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The Treatment of Anxiety Disorder and Major Depressive Disorder in a Patient with Burning Mouth Syndrome

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ABSTRACT

Burning mouth syndrome (BMS) is a complex condition that is characterized as a recurrent burning or scalding sensation in the mouth. The cause of BMS is unknown, and there have not been any universally effective treatments. The current report presents the case of a 58-year old man whose onset of burning mouth syndrome triggered moderate major depressive disorder and generalized anxiety disorder. The patient lost approximately 50 pounds over the course of one year and his self-esteem was lowered, however his psychiatric symptoms were magnified following a deep scale dental cleaning. The patient underwent treatment in the clinic for approximately 6 months; his treatment consisted of medication management and psychotherapy. After inadequate medication response, he was eventually recommended for pharmacogenomic testing. The test indicated that the patient was heterozygous for the C677T polymorphism in the MTHFR gene which is associated with reduced folic acid metabolism. Following supplementation of folic acid and B12, as well as treatment with antidepressants, the patient showed improved symptoms of depression and anxiety. It was noted that during his treatment, the patient's burning mouth syndrome was in turn stabilized and he gained back any lost weight. This case report adds to the ongoing literature on effective burning mouth syndrome treatment as well as its psychiatric implications.

Keywords

Burning mouth syndrome, Anxiety disorder, Depression, Folate deficiency, Pharmacogenomic test.

Introduction

Burning mouth syndrome is a condition that is often characterized by pain, burning, tingling, and dryness in the mouth. The discomfort is usually located on the tongue but can also affect the gums, palate, and lips. The symptoms can occur every day and last for months or even years [1]. The triggers could be recent dental procedures, certain medications, or stressful events [2]. The stress and anxiety precipitate a sympathetic response causing dryness of the mouth and aggravating the burning sensation. The syndrome is predominantly common among postmenopausal females.

The etiology of burning mouth syndrome is not fully understood. It is thought to be affected by both nervous system and psychological factors [3]. Studies have shown that patients with burning mouth syndrome possess a deficiency of folate and vitamin B12 [4]. The conditions associated with the syndrome are xerostomia, oral fungal infection, lichen planus, gastroesophageal reflux disease, depression, and anxiety.

Depression is a disorder characterized by a persistent decreased mood that causes significant impairment in daily life. Low levels of serotonin and norepinephrine are linked to the condition [5]. Folate plays an important role in synthesis of these neurotransmitters. A mutation in the MTHFR gene causes a defect in folate metabolism. The mechanism connecting folate deficiency to low serotonin is not understood, it may involve S-adenosylmethionine (SAMe). SAMe is a methyl donor from methionine. Folate is involved in the generation of methionine from homocysteine. Therefore, MTHFR mutation is believed to be associated with depression. Studies have shown that folate supplements enhance the effects of the SSRIs and

SNRIs [6]. The symptoms of burning mouth syndrome can disrupt sleep, cause frustration, interfere with job performance, and affect social life. These symptoms can increase or trigger psychological problems, including depression and anxiety.

We are reporting the case of a patient with burning mouth syndrome who was successfully treated with desvenlafaxine and L-methylfolate supplements. In this report we are focusing on the connection between burning mouth syndrome, depression, anxiety, and folate deficiency.

Case Presentation

Patient XY is a 58 year old caucasian male with no history of substance abuse who presented to the clinic with increased thoughts of anxiety and sadness. The patient noted racing thoughts, panic attacks, feelings of depression, low self-esteem, a loss of interest in things once found interesting, and insomnia. Approximately 5 months prior to his first visit, the patient was diagnosed with the chronic medical condition of burning mouth syndrome. The condition caused the patient to suffer from symptoms such as dry mouth and burning sensations in his tongue and gums. He was soon after diagnosed with benign migratory glossitis, known colloquially as geographic tongue, which consists of harmless erythematous raised patches on the surface of the tongue [7]. The patient stated that many of his psychiatric symptoms arose following his diagnoses and the changes he had to implement to cope with his condition. The patient was in a visible job in the beverage industry and found his symptoms to interfere with his work by causing a loss of confidence. His condition further affected his ability to eat and drink, leading the patient to lose close to 50 pounds over the course of 4 months. He also reported his sleep interrupted by awakening from discomfort, dry mouth, and intermittent gagging. The patient's symptoms exacerbated following a deep cleaning dental procedure, after which he began to experience psychosomatic symptoms of panic attacks. The patient attended neurologists, dentists, and psychiatrists and was prescribed Ativan to help his insomnia. Patient XY was eager to return to his life before his diagnosis.

