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Letter to the Editor

Neuroleptic malignant syndrome (NMS) associated with amisulpride and sertraline use: A case report and discussion

1. Introduction

Neuroleptic malignant syndrome (NMS) is a rare, unpredictable adverse reaction associated with antipsychotic use (Lazarus et al., 1989). Second generation antipsychotics (SGAs) were initially assumed to be free from the risk of inducing NMS because of their more favorable pharmacodynamic profile. But several case reports suggests that none of the SGAs are free from the risk of NMS (Belvederi Murri et al., 2015). Antidepressants like venlafaxine, sertraline and mood stabiliser like lithium can also precipitate NMS (Lu et al., 2006; Miranda et al., 2011; Patil et al., 2016). We report a case of Neuroleptic malignant syndrome (NMS) in an elderly female prescribed amisulpride and sertraline.

2. Case description

A 70-year-old female with a history of recurrent depressive disorder and type 2 diabetes mellitus presented to a tertiary care hospital emergency room for an episode of mental status changes. She was taking sertraline 125 mg/day and amisulpride 50 mg/day and was disoriented, hyperpyrexic with tachycardia, 'lead-pipe' rigidity and tremor. She presented with an elevated white blood cell count of 11,100/μL, creatine phosphokinase (CPK) levels 770 U/L, and a temperature of 101.8 °F. Medical workup revealed euvolemic hyponatraemia fitting a diagnosis of Syndrome of Inappropriate ADH (SIADH) secretion and urinary tract infection. A brain MRI scan showed no acute pathology. The patient was started cefaperazone sulbactum for urinary tract infection and admitted to the hospital.

Following admission, the patient's fever resolved and she became talkative but remained confused. Possibility of NMS considered and psychotropics stopped. The patient was treated with hypertonic saline followed by tolvaptan and fluid restriction. The patient improved clinically over a period of one week and discharged. During first follow up after 1 week, she was found to have moderate depressive features. Oral Agomelatine 25 mg/day was started for the same and she became symptomatically better over a period of 2 weeks.

3. Discussion

We believe that the above case adds to the literature base describing NMS in association with both amisulpride and sertraline. In our case the Diagnostic and Statistical Manual of Mental Disorders-5 criteria for NMS were satisfied given the presence of muscle rigidity and elevated temperature accompanied by mental status changes, leukocytosis, mild elevation in CK and autonomic changes (American Psychiatric Association, 2013).

There is no universally accepted set of diagnostic criteria for NMS and there is considerable overlap between the features of NMS and serotonin syndrome, allowing for significant diagnostic blurring (Sachdev, 2005). The recent stability in the patient's medication regime, the urinary tract infection and the prescription of the second-generation antipsychotic amisulpride are at odds with Sternbach's criteria for a diagnosis of serotonin syndrome which include a recent change in a potent serotonergic agent, the absence of a history of substance misuse or infectious (or metabolic) disease and the absence of an antipsychotic agent (Sternbach, 1991). Prominent autonomic instability and the presence of a leukocytosis, slow duration of onset and recovery, the absence of hyperreflexia, clonus or hyperactive bowel sounds lend further support to the diagnosis of NMS (Marlowe and Schirgel, 2006).

NMS has been described with all second-generation antipsychotics such as amisulpride, including even the recently licensed preparation asenapine. A recent review on SGA-induced NMS could identify only seven cases of amisulpride-induced NMS (Belvederi Murri et al., 2015). Unlike our case, most cases occurred in older males, presented with high levels of CK, on a mean dose of 480 mg/day. Among the cases four occurred following an increase in the dose of the drug, were as one led to the death of the patient (Belvederi Murri et al., 2015). NMS has generally been associated with high doses of antipsychotic medication as opposed to the low dose in this case although Lazarus and colleagues did conclude that NMS appeared not to be dose dependent (Lazarus et al., 1989). Sertraline combination could have been an added risk factor (Stevens, 2008). NMS-like syndrome are commonly described with all SSRIs including Sertraline (Miranda et al., 2011). In their recent comparison of NMS induced by first- and second-generation antipsychotics, Trollor and colleagues noted a high rate of concurrent prescription of serotonergic antidepressants in their sample (Trollor et al., 2012). When serotonergic antidepressants are used with antipsychotics, it is proposed that serotonergic inhibition of central dopaminergic activity trough the stimulation of 5-hydroxytryptamine (5HT2A) receptors theoretically increases the potential for NMS already associated with the antipsychotic agents (Odagaki, 2009). There exist reports of NMS occurring in patients prescribed venlafaxine in isolation (Lu et al., 2006).

Old age, physical illness, hyponatraemia, co-prescribed sertraline and non-schizophrenic psychiatric disorder have all been the risk factors for NMS in the present case. Physicians faced with a similar clinical presentation should consider antipsychotic-induced NMS in their differential diagnosis.

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N.A. Uvais*
Department of Psychiatry, Iqraa International Hospital and Research Centre, Calicut, Kerala, India
E-mail address: druvaisna@gmail.com

Prathosh Gangadhar
Department of Endocrinology, Iqraa International Hospital and Research Centre, Calicut, India

V.S. Sreeraj
Department of Psychiatry, National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore, Karnataka, India

P.A. Rafeeque
Department of Medicine, Iqraa International Hospital and Research Centre, Calicut, India

* Corresponding author.