophrenics, when in fact they may be suffering from non-epileptic attack disorders. These patients, at least in my own practice, are often post-pubertal adolescents who frequently have a vague history of a preceding viral illness or head injury. They present with the episodes of depression, paranoia, associated with outbursts of irrational behaviour and aggression, often with auditory perceptual changes such as hyperacusis, hypoacusis or frank auditory hallucinations.

Subtle examinations of these patients even before treatment often demonstrate minimal extrapyramidal dysfunction, which may be enhanced with neuroleptic treatment. Although most patients do not demonstrate any abnormality on routine electroencephalogram studies, they do present with non-epileptiform psychogenic seizures, or psychoseizures,² and recent positron emission tomography scan studies seem to confirm abnormal neuronal outbursts in these patients. One patient who was most refractory to most neuroleptics, responded well when treated with valproic acid.

It therefore seems to me that many so-called schizophrenics are actually suffering from a non-epileptic seizure disorder or psychoseizures, and that the ongoing work by Binnie and his colleagues will hopefully solve some of these questions.

Peter K. Lewin
Hospital for Sick Children,
Toronto,
Ontario,
Canada M5G 1X8.

References

1. Binnie, C.D. Non-epileptic attack disorder. *Postgrad Med J* 1994, 70: 1–4.
2. Lewin, P.K. Schizophrenia research (letter). *Am J Psychiatry* 1993, 150: 1754–1755.

Androgen-producing adenoma masked by obesity

Sir,

Cushing's disease is uncommon in hospital practice while obesity is common. It is therefore important to exclude Cushing's disease and other causes of excess androgen production as a cause of hirsutism in obese individuals.

Recently a patient with simple obesity in whom Cushing's disease was initially excluded but who had an adrenal adenoma, was admitted to our hospital.

The 72 year old woman was admitted with an acute myocardial infarction. She was markedly hirsute with

male pattern hair. She required to shave daily. A review of the case sheet demonstrated that marked hirsutism had originally been noted 11 years previously and during three subsequent admissions to hospital.

Hirsutism was originally noted when she presented with vitreous haemorrhages, was found to be hypertensive, with a blood pressure of 190/100 mmHg and was referred to an endocrine unit.

Clinical features included male distribution of hair over her back, face, breasts and abdomen. Centripetal obesity and purple striae were also noted. A normal diurnal serum cortisol rhythm was present with a concentration of 727 nmol/l at 9.00 a.m. and 84 nmol/l at 12 midnight. An overnight dexamethasone suppression test response was also normal as were her skull and chest X-rays. A glucose tolerance test demonstrated impaired glucose tolerance. A diagnosis of hirsutism, secondary to simple obesity, was made.

On the current admission her serum testosterone concentration was 10.8 nmol/l (reference range <3.5 nmol/l), androstenedione concentration was 50 nmol/l (reference range 2–11 nmol/l) and dehydroepiandrosterone sulphate concentration was raised at 38 μ mol/l (reference range 2–10 μ mol/l). Computerised tomography of the adrenal glands demonstrated a 3 cm solid left adrenal mass. She declined surgery.

This woman illustrates the relatively benign course that may be followed by androgen-producing adenomas, but nonetheless she had suffered psychologically and the excess androgen production may have contributed to the development of her coronary arteriosclerosis.

Paula Chattington
M.J. Diver²

W.D. Neithercut¹

Department of Medicine for the Elderly,
and ¹Department of Chemical Pathology,
Wirral Hospital NHS Trust,
Wirral,

and ²University Department of Clinical Chemistry,
Royal Liverpool University Hospital,
Liverpool, UK.

Tertiary hyperparathyroidism in nutritional osteomalacia

Sir,

Tertiary hyperparathyroidism is a rare complication of severe nutritional osteomalacia.¹ We report here an instance of this complication of long-standing osteomalacia.

A 30 year old single Kuwaiti woman presented with bone pains involving the pelvic girdle and rib cage, and progressive proximal muscle weakness of 2 years' duration. There was no history of diarrhoea, weight loss or anticonvulsant use, but the nutritional history assessed by a 24 hour recall showed an average calcium intake of 230 mg/day, phosphate intake of 400 mg/day, and vitamin D intake of 100 IU/day. From age 14 she had