A case of Olfactory reference syndrome

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Case report

An army soldier in his early thirties was referred to the neurology ward with a history of urinary incontinence for the last 2 months. He complained of incontinence of urine and an unpleasant odour of urine which was noted by others as well. He complained that some of the gestures of his colleagues such as wiping their nose, or covering their faces as a response to his unpleasant body odour. He stated that these thoughts occupied most of his waking time although he acknowledged that his thoughts on the odour were excessive and unreasonable. Detailed questioning revealed he was repeatedly checking his underwear for urinary stains and was taking extra baths and wearing additional undergarments which he frequently changed in order to mask the odour. Clinical evaluation revealed that the preoccupation of the smell of urine was what led to him presenting as incontinence and there was no objective evidence of urinary incontinence. Detailed neurological evaluation excluded spinal cord pathology and a contrast enhanced CT of the brain and EEG was normal.

A detailed history from our patient revealed that what he experienced was not true incontinence but the smell of urine which he perceived and attributed to incontinence which could be a discrete entity (the olfactory reference syndrome) or could be a component of other disorders such as schizophrenia, depression or temporal lobe epilepsy. Clinical assessment comprising of neurological and psychiatric aspects as well as the investigation findings concluded that the patient had olfactory reference syndrome. He was commenced on cognitive behavioural therapy and he showed significant improvement.

Discussion

Olfactory reference syndrome (ORS) is defined as a psychiatric condition characterised by persistent preoccupation about body odour accompanied by shame, embarrassment, significant distress, avoidance behaviour and social isolation. Referential thinking is where patients have delusions of reference, falsely believing that other people perceive the odour. ORS symptoms most often begin when the patients are around their mid 20s but some reports suggest onset during puberty and adolescence, with a male preponderance of 2:1. The individuals are preoccupied with the belief they emit an unpleasant or offensive body odour. Flatulence, faecal or anal odours, general body odours, halitosis and genital odours are the most common but they also perceive other odours such as sweat, armpit odour, sperm, urine and malodorous hands and feet. There is referential thinking and they misinterpret the behaviour of others assuming it is a reaction to how the patient smells. The patient is ashamed, embarrassed and concerned about offending others that they engage in repetitive and safety behaviours intended to check, eliminate or camouflage the odour.

ORS leads to functional impairment where the individual often avoids other people or believe that others avoid them. They are worried and avoid activities, break off engagements, refuse travel and become housebound. The distress and impaired functioning leads to psychiatric hospitalisation, depression and suicidal ideations [1].

This is a chronic illness persisting for years and possibly worsening with time if the patient does not receive appropriate treatment. ORS may be associated with depression, personality disorders, schizophrenia, hypochondriasis, alcohol and drug abuse, obsessive compulsive disorder and body-dysmorphic disorder.

Worldwide there have been only 84 reported cases of ORS [2]. ORS has been described for more than a century but the pioneering report describing 36 patients was in 1971 [3,4].

ORS is not included in the Diagnostic and Statistical Manual of Mental Disorders (DSM IV). For the DSM V it is being proposed to add ORS to an appendix of conditions that need further research in order to have an agreed definition that researchers can use [5].

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Introduction

Although acute onset hypokalaemic paralysis is an uncommon cause of acute weakness, morbidity and mortality may occur due to respiratory failure and cardiac arrhythmias. Therefore it is important for a physician to have an idea regarding the underlying causes and its prompt management. To the best of our knowledge, this is the first case of congenital adrenal hyperplasia (11 beta hydroxylase deficiency) associated with hypokalaemic paralysis to be reported.

Case report

A 17-year old female, diagnosed to have congenital adrenal hyperplasia at birth (11 beta hydroxylase deficiency) came with acute onset weakness of both legs which had progressed over two days and on admission she was almost bed bound. There was no history of diarrhoea or fever. She did not have urinary or bowel incontinence. She had mild weakness of both upper limbs. There were no similar episodes in the past and no family history of a similar problem.

On examination she was afebrile, conscious, without any wasting or fasciculations and with a Glasgow coma scale of 15/15. Power in the upper limbs was grade four and reflexes were diminished. In the lower limbs, power was zero with no sensory impairment and reflexes were absent. Full blood count, serum calcium, serum phosphate and serum magnesium were normal. The only abnormal finding was serum potassium of 1.8 mmol/l which was confirmed by repeated testing. Intravenous potassium replacement was done (60 mmol KCl over 24 hours with normal saline). On second day, serum potassium increased to 2.8 mmol/l and then to 4.3 mmol/l. By second day, her power improved considerably and by day three, power and tone in the limbs were normal.

The diagnosis of hypokalaemic paralysis should be suspected in any patient with sudden onset areflexic pure motor weakness involving one or more limbs without alteration in conscious level or sphincter function and laboratory evidence of hypokalaemia [1]. The cardinal laboratory feature is serum potassium <3.5 mmol/l. Hypokalaemia and paralysis occur either due to an acute shift of potassium into the cells or due to a large deficit of potassium [2]. Symptomatology results from increased ratio between intracellular and extra cellular potassium concentration which modifies membrane polarisation and thereby alters the function of excitable tissues nerve and muscle [1]

Hypokalaemic paralysis represents a heterogenous group of disorders, which leads to a final common pathway presenting as acute weakness and hypokalaemia [3]. Hypokalaemic paralysis can be divided into two major groups [3,4]. The first group include patients with hypokalaemic periodic paralysis which is due to an acute shift of potassium into the cells, e.g. thyrotoxic periodic paralysis, familial periodic paralysis, hypernatraemic hypokalaemic paralysis and sporadic periodic paralysis.

The second group consists of patients presenting with hypokalaemic paralysis which is due to potassium depletion, e.g. mineralocorticoid excess (adrenal adenoma, adrenal hyperplasia, licorice ingestion), renal tubular acidosis, diuretics and magnesium depletion.

References