CASE REPORT

Quetiapine-Induced Dysphagia in a Developmentally Disabled Woman with Bipolar Affective Disorder

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Abstract

A case of quetiapine-induced dysphagia has been reported in a 40 years old woman suffering from bipolar affective disorder and moderate to severe learning disability. The dysphagia disappeared following the cessation of quetiapine. The relevant literature on antipsychotic-related dysphagia has been reviewed. It is suggested that the low potency antipsychotic-induced dysphagia may go unnoticed or misdiagnosed especially in a patient with learning disability and clinicians need to be aware of this rare, reversible but potentially serious adverse side effect. The timely recognition can reduce unnecessary investigations and ordeal for the patients and their carers (German J Psychiatry 2011 (2); 14(2): 95-97).

Keywords: Quetiapine, dysphagia, antipsychotics, bipolar affective disorder, learning disability

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Introduction

Dysphagia has been reported to be one of the uncommon adverse side effects of neuroleptic medication especially of atypical antipsychotic drugs due to their low propensity to produce extrapyramidal side effects or other movement disorders (Stewart, 2003; Pierre, 2005). Dysphagia may result from any disturbances in the coordination of the anatomical, physiological, and neurological processes involved with the normal motor sequence of deglutition. Swallowing difficulties are not uncommon in psychiatric patients, especially with comorbidity of mental retardation or neurological impairments, in whom rapid eating and bradykinetic dysphagia are the most common causes (Fioritti et al, 1997). A frequent relationship is seen between patients with brain damaged due to mental retardation and choking (Hollister & Kozek, 1965). Other common risk factors of dysphagia are neurological conditions such as Parkinsonism, dystonia or dyskinesia (Groher & Bukatman, 1986), polypharmacy due to anticholinergic effects by various drugs, including neuroleptics, antidepressants and central anticholinergics (Craig, 1980), institutionalization, poor dentition, and older age (Sokoloff & Pavlakovic, 1997). The incidence of fatalities caused by food choking has been reported to be 0.66 per 100 000 in the general population (Mittleman & Wetti, 1982). It has been opined that both early and late movement disorder (tardive dyskinesia) induced by neuroleptic medication, as studied by manometry on the oesophagus and pharynx, may produce complex dyskinesia of the tongue and oro-pharynx that may lead to asphyxia or dysphagia (Weiden & Harrigan, 1986; Moss & Green, 1982; Yassa & Lal, 1986; Hughes et al, 1994).

The medication-induced dysphagia may go unnoticed or misdiagnosed in a patient who is already mentally challenged with serious consequences such as aspiration pneumonia and asphyxia that may be fatal. We are reporting a case of Quetiapine induced dysphagia in a mentally retarded woman with bipolar affective disorder and hypothesize a possible explanation of its occurrence.

