Gait ignition failure syndrome may not be heretofore been described.

METHODS: Case Study: A 65-year-old right-handed woman underwent a right frontal parasagittal arteriovenous malformation embolization 25 years prior to presentation. After a fall resulting in a T12 and L1 compression fracture, two kyphoplasties were performed. After the second kyphoplasty, one year prior to presentation, she developed new onset of gait ignition failure, left anterior thigh pain, lower back pain at L3 with radiation to both hips, and bilateral lower extremity weakness. The gait difficulty duration correlates to the duration she is in a seated position. Upon standing, she is unable to move her legs and exhibits basophobia, feeling she may fall due to weakness and she is unable to lift up her left foot to initiate gait, as if it is glued to the floor. She is able to initiate gait after one minute, but has an unsteady scissors-gait for the first few steps. Afterwards, her gait returns to baseline. Anteroflexion was noted to eliminate her back and leg pain.

RESULTS: Gait examination shows inability to initiate gait after standing, feeling as if frozen. However, she demonstrated scissors-gait after 30 seconds for 3-5 steps, which gradually improved to baseline. Her quadriceps femoris reflex was absent on the right, 3+ on the left. Her Achilles reflex was absent on left. MRI indicated spinal stenosis with broad based osteophytes at T9-T12 and bilateral neural foraminal stenosis at L1-S1. Exercise therapy designed for spinal stenosis was initiated, and resulted in elimination of gaitignition failure.

CONCLUSION: Gait ignition failure syndrome may not be necessarily due to frontal or midbrain dysfunction, but can be secondary to lumbosacral impairment. In this patient, dysfunctional arachnoid villi in the lumbosacral nerve roots may have led to transient increases in pressure throughout the neural axis, including the brain, and associated NPH-like symptoms, such as magnetic gait. Seeing that posture affects epidural pressure in lumbar spinal stenosis, with a decrease pressure in response to anteroflexion and reduced pain [Takahashi 1995], one can postulate that this may be a mechanism affecting the patient. Furthermore, since her symptoms are episodic and directly associated with the duration of time she is seated, one may deduce gait ignition failure to be a manifestation of cerebrospinal fluid or intracranial pressure changes influenced by posture. In addition, symptom resolution via exercise therapy strongly suggests that gait apraxia can also be a manifestation of lumbosacral dysfunction. Therefore, those with gait/ignitionfailure syndrome warrant evaluation for lumbar sacral pathology.

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ABSTRACT: Study Objective: Burning mouth syndrome (BMS) is characterized by a burning sensation in the tongue or other oral sites [Grushka 2002]. Vitamin B complex deficiencies have been associated with BMS, including B1 (thiamine) [Lamey 1988]. Replacement with thiamine and other B vitamins was noted to cause relief of BMS in 34 of 150 patients [Lamey 1988]. BMS secondary to vitamin deficiencies have been discussed; however, hemicolectomy and hyperalimentation associated thiamine deficiency inducing chronic BMS has not heretofore been described.

METHODS: Case Study: A 63 year old female presents with a two year history of BMS pain, two weeks following a hemicolectomy from terminal ileum to transverse colon and five days of hyperalimentation. She describes it as a burning pain, 8/10 in severity, localized to both lips, anterior tongue, and middle tongue. It is aggravated with eating and drinking, increasing to 10/10 on the pain scale. Alleviation of pain is seen when ice, Blistex, or lidocaine-mouthwash is used, decreasing the pain to 4/10. Diurnal variation was noted, wherein the pain is exacerbated later in the evening.


CONCLUSION: Although, BMS can be seen with thiamine deficiency [Lamey 1988], it has yet to be described status-post hemicolectomy and hyperalimentation. Thiamine is absorbed systemically in the upper jejunum, as well as in duodenum and ileum in conjunction with folate [Friedamann 1948]. Thiamine deficiency is associated with Wernicke-Korsakoff Syndrome and Wet/Dry Beri-Beri; however, these abnormalities are associated with a significant decrease of serum vitamin B1 [Martin 2004]. Even with near normal levels of thiamine, her BMS pain
may be a prodromal syndrome which may act as a biological marker of dietary vitamin deficiency.

