A Case Report of Psychotic Symptoms in Social Anxiety Disorder

Sir,

Social phobias come under the category of phobic anxiety disorders and are centered around a fear of scrutiny by other people, usually leading to avoidance of social situations.[1] Although social anxiety has been found to be the most common comorbidity in people with psychosis,[2] social phobia or anxiety per se is a neurotic disorder in which the patient usually maintains an adequate touch with reality and is rarely associated with psychotic symptoms, beyond the self-referential feelings often observed in social anxiety disorder (SAD). Although there are a few contradictory pieces of evidence of the presence of psychotic symptoms in anxiety disorders,[3,4] it is usually accepted that there are clear lines of demarcation between anxiety disorders and psychosis. The exact prevalence of psychotic symptoms in SAD is not known. Although the presence of psychotic symptoms in SAD would have an immense impact on the severity, management, and prognosis of the disorder, there are only case reports available and the matter is not yet studied in a systematic manner. This case report details how a person with SAD gradually developed disorder-congruent delusions.

CASE REPORT

A 34-year-old married male, graduate in business administration, working as a clerical staff in the Middle East, premorbidly having anxious avoidant personality, presented to the clinical psychology department with a 3 years history of gradual onset of fear of blushing when meeting people and avoiding social interactions with familiar people, especially those in authority, after he started working in a new office. He had less anxiety when meeting unfamiliar people. His social phobia and avoidance increased in the last 2 years, with social interactions getting restricted to only his wife and child as well as colleagues during office work. He started avoiding phone conversations with friends and extended family members, as he believed that they would notice the change in his voice and come to know about his discomfort.

For the last 6 months, he started believing that his blushing during social interactions is offensive to others in the office. Moreover, he was convinced that his “fear of blushing” was contagious and was being transmitted to other people. He reported that he had transmitted his blushing to his supervisor who also started blushing during social encounters. He also believed that his supervisor was offended by the patient’s presence, as he would blush more. Hence, the patient has been avoiding meeting his supervisor. Off late, he felt that more people in the office were finding him offensive, and he was transmitting the blushing to all of them. He wanted to stop going to the office, as he felt responsible for others’ discomfort. He returned to Kerala to get his problem treated.

There is no history suggestive of severe depression, as the patient did not have marked anhedonia, fatigue, or diminished activity. He had past history of low mood, feeling tired, and increase in sleep and appetite that persisted for a few months after he failed in his pre-degree examination 18 years back and it resolved without treatment. Family history of depression in paternal uncle, personal history of restrictive upbringing by parents, and premorbid anxious-avoidant traits were reported. Mental status examination showed low mood, worries about his social anxiety, and firm belief about others finding him offensive as he was transmitting “fear of blushing” to them. He admitted to the possibility that there could be something wrong in his mind, and hence, wanted treatment.

A detailed psycho-diagnostic assessment indicated average intellectual functioning, social anxiety, depressive symptoms, and a high tendency for fantasy. On Beck Depression Inventory scale, he got a score of 26 indicating moderate depressive symptoms; on Beck Anxiety Inventory scale, a score of 16 indicating mild anxiety symptoms; and on Social Interaction Anxiety scale, a score of 43 indicating presence of social anxiety disorder. The diagnosis of SAD was retained as the client did not fit the criteria for severe depression with psychotic symptoms or persistent delusional disorder.

Management involved a combination of paroxetine controlled release tablets and cognitive behavior therapy (CBT) involving cue-controlled relaxation, graded exposure, and cognitive restructuring. At 1 year follow-up, the patient still continued having the delusion that his boss had developed social anxiety and blushing through him, but it appeared to have become encapsulated and to be not interfering in his daily functioning in the office. He continued interacting with his boss through phone whenever possible. He no longer believed that he was transmitting social anxiety to all his
colleagues. His interaction with colleagues was normal, and he was no longer reluctant to go to his office.

**DISCUSSION**

This case is different from a typical case of social phobia in two aspects: first by the presence of a firm belief that his symptoms of social anxiety, especially his fear of blushing, were contagious and his concern over spreading this fear to more and more people, and second, by his conviction that others found his social discomfort offensive.

The false belief in this patient is similar to the offensive subtype of Taijin-Kyofusho (TKS), a condition mentioned under SAD in Diagnostic and Statistical Manual of Mental Disorders 5. The offensive subtype of TKS includes patients with a delusional conviction of offensiveness-persistent and excessive fear of causing offence to others in social circumstances by physical characteristics such as blushing, facial expressions, body odors, or intestinal noises. Cultural and societal norms engendering guilt, shame, and embarrassment are also likely etiological factors. Belonging to a collective society with restricted upbringing, the patient would be more attentive and sensitive to the thoughts, feelings, and behaviors of significant others.

Three explanations have been suggested for the psychotic manifestations in SAD: (1) the individual’s inability to challenge his social fears; (2) stressor and perpetuating role of SAD; and (3) the possibility of a primary thought abnormality leading to psychotic self-reference. Greater paranoid ideation, in a non-clinical sample, was found to be associated with higher levels of social anxiety, avoidance, apprehension about evaluation, self-observation, and low self-esteem.

The transformation of social apprehensions to a delusional level could also be explained using the changes in “social brain” and perception because of the increasingly worsening social isolation. This could explain how the patient’s initial fears of blushing and discomfort progressed into a delusional level with increasing isolation.

The patient improved with treatment focused on SAD - using Selective Serotonin Reuptake Inhibitor and CBT. Antipsychotics have been found to have a lower efficacy in SAD even when delusions are present. This may be because of the hypoactivity of dopaminergic circuits and D2 receptor found in SAD. Antipsychotics could further reduce dopamine action in a system that is already in deficit.