Case Report

Ms X, a Caucasian female in her 40s, a known case of bipolar affective disorder and moderate to severe learning disability, was admitted to the psychiatry ward via the accident and emergency department as an informal patient from the care home where she had lived for more than 15 years. At the time of admission she was on quetiapine 300 mg daily, semi-
sodium valproate 500 mg twice a day and carbimazole 10 mg twice a day. Earlier she had been stable on lithium and olanzapine for a long time. About four months prior to her current admission, she was switched from olanzapine to quetiapine and lithium carbonate to semisodium valproate because of excessive sedation experienced by the patient and some possible concern noted with the thyroid functions. She had started showing deterioration in her mental state to an unmanageable level prior to admission. She exhibited disinhibited behaviour, increased level of energy, hallucinatory experiences, and agitation. The staff at the care home struggled to cope with her. She showed inappropriate emotional reaction in the form of laughing and crying and wandering around in the ward. Her speech was loud and showed pressure of speech, her mood was objectively and subjectively elated and irritable and she lacked insight. Her presentation was complicated somewhat because of her moderate-severe learning disability. A couple of days after her admission, semi-sodium was increased to 750 mg two times a day and the dose of quetiapine was gradually built up to 750 mg per day in two divided dosages. She remained quite unsettled, psychotic, and struggled to cope with her. She showed delusion of persecution, accusing staff members to be mixing poison in her drinks, and would only take fluids in small quantities. A cranial computer tomography head was normal apart from the thickening of the skull bones. Two weeks after her current admission she started presenting symptoms of dysphagia and was seen by a speech and language therapist, who recommended taking food of appropriate consistency under supervision. A modified barium swallow confirmed dysphagia. Quetiapine-induced dysphagia was suspected, as she never had dysphagia before the commencement of quetiapine. Quetiapine was gradually substituted with olanzapine and dysphagia disappeared completely and has not reoccurred since then. She settled down reasonably well in her mental state and was discharged on olanzapine for a long time. About four months prior to her current admission, she was switched from olanzapine to quetiapine and lithium carbonate to semisodium valproate because of excessive sedation experienced by the patient and some possible concern noted with the thyroid functions. She had started showing deterioration in her mental state to an unmanageable level prior to admission. She exhibited disinhibited behaviour, increased level of energy, hallucinatory experiences, and agitation. The staff at the care home struggled to cope with her. She showed inappropriate emotional reaction in the form of laughing and crying and wandering around in the ward. Her speech was loud and showed pressure of speech, her mood was objectively and subjectively elated and irritable and she lacked insight. Her presentation was complicated somewhat because of her moderate-severe learning disability. A couple of days after her admission, semi-sodium was increased to 750 mg two times a day and the dose of quetiapine was gradually built up to 750 mg per day in two divided dosages. She remained quite unsettled, psychotic, and struggled to cope with her. She showed delusion of persecution, accusing staff members to be mixing poison in her drinks, and would only take fluids in small quantities. A cranial computer tomography head was normal apart from the thickening of the skull bones. Two weeks after her current admission she started presenting symptoms of dysphagia and was seen by a speech and language therapist, who recommended taking food of appropriate consistency under supervision. A modified barium swallow confirmed dysphagia. Quetiapine-induced dysphagia was suspected, as she never had dysphagia before the commencement of quetiapine. Quetiapine was gradually substituted with olanzapine and dysphagia disappeared completely and has not reoccurred since then. She settled down reasonably well in her mental state and was discharged on olanzapine 20mg daily and semisodium valporate 1gm twice a day.

Discussion

We believe that this is perhaps the first report of quetiapine-induced dysphagia in a woman of bipolar affective disorder with developmental disability of moderate to severe type. It has been pointed out that the psychotropic medication may depress bulbar centres and result into inhibition of the cough, gag and swallow reflex (Wardell, 1957; Feldman, 1957; Farber, 1957; Reinert & Hermann, 1960). Psychotropic drugs may also produce dopaminergic and cholinergic blockade producing peripheral and central effects on swallowing and potential impairment of oesophageal motility and the gag reflex (Craig, 1980).

The low risk of extrapyramidal side effects and tardive dyskinesia with quetiapine is due to its low affinity for D2 receptors (Emsley et al, 2004), and only few cases of tardive dyskinesia have been reported with the use of quetiapine (Sharma, 2003). Although tardive dyskinesia occurs following a prolonged use of antipsychotics and usually manifests as involuntary movement disorder of the tongue and the orofacial area, it can rarely present as dysphagia (Gregory et al, 1992). Further, neuroleptic medications may produce dry mouth, and some of them can cause movement disorders that may affect the muscles of the tongue and face which are associated with the swallowing. The quetiapine-induced dysphagia in our case could have a similar explanation.

Dysphagia, in a woman of mental retardation with limited verbal abilities to self-reporting, with an atypical antipsychotic medication like quetiapine that has low risk of producing movement disorders, can be easily overlooked unless a high vigil to look for such a rare side effect such as dysphagia is kept in mind. Not only that an early recognition of drug-induced dysphagia can prevent unnecessary investigations and ordeal for the patients and their carers.

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