Coprophagia in a Patient with Neuro-Behçet Disease: A Case Report

Aise Tangılntız1*, Ebru Sahan2, and Ahmet Ozturk3
1Department of Psychiatry, Bezmiâlem Foundation University, Adnan Menderes Boulevard, Turkey
2Department of Psychiatry, Marmara University, Feyzi Çakmak, Turkey
3Department of Psychology, Istanbul Sabahattin Zaim University, Halkalı Merkez, Turkey

Abstract
Psychiatric symptoms in patients with structural brain anomalies may be very hard to manage. A patient with Behçet Disease associated neurological signs was consulted to a consultation-liaison psychiatrist for evaluation and treatment of coprophagia. Little is known about the etiology and treatment of coprophagia. We’ll review what is known about this disorder as well as treatment options.

Keywords: Coprophagia • Behçet disease • Etiology

Introduction
Coprophagia which means smearing or eating feces is a relatively rare disorder associated with neurological and psychiatric disorders. The etymology stems from Greek origins: “copros” meaning feces and “phagein” meaning to eat. It has been largely ignored in the literature. The German term ‘Skatophagie’ (scatophagia) was proposed by a group of Austrian psychiatrists in the 1870s and in modern times this behavior is designated by several neological terms: ‘coprophaghy’, ‘coprophilia’ and considered an unusual variant of pica [1,2]. This strange behavior requires a comprehensive differential diagnosis including both neurologic and psychiatric disorders such as frontal lobe tumor [3] and encephalitis [4] or mental retardation [5,6], obsessive compulsive disorder [2], schizophrenia [7], and dementia [8]. Since these disorders do not share a common pathophysiology, the question then arises of whether there may be common features shared by these disorders that may hint at the emergence of coprophagia in such patients.

The underlying cause need to be explored first and then this should be appropriately treated. In patients having enough cognitive abilities and who are psychologically minded, an exploration of underlying emotions may be fruitful. Behçet's disease is a rare systemic inflammatory disease characterized by oral aphthous ulcers, genital ulcers, ocular lesions and other systemic manifestations. In some cases, the disease affects the central nervous system, called Neuro-Behçet Disease. NBD is an inflammatory perivasculitis [9-15]. The characteristic MRI lesion in parenchymal involvement is an upper brainstem lesion that extends into the thalamus and basal ganglia on one side. Bilateral lesions are less common [16,17]. We present an adult man who has neuro-Behçet's disease, eats and touches his stool.

Case Report
A 32-year-old man with a diagnosis of Behçet's disease for 4 years was brought to psychiatry outpatient clinic after his wife saw him while eating and touching his feces. He was married and had two children. He had graduated from high-school and worked as a technician until 2 years ago when Behçet disease associated apraxia began. When he was admitted to our clinic, his wife reported that he was eating and touching his feces about 5-6 times per day for the last 2 months. In psychiatric examination, he was looking older than his chronological age; He had eye contact and was kind with the interviewer. His verbal communication was very limited; he was mute for long periods. His mood was euthymic, his affect was restricted. His immediate and short-term memory was intact. Attention span was within normal limits. Ability of reasoning was impaired. He had disorganised and regressive behaviour. His thought content was negative for suicidal or homicidal ideations or psychotic symptoms. He had dysarthria, apraxia and ataxic walking. His vital signs, examinations of head, neck, circulation, respiratory and gastrointestinal systems were normal. Blood tests showed no abnormalities. Cranial MRI findings included hyper-intense multiple lesions in bilateral basal ganglions, globus pallidus, caudate nucleus, periventricular areas, fronto-temporoparietal areas and subcortical white matter and brain stem. Atrophy was determined in fourth ventricle, third ventricle and lateral ventricle and also cerebellar foliage was atrophied accordingly to age group. Furthermore, he had a normal abdominal X-ray and endoscopically a normal GI work-up which revealed no fistulas. His medications included only infliximab for Behçet's disease. First, risperidone 1 mg/day treatment was administered and after 3 weeks his

*Address for Correspondence: Tangılntız A, Department of Psychiatry, Bezmiâlem Foundation University, Adnan Menderes Boulevard, Turkey, E-mail: draysetanyildiz@gmail.com

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Figure 1. A normal abdominal X-ray and endoscopically a normal GI work-up which revealed no fistulas.
coprophagic behavior continued. Then, carbamazepine 400 mg/day, amisulpride 800 mg/day, haloperidole 10 mg/day, fluoxetine 40 mg/day were prescribed respectively. Each has been used for at least 2 months and unfortunately, none of them produced any improvement. Then we started on olanzapine 5 mg/day. 1 month later, olanzapine dosage was increased to 10 mg/day. He came to psychiatry outpatient clinic every month on follow-up and 5 months after the initial evaluation, coprophagia mostly subsided.

Discussion

Complications of coprophagia include sialadenitis [8], infestation with intestinal parasites [6,9], as well as other infectious concerns. There have also been reports of airway obstruction and death due to coprophagia [10]. Given the potential for serious complications from coprophagia, a wide range of interventions both pharmacological and behavioral have been documented in literature [11-13]. Pharmacological treatment for coprophagia has no guidelines; recommended classes of medications have been limited to a few first and second-generation antipsychotic agents and mood stabilizers. Nonpharmacological strategies are mostly based on behavioral theories [14]. There is no consistent brain imaging finding in patients with coprophagia. However, in demented patients frontotemporal neurodegeneration is the suspected cause of coprophagia [11]. In our patient, coprophagia started 2 years after being diagnosed with Behçet’s disease. He had poor insight and restricted speech. The underlying reason for coprophagia may be related with frontoparietal lesion in his brain. Even though, haloperidol has been regarded as the most efficient antipsychotic for the coprophagia by Joseph et al., it did not comply with our patient. Many different pharmacologic therapies were implemented, yet only olanzapine was associated with discontinuation of the behaviour.

Conclusion

Given that coprophagia may cause both infectious and emotional complications like disgust in caregivers and health providers, it is important to treat the patient efficiently to prevent being left alone by others, deterioration to more severe complications and even death. There are no prior reports of the use of olanzapine in the treatment of coprophagia and we would like to report that it could be an option for patients with coprophagia.

References
