CASE REPORT

Anorexia Nervosa Features in Schizophrenia: A Starving Mind within a Susceptible Brain

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Abstract

Schizophrenia poses challenges in diagnosis and treatment strategies when the predominant clinical features represent another spectrum of disorder like eating disorders. Nevertheless, symptoms of eating disorder in schizophrenia are not entirely uncommon although never included in the diagnostic criteria. We reported a case of a woman who first presented at seventeen years old with food refusal and underlying psychosis resulting in severe weight loss, but defaulted treatment for almost a decade. In 2009, she presented again with sinus bradycardia and secondary amenorrhoea. In this report, we discussed about the patient’s vulnerability for psychosis and its psychodynamic drive which led to a complex symptoms manifestation. Our aim is to highlight its diagnostic dilemma and suggestions to treatment strategies unique to this presentation.

Keywords: Anorexia Nervosa, Schizophrenia

Introduction

Features of anorexia nervosa (AN) in schizophrenia represent the complexity of schizophrenia. Comorbidity between anorexia nervosa and schizophrenia has been reported1,2,3 and need to be considered. There is a need to differentiate symptoms of AN as part of the spectrum of schizophrenia, those that co-occur as distinct clinical entity, and those that represent overlapping comorbidity. It is also of clinical interest as Kraepelin4 and Bleuler5 once described disorganized and abnormal food intake as being characteristic of schizophrenia.

Subsequent studies found 3 to 12 % of patients with AN may have a final diagnosis of Schizophrenia6,7. It was suggested that AN acts as a defence against psychotic breakdown whereby treating the abnormal eating may uncover the underlying psychosis into full blown psychotic phenomenon8. However, abnormal eating in schizophrenia, as opposed to symptom of AN was described as delusional in nature8.
which corresponds to psychopathology like somatic passivity and made action. While there may be presence of distorted body image and fear of being fat in schizophrenia, the patients rarely have concerns of the food caloric content and other eating preoccupations and obsessions typically seen in AN. While the body image distortion in AN may also amount to delusional proportion, the themes are usually restricted to the primary eating disorder. These characteristic differences and overlapping psychopathology of the two conditions would give good guidance for clinicians in tackling this complex phenomenon.

In this case report, we addressed the diagnostic dilemma and challenges in treatment engagement and strategies. We reported a woman in her late twenties who has schizophrenia since adolescence. One main feature throughout her illness was her food refusal in order to maintain a petite built. She stopped treatment after brief interventions in her teens but reappeared ten years later when her dangerously low weight (32kg) came to an attention of a general practitioner (GP). In this report, we also discussed about the role of atypical antipsychotics in AN by utilizing its weight-gaining propensity.

CASE REPORT

History
A 28-year-old single woman first sought psychiatric treatment at age seventeen for severe weight loss and abnormal behavior. She improved but unfortunately was lost to follow-up until a decade later.

In 2009, she presented with similar complaints of food refusal and abnormal eating habit as she fixated on maintaining low bodyweight. She ate only at midnight once every two days. She chewed slowly a normal amount of food consisting of elaborate meal of many types of dishes. She also exercised excessively for few hours everyday but there was no laxative use or induced vomiting. There was a brief use of over-the-counter diet supplements in the course of the illness. Her reasoning for the fixation on being petite was noted to be superficial and concrete. For example, she explained ‘I eat little because I’m a girl.’

She liked to cook and serve food to others but did these oddly. For example, she served inedible food to her parents and neighbours every hour. Other strange behavior include dressing inappropriately, e.g. wearing underpants in the living room) and soaking in inflated tub. Further assessment revealed underlying abnormal thoughts and auditory hallucinations (second and third person) in the themes of distorted body image and eating. The voices derogatorily mocked her small-framed body. She also had delusions of reference, persecution and thought broadcasting in similar themes. There was absence of affective symptoms.

Physically, she experienced intermittent generalized muscle and bone aches. There was no history of fracture, seizure or blackout. She had secondary amenorrhea since the illness onset but experienced few breakthrough bleeding, the last of which was few months prior to the recent illness.

Over the years, she deteriorated in her social, interpersonal and occupational functioning although her personal care and handling of simple household chores were preserved. She was a graphic design student at the illness onset, but never completed her studies or had permanent employment ever since.

She was never involved in meaningful heterosexual relationship. In the early course
of her illness, she experienced life stressors when her mother became ill with systemic lupus erythematosus (SLE) and later when she discovered that her father had a second marriage, which caused family disharmony.

She is the youngest of three siblings from upper-middle class family. Prominently, her siblings were both high achievers and good looking while the patient was constantly teased due to her small-built and dark appearance. There was family history of psychosis in the late paternal grand-uncle. Premorbidly, she was an obedient child but rather withdrawn and emotionally aloof.