Upon examination, the patient was overly worried, but cooperative. The Clinically Useful Depression Outcome Scale (CUDOS) questionnaire, a user-friendly measurement scale for the diagnosis of major depressive disorder, was administered [8]. Patient XY had a CUDOS score of 41, indicating moderate depression. Additionally, a Mood Disorder Questionnaire (MDQ) was self-reported to screen for bipolar disorder [9]. Patient XY had a negative MDQ score, indicating an absence of bipolar disorder. The patient was diagnosed with anxiety disorder and major depressive disorder and was prescribed gabapentin (Neurontin). Patient XY did not respond to his dosage of gabapentin, and instead was prescribed alprazolam (Xanax).

At a follow-up appointment 1 month after his initial visit, the patient scored a Patient Health Questionnaire- 9 (PHQ-9) score of 21 out of 27 and a Generalized Anxiety Disorder-7 (GAD-7) score of 19 out of 21, indicating that his severe depression and anxiety persisted. It was further recommended that the patient complete an Assurex Genesight Psychotropic Test to better understand which psychotropic medications may be best suited for the patient based on his individual genetic profile [10].

The pharmacogenomic testing revealed a plethora of new information regarding the patient's Cytochrome P450 enzyme system. The CYP450 enzymes are known to play a part in the processing of medications, and due to genetic traits that cause variation in said enzymes, medications may affect patients differently [14]. Patient XY's profile showed a CYP2C9 pharmacokinetic genotype that is consistent with reduced enzyme activity. Additionally, his profile showed that the patient is heterozygous for the short/long promoter polymorphism of the serotonin transporter gene. Individuals with this polymorphism tend to display a decreased response to selective serotonin reuptake inhibitors [10]. Of the multiple medications tested, desvenlafaxine succinate (Pristiq) resulted as an antidepressant that was potentially compatible with Patient XY's genetic profile. Beyond the scope of psychotropic medications, the genomic test determined that Patient XY was heterozygous for the C77T polymorphism in the MTHFR gene. Methylenetetrahydrofolate reductase (MTHFR) is a key enzyme in the metabolism of folic acid to folate, and a genotype with the C677T polymorphism in the MTHFR gene has been associated with reduced folic acid metabolism and moderately decreased folate levels in the blood [11].

The results of the testing altered the trajectory of Patient XY's treatment. He was not responding well to alprazolam, and was instead prescribed Pristiq. Further, it was recommended that the patient take supplements of L-methylfolate and Vitamin B12. The patient's neurologist, who he was seeing for his BMS, had also recommended the supplementation of folate. Bloodwork was ordered to measure the patient's serum levels of folate and Vitamin B12, and it was seen that the patient's folate and vitamin B12 levels were within normal limits but on the lower end of the scale (Table 1).

	Patient XY's Serum Level	Limits
FOLATE	13.6 ng/mL	5.9-960 ng/mL
VITAMIN B12	244 pg/ml	180-914 pg/mL

Table 1: Patient XY's blood serum levels of folate and vitamin B12 when compared to approximate limits.

For the months following his change in treatment, Patient XY began to show significant improvement. He reported decreased anxiety, improved mood, and a gradual return to his normal life. Following 6 months of treatment, the patient's PHQ-9 score was 2 out of 27 and his GAD-7 score was 1 out of 21, indicating clinical remission. Most notably, he experienced stabilization of his BMS symptoms. Patient XY's burning and dry mouth symptoms were stable towards the end of his treatment, and he was able to eat and drink normally. The patient was sleeping well and was not awoken in the night due to discomfort. From his initial visit to his last visit, the patient gained back 44 pounds that he had lost due to BMS. Remarkably, the stimulus that triggered the patient's

anxiety disorder and major depressive disorder had improved with his psychiatric treatment.