The above case highlights that the commonly assumed demarcation between anxiety disorders and psychosis is questionable and points to the fragility of current diagnostic constructs. Affective and psychotic phenomena often co-occur, and such a co-occurrence predicts a poorer course and outcome, with greater persistence of schizotypal and negative symptoms, more illness behavior, greater likelihood of service use, and more evidence of familial liability for mental illness. New diagnostic subcategories or expanding the social anxiety diagnosis to include psychotic symptoms, as in mood disorders, would have to be considered. This case also suggests that SAD with psychosis could be an entity midway in the dimensional spectrum between SAD and delusional disorder. In addition, the role of social isolation, secondary to social phobia, in the development of psychotic symptoms among patients with social phobia also needs to be further explored.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**Gitanjali Natarajan, Sangeetha P. Louis, Praveen Arathil**

Departments of Clinical Psychology and Psychiatry, Amrita Institute of Medical Sciences, Kochi, Kerala, India

**Address for correspondence:** Dr. Gitanjali Natarajan
Department of Clinical Psychology, Amrita Institute of Medical Sciences, Ponekkara P.O., Kochi - 682 041, Kerala, India.

E-mail: gitanjalin@aims.amrita.edu

**REFERENCES**

1. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva: World Health Organization; 1992. p. 113-4.

2. Michail M, Birchwood M. Social anxiety disorder in first-episode psychosis: Incidence, phenomenology and relationship with paranoia. Br J Psychiatry 2009;195:234-41.
Letters to Editor

3. Veras AB, do-Nascimento JS, Rodrigues RL, Guimarães ACA, Nardi AE. Psychotic symptoms in social anxiety disorder patients: Report of three cases. Int Arch Med 2011;4:12-7.

4. Wigman JT, van Nierop M, Vollebergh WA, Lieb R, Beesdo-Baum K, Wittchen HU, et al. Evidence that psychotic symptoms are prevalent in disorders of anxiety and depression, impacting on illness onset, risk, and severity implications for diagnosis and ultra–high risk research. Schizophr Bull 2012;38:247-57.

5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. (DSM-V). Arlington, VA: American Psychiatric Association; 2013. p. 205.

6. Lim L. Taijin-Kyofu-Sho: A subtype of social anxiety. Open J Psychiatr 2013;3:393-8.

7. Martin JA, Penn DL. Social cognition and subclinical paranoid ideation. Br J Clin Psychol 2001;40:261-6.

8. Hoffman RE. A social deafferentation hypothesis for induction of active schizophrenia. Schizophr Bull 2007;33:1066-70.

9. Van Os JIM, Driessen GER, Gunther N, Delespaul P. Neighbourhood variation in incidence of schizophrenia. Br J Psychiatry 2000;176:243-8.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Access this article online

Website:
www.ijpm.info

DOI:
10.4103/ijpsym.IJPSYM_275_18

How to cite this article: Natarajan G, Louis SP, Arathil P. A case report of psychotic symptoms in social anxiety disorder. Indian J Psychol Med 2019;41:291-3.

© 2019 Indian Psychiatric Society - South Zonal Branch | Published by Wolters Kluwer - Medknow

Catatonia Associated with Hypernatremia

Sir,

Catatonia as a clinical entity is associated with affective disorders, psychotic disorders, and organic conditions. Acute organic catatonia is often associated with metabolic, neurological, and toxic conditions.[1] Although acute organic catatonia has been linked to various causes, there is a lack of literature on the association of acute organic catatonia with hypernatremia. In this report, we present a case of acute organic catatonia associated with hypernatremia, which improved on correction of the hypernatremia.

CASE REPORT

A 67-year-old lady presented with signs and symptoms suggestive of Alzheimer dementia for 5 years and was on treatment with Tab. donepezil 10 mg/day for the last 4 years. Since a year prior to the presentation, she was started on Tab. quetiapine 50 mg at night time for sleep disturbances and behavioral problems. She presented to the emergency outpatient services with acute onset symptoms of 1 month duration, characterized by posturing, mutism, staring, negativism, and urinary and fecal incontinence. History revealed a reduction in oral intake over the last 10–15 days. There was no associated history of fever, sore throat, running nose, or symptoms suggestive of urinary tract infection or skin lesions. In terms of psychiatric symptoms/syndromes, there was no history suggestive of depressive symptoms, sudden worsening of cognitive symptoms, or psychotic symptoms. There was no history of head injury, epilepsy, substance use, hypo- or hyperthyroidism, excessive sweating, or any other medication intake or overdose of medications.

On examination, she exhibited posturing, mutism, waxy flexibility, grasp reflex, and negativism. Further, there was evidence of dehydration and low blood pressure (100/66 mmHg). Physical examination did not reveal any evidence of neck rigidity or gross nutritional deficiencies. Neurological examination was not suggestive of any motor deficit. A provisional diagnosis of organic catatonia was considered. Her Bush Francis Catatonia Rating Scale (BFCRS) score was 21.

On investigation, her hemogram, liver function test, blood glucose levels, X-ray chest PA view, electrocardiogram, and computerized tomography scan of the brain did not reveal any abnormality. However, she was found to have raised serum sodium levels (170 mmol/L), raised serum urea (180 mg/dl), and raised serum creatinine levels (1.7 mg/dl). Other electrolytes were within normal range. Her arterial blood gas analysis also did not reveal any abnormality. Her blood culture reports mentioned “sterile.” There was no evidence of autonomic fluctuation during the initial few hours of assessment.

In terms of management, all medications were stopped. Though lorazepam challenge test was considered,