Gender identity disorder presenting in a girl with Asperger’s disorder and obsessive compulsive disorder

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Introduction

Gender identity refers to an individual’s sense of being male or female. It is typically established by the age of 3 or 4 years (1). Gender identity disorder (GID) is defined as behaviour that signifies cross gender identification. In children and adolescents, GID presents as persistent and intense desire to be the other sex (2). Some associated behaviours include an aversion to the same sex, and a preference for cross gender clothing, toys, peers and role play.

Case report

We report a case of GID in a girl who was followed up as an outpatient from the age of 9 years. Initially the main complaint by the parents was her socially awkward behaviour that had persisted for over a year. This included laughing or talking to herself, repeatedly asking irrelevant questions, displaying odd mannerisms and frequent physical aggression, with disregard to situations and the presence of outsiders. A behaviour that caused much distress to the parents was her habit of suddenly bending down and licking the floor, sometimes even inside a bus. It became evident later that this was triggered by a recurring intrusive and irrational thought that her parents would suddenly die and that she would be left on her own. There were other problems such as not having any friends and rejection of peer group activities. Restlessness in the classroom and lack of cooperation were the complaints from her teacher. Despite all these problems, she was a high achiever in school work and was particularly talented in drawing.

At about 14 years she began insisting on being a male and deeply resented any reference to her as a female. There were instances of physical violence towards persons who tried to reason with her on this issue. After menarche a year later, she rejected all medication, believing that these were given to induce menstrual periods. She demanded immediate referral for sex reassignment surgery and hormonal treatment. There was no cross-dressing but she attempted to hide her breasts by adopting a hunchback posture.

In addition to the diagnosis of GID, persistent irrational thoughts and associated rituals justified a diagnosis of obsessive compulsive disorder (OCD). Her persistent characteristics of poor social interaction and other behaviour patterns supported a diagnosis of Asperger’s disorder (2). Of the drug treatments she received, the response to haloperidol was variable but a significant improvement in OCD was obtained with clomipramine. Drugs did not have any impact on the distress about her assigned sex. Throughout her contact with us, poor compliance in keeping clinic appointments and taking medication was a major challenge to management. Now at 20 years, she still has OCD but less overt features of GID. Currently, she is preoccupied about the GCE Advanced Level examination and has already missed one attempt because of excessive anxiety, doubts and low confidence.

Discussion

We could not find any previous documentation of GID associated with Asperger’s disorder and OCD in children and adolescents. A single case report that resembles our patient to some extent is that of a high functioning autistic female adolescent with transsexualism (3). Our patient also wished to live the life of a male (transsexualism), though her age did not permit such independence. Adults with OCD and GID have been reported (4) but not children or adolescents. In cases with both diagnoses, the primary diagnosis is more likely to be OCD than GID (1,4).

Hence, it is possible that the gender identity problem in this patient represented psychopathology of OCD, though lack of response to medication is not explainable. It is likely that the presence of Asperger’s disorder complicated the clinical picture. These issues have implications for continued drug treatment and the management of possible future demands for sex reassignment. Other case reports on GID have indicated an association between sex chromosomal abnormalities and GID (5) but we have not established the karyotype in our patient.

References


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Staphylococcus aureus with reduced susceptibility to vancomycin

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(Index words: MIC, VISA strains, teicoplanin, disc diffusion antibiotic sensitivity tests)

Introduction
Staphylococcus aureus is a common cause of hospital and community acquired infections (1). It is also the commonest cause of surgical site infection and causes blood stream infections as well (2).

Anti-Staphylococcal penicillins (methicillin, cloxacillin, flucloxacillin) were successfully used in treating penicillin resistant staphylococcal infections. Since the emergence of methicillin resistant Staphylococcus aureus (MRSA), vancomycin has been the mainstay in the treatment for staphylococcal infections. Until recently resistance to vancomycin had not been described. Intermediate level vancomycin resistance, called VISA strains, with minimum inhibitory concentration (MIC) for vancomycin of 8 to 16 mg/l have been reported recently among clinical isolates of MRSA from Japan (3) the USA (4) and France (5). Reduced susceptibility to vancomycin has not been reported previously in Sri Lanka (6). We describe here the first clinical infection due to Staphylococcus aureus with reduced susceptibility to vancomycin.

Case report
A 60-year old man was admitted to a surgical ward at the National Hospital in December 2001, with a history of intermittent claudication of 30 months' duration. He was a known diabetic and hypertensive. He underwent aortotomy and endarterectomy of the occluded aorta and replacement with a Goreflex bifurcation graft a week later.

One week after surgery he developed a surgical site infection (SSI), and was treated with oral amoxicillin-clavulanic acid for two weeks. The only isolate was a coliform organism. Subsequent cultures grew MRSA and treatment with intravenous vancomycin 500 mg 8 hourly was given for one week. A patent graft and a good outflow were observed in the CT scan before discharge from hospital.

In April 2002 he was re-admitted with backache and lower limb paresis. There was no response to two weeks of anti-tuberculosis treatment. The CT scan revealed a para-vertebral abscess with destruction of bone at lumbar 4 - 5 level. CT guided aspirate yielded MRSA, and treatment with intravenous vancomycin and fusidic acid was continued for 4 weeks. MRI scan while on treatment revealed pyogenic osteomyelitis of the vertebrae. After intravenous therapy, oral fusidic acid and oral azithromycin were given for another 2 weeks. Antibiotic sensitivity test on this isolate by disc diffusion method (NCCLS) (7) demonstrated resistance to methicillin, gentamicin, cephalosporin, ciprofloxacin, cotrimoxazole and erythromycin, and sensitivity to vancomycin, teicoplanin and fusidic acid (7). Despite treatment, the patient developed a collection of pus at the surgical site in the right groin which yielded MRSA again.

A CT scan revealed erosion of bones at lumbar vertebral level. CT guided aspirate from the bone also yielded MRSA. This isolate was sensitive to vancomycin (zone diameter 15 mm) and teicoplanin (zone diameter 13 mm) by the NCCLS method (7). The isolate was resistant to fusidic acid, ciprofloxacin, cotrimoxazole, ampicillin-salbactam, erythromycin, azithromycin and tetracyclines. Treatment with intravenous teicoplanin (400 mg daily for 2 days followed by 200 mg daily) was started in June 2002 as we suspected a reduced susceptibility to vancomycin.

He gradually improved with intravenous teicoplanin. After 6 weeks of treatment a persistent discharge at the incision site was observed which yielded MRSA. This isolate showed a similar sensitivity pattern as the previous isolate. Detection of MIC for vancomycin by the E-test revealed an MIC of 6 mg/l, compatible with reduced susceptibility to vancomycin (8). Further investigation with a sinogram excluded an extension to the hip joint cavity and CT scan showed no evidence of deep seated infection. Intravenous teicoplanin was discontinued after 7 weeks.

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