Discussion

In this case, it is noteworthy that Patient XY shows not only depression but also low self-esteem after being diagnosed with burning mouth syndrome. In previous patients, it was stated that burning mouth syndrome increased the levels of anxiety and depression which was measured by GAD-7 score [12]. With Patient XY, the level of anxiety was also measured by the GAD-7, and it gradually decreased with the treatment provided. The important step in treatment of Patient XY was the pharmacogenomic testing. The above test revealed the presence of C677T polymorphism in the MTHFR gene which is responsible for the folate metabolism defect. Another element that has been associated with burning mouth and depression is deficiency in folate. Patient XY had a lab report done and it showed that the patient was on the lower side of the spectrum for folate levels. It was previously noted that folate deficiency could lead to depressed mood and providing L-methylfolate supplements would help with the symptoms of depression and enhance the actions of the antidepressants [6]. Patient XY was recommended to take L-methylfolate supplements and stated that he was feeling a little more stable after the treatment. The patient responded very well to desvenlafaxine (Pristiq), which was listed as a potentially compatible drug with the patient's genetic profile, leading to a remission of depression and anxiety. This treatment also helped lead to a remission of his BMS symptoms.

According to this conceptualization, the case of patient XY showed lower GAD scores after receiving 6 months of treatment. Patient noted that he felt more stable and his anxiety was lowered significantly. Patient XY also reported that folate and Pristiq helped him with his burning mouth syndrome and that he could eat like before.

Many authors hypothesized a correlation between burning mouth syndrome, depression, and anxiety [12]. The case of patient XY seems in line with these hypotheses, showing that the diagnosis of burning mouth syndrome elevated anxiety and depression. As previously mentioned, the sympathetic responses of anxiety include dry mouth, which can aggravate BMS. The frustration of the devastating symptoms of burning mouth syndrome as well as the interference with a patient's personal life can trigger decreased mood. As stated above, burning mouth syndrome, depression, and anxiety could be connected to the folate deficiency.

A potential research route could further explore the presence of the C77T polymorphism of the MTHFR gene in BMS patients. There is currently little known about the genetic components of burning mouth syndrome, other than the correlation between a polymorphism of the dopamine D2 receptor (DRD2) gene and the perception of BMS pain [13]. A deficiency of folate was seen in multiple BMS patients in Bulgaria [4], which brings the question whether or not all of these patients had a similar genetic profile that affected their folic acid metabolism. A future study could conduct pharmacogenomic testing on BMS patients to determine whether or not they have the same C77T polymorphism in their genetic profile. Additionally, this test could indicate whether the patients are compatible with Pristiq treatment, as it is possible that Pristiq played a part in stabilizing Patient XY's symptoms of BMS.

In conclusion, L-methylfolate supplements and Pristiq could be a possible effective treatment of burning mouth syndrome as seen in the case of Patient XY. In order to better understand this connection, larger clinical trials or further research into the subject should be conducted.

References

1. Gurvities GE, Tan A. Burning Mouth Syndrome. World J Gasteroenterol. 2013; 19: 665-672.
2. https://www.mayoclinic.org/diseases- conditions/burning-mouth-syndrome/symptoms-causes/syc-20350911
3. https://www.health.harvard.edu/newsletter_article/burning_mouth_syndrome
4. Krasteva A, Kisselova A, Dineva V, et al. Folic Acid and Vitamin B12 Levels in Bulgarian Patients with Burning Mouth Syndrome. Journal of IMAB. 2013; 19: 422-425.
5. https://www.medicalnewstoday.com/articles/232248#treatment_SSRIs
6. Young S. Folate and depression-a neglected problem. J Psychiatry Neurosci. 2007; 32: 80-82.
7. Khan S, Shah SA, Mujahid TA, et al. Benign Migratory Glossitis. Case Report and Literature Review. Biomedical Journal of Scientific & Technical Research. 2017; 1: 1548-1550.
8. Zimmerman M, Chelminski I, McGlinchey J, et al. A clinically useful depression outcome scale. Compr Psychiatry. 2008; 49: 131-140.
9. Zimmerman M, Galione J. Screening for bipolar disorder with the Mood Disorders Questionnaire. A review. Harv Rev Psychiatry. 2011; 19: 219-228.
10. Health Quality Ontario. Pharmacogenomic testing for psychotropic medication selection. A systematic review of the assurex genesight psychotropic test. Ont Health Technol Assess Ser. 2017; 17: 1-39.
11. Hiraoka M, Kagawa Y. Genetic polymorphisms and folate status. Congenit Anom (Kyoto). 2017; 57: 142-149.
12. Malik R, Goel S, Misra D, et al. Assessment of anxiety and depression in patients with burning mouth syndrome: A clinical trial. J Midlife Health. 2012; 3: 36-39.
13. Kolkka M, Forssell H, Virtanen A, et al. Neurophysiology and genetics of burning mouth syndrome. Eur J Pain. 2019; 23: 1153-1161.

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