BMS is highly prevalent in postmenopausal women, wherein trigeminal nerve sensitivity may amplify and worsen pain, given a decrease in estrogen and progesterone [Martin 2007], indirectly influencing her BMS pain. Salivary output and composition can alter due to a drop in estrogen and progesterone as well, allowing baseline reduction of proprioceptive input on the tongue. Ergo, acting through Melzack and Wall’s Gate Control Theory of Pain to disinhibit small C-fibers, it may be perceived as burning pain [Melzack 1965]. Given this case, in those who undergo abdominal surgery or hyperalimentation, query regarding BMS symptoms is warranted.

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The Effects of Valbenazine on Tardive Dyskinesia: Subgroup Analyses of 3 Randomized, Double-Blind, Placebo-Controlled Trials
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ABSTRACT: Study Objectives: The approval of valbenazine (INGREZZA; VBZ) for the treatment of tardive dyskinesia (TD) in adults was based on results from double-blind, placebo (PBO)-controlled trials. These studies demonstrated the efficacy of once-daily VBZ based on intent-to-treat analyses. However, because many different types of patients can develop TD, subgroup analyses describing treatment outcomes by various patient factors were also conducted.

METHODS: Data were pooled from three 6-week trials: KINNECT (NCT01688037), KINECT 2 (NCT01733121), KINECT 3 (NCT02274558), with outcomes analyzed by VBZ dose (80 mg, 40 mg) and PBO. Descriptive analyses conducted using the Abnormal Involuntary Movement Scale (AIMS) total score included: mean change from baseline to Week 6; and AIMS response, defined as 50% improvement from baseline to Week 6. Subgroups were defined as follows: age (<55 years, ≥55 years), sex (male, female), psychiatric diagnosis (schizophrenia/schizoaffective disorder, mood disorder), CYP2D6 genotype (poor metabolizer [PM], non-PM), body mass index (BMI) (<18.5, 18.5 to <25, 25 to <30, ≥30 kg/m2), concomitant antipsychotic (yes, no); type of antipsychotic (atypical, typical/both); lifetime history of suicidality (yes, no); concomitant anticholinergic (yes, no); TD duration (<7 years, ≥7 years).

RESULTS: The pooled population included 373 participants (VBZ 80 mg, n = 101; VBZ 40 mg, n = 114; PBO, n = 158). Mean improvements from baseline to Week 6 in AIMS total score were greater overall with VBZ compared to PBO. Within subgroup categories, AIMS score improvement with VBZ 80 mg (recommended dose) was greater in CYP2D6 PMs (n = 17; 80 mg, -6.8; 40 mg, 2.4; PBO, 0.5), participants taking no concomitant antipsychotics (n = 64; 80 mg, -4.9; 40 mg, -3.0; PBO, 0.0), and overweight participants (BMI 25 to <30 kg/m2, n = 115; 80 mg, -4.2; 40 mg, 2.7; PBO, -0.7). Overweight participants also had the highest AIMS response rates at Week 6 (80 mg, 57.7%; 40 mg, 31.6%; PBO, 11.8%), followed by participants taking typical/both antipsychotics (n = 67; 80 mg, 57.1%; 40 mg, 20.0%; PBO, 25.0%), and those taking anticholinergics (n = 126; 80 mg, 52.9%; 40 mg, 22.7%; PBO, 6.3%).

CONCLUSION: These preliminary analyses indicate that TD improvements were generally greater with VBZ than PBO across most subgroups. However, the small sizes of some subgroups may need to be considered when interpreting results. Additional analyses within subgroup categories are ongoing and will be presented at the meeting.

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The Burden of Tardive Dyskinesia Secondary to Antipsychotic Medication Use Among Patients With Mental Disorders
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ABSTRACT: Introduction: Extrapyramidal symptoms (EPS), including tardive dyskinesia (TD), may result...