Examinations and Investigations
Mental state examination on admission showed a small-built lady who was soft-spoken with superficial rapport. Her affect was restricted. She had delusion of distorted body image and overvalued ideas on food. Her social judgment and insight were poor. Physical examination showed a thin lady (BMI of 14.4kg/m$^2$) with sinus bradycardia i.e. (50 beats per minute (bpm)) and fully developed secondary sexual characteristics. Biological investigations showed reduced midcycle peak luteinizing hormone (LH) and follicular stimulating hormone (FSH) levels (2.0 IU/L and 6.3 IU/L respectively) and reduced bone mineral density (BMD) consistent with osteoporosis.

Diagnosis and Treatment course
She was diagnosed to have schizophrenia with secondary anorexia nervosa-like symptoms. During the early admissions, she was started on tablet olanzapine. She improved and regained weight but stopped medications after discharge. She relapsed but her family became accustomed to her odd behavior and accepted it. She never behaved aggressively. However, they became concern of her physical health as her weight deteriorated.

This time (2009), admission was warranted again due to severe weight loss. The immediate treatment goals were to restore weight and control the psychosis. She was co-managed by multidisciplinary teams of psychiatry, dietetic, endocrine, orthopedic and gynecology. Re-feeding regime was started with close monitoring of weight and vital signs. She was surprisingly cooperative to treatment and showed no resistance to regulated eating. Olanzapine (titrated up to 20mg daily) was restarted primarily to treat psychosis. Upon discharge six weeks later, her weight was restored to 35 kg (BMI=15.76kg/m$^2$) with stable vital signs (blood pressure=90-100/60-70 mmHg, pulse rate=70-80 bpm) and reduced psychotic symptoms. Despite the improvement, she never gained insight. Nevertheless, we focused on engaging and educating her family. Soon after discharge, monthly depot intramuscular flupenthixol was added as she took medications irregularly. She ate more regularly and less of abnormal behavior was observed. Later, she also managed to secure a part-time job at a boutique. Her current weight was 39.2 kg (BMI=17.7 kg/m$^2$).

Discussion
Distortion of body image is recognized as one of elementary features of schizophrenia$^{10}$. Such cognition may predispose patients with schizophrenia to an entire spectrum of eating disorders from restrictive to disinhibition. A diagnostic dilemma arises from this phenomenology: is schizophrenia and AN co-exist as co-morbidity, or is the eating disorder a part of Schizophrenia spectrum? While the patient fulfilled diagnostic criteria for both schizophrenia and AN$^{11}$, the psychosis predominated the full clinical picture. The psychodynamic drive to starve oneself needs further exploration, but may be distorted by
the neurochemical imbalances producing psychotic processes in schizophrenia.

She is the youngest in a family whose other members constantly teased her physical appearance which disrupted healthy self-image at adolescence phase. This might be the drive behind her eating disorder. Her mother’s illness and perceived father’s betrayal of second marriage introduced chaos in the patient’s life. The inferiority of having imperfect body image and lack of control in life was compensated by gaining control over own body by starving. Genetic vulnerability for psychosis and a fragile personality development with schizoid traits provided immature coping through regression and psychotic manifestation.

Interestingly, eating-related cognitive dimensions were found to be correlated strongly with patients' perceived distress of psychiatric symptoms rather than the severity of overt psychotic symptoms. This implies that cognitive distortions correspond to the patients’ reported distress (caused by the symptoms) rather than the degree of psychiatric manifestation. This finding is observed in this patient. She only revealed her experience of having abnormal thoughts and hallucinations to others (including the treating doctor and family members) much later after establishment of some rapport. This however, was causing her great distress to which she responded by the manifestation of abnormal eating behavior.

In strategizing treatment for the patient, it is important to resolve the diagnostic issue which helped clarifying the treatment pathway. In cases of severe weight loss, medical stabilization and weight restoration took priority. Once stabilized, we decided to primarily treat her psychotic symptoms. In view of the patient's history of poor compliance and insight, her family were engaged and given psychoeducation. Psychotherapy was limited to supportive despite few psychodynamic conflicts identified as the patient was not psychologically-minded.

For pharmacotherapy, atypical antipsychotics like olanzapine can be a “double-edged sword”. Its use has been reported in AN to aid weight gain by increasing appetite and restricting energy use as sleep improves due to sedation. However, it might risk adherence as patients with AN commonly resist weight gain. In this case, although the patient showed no overt resistance to treatment, she had problems with adherence and therefore depot antipsychotic was the choice long-term therapy.

References


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