nocebo effect could be in action, this is unlikely since dizziness was not presented as a potential side effect on initiation of the medication. In addition, methylcobalamin can cause hyperviscosity syndrome, but due to an absence of visual disturbances and altered mental status, it is also improbable. The relatively rapid onset with initiation and resolution upon discontinuation of this medication strongly suggests that it is not a coincidence, rather an origin for the dizziness. Those who are treated with CFLN-NAC should be queried as to new onset dizziness. For those already dizzy, one should consider other treatment options.

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Menstrual Synchrony of Burning Mouth Syndrome

Jasir T. Nayati, CNMT[1]; and Alan R. Hirsch, MD[2]

[1] St. James School Of Medicine, Park Ridge, IL / Anguilla Campus
[2] Smell and Taste Treatment and Research Foundation, Chicago, IL

ABSTRACT: Study Objective: Burning mouth syndrome (BMS) is characterized by oral mucosal burning sensations, with normal clinical and laboratory results. Menstrual synchrony of migraines and epilepsy have been discussed; however, menstrual synchrony of BMS has not heretofore been described.

METHODS: Case Study: A 29 year old right-handed female exhibited intermittent BMS symptoms, one month after suffering a left parietal infarction. She describes the pain as a burningsensation, localized to the bilateral and anterior aspects of her tongue. It lasts for four days, starts three days prior to her menses, and occurs twice a month. She is unable to correlate any patterns or triggers that may cause to exacerbate her BMS. She denies any taste disturbances, hot-flashes, night sweats, and perspiration.

RESULTS: Abnormalities during neurological examination were noted. Cranial nerves (CN) III, IV, and VI showed bilateral lateral first degree end-gaze unsustained nystagmus. CN IX and X showed decreased bilateral gag reflex. A right pronator drift with a right abductor digiti minimi sign was seen in the motor examination. The cerebellar examination was positive for bilateral dysmetria during the Finger-To-Nose examination, and exhibited Holmes rebound phenomena, right more than left. Sensory examination showed decreased light touch in the lower extremities, right more than left. Hoffman reflex was bilaterally positive. Mental status examinations demonstrated poor similarity interpretation and calculation ability. Her neuropsychiatric testing was normal, and included the Go-No-Go and Animal Fluency Testing. MRI of the brain exhibited gliosis/laminar necrosis in the left inferior parietal lobe, and an 8mm descent of cerebellar tonsils below the foramen magnum.

CONCLUSION: The potential mechanism for catamenial BMS is manyfold. Estrogen and progesterone both have nociceptive properties. Premenstrual drop or reduction of estrogen and progesterone may act to disinhibit pain [Vincent 2008], with pain modulation being more effective during the ovulatory phase (high estrogen and low progesterone) [Rezaii 2012]. Depression in the presence of Late Luteal Phase Dysphoric Disorder may function to exacerbate the perception of underlying pain throughout the body, including the mouth and tongue. Decrease in estrogen and progesterone levels may also alter salivary output and composition. This may allow baseline reduction of proprioceptive input on the tongue, thus acting through Melzack and Wall's Gate Control Theory of Pain to disinhibit small C fibers, which is perceived as burning pain [Melzack 1978]. Along with menses, olfactory ability drops, and food preferences are often reported to change [Keller 2013]. A decrease in estrogen and progesterone can also enhance trigeminal nerve sensitivity [Martin 2007], which exacerbates pain. This may indirectly influence or be associated with her BMS. Such observations justifies a trial of hormonal agents for therapy of BMS.

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Gait Ignition Failure Syndrome Secondary to Spinal Stenosis

Jasir T. Nayati, CNMT[1]; Angela Rekhi[2]; and Alan R. Hirsch, MD[3]

[1] St. James School Of Medicine, Park Ridge, IL/ Anguilla Campus
[2] Aureus University School of Medicine, Fairlawn, NJ/ Aruba Campus
[3] Smell and Taste Treatment and Research Foundation, Chicago, IL

ABSTRACT: Case Objective: Gait ignition failure syndrome, where immobility occurs only upon initiation of ambulation and normal gait ensues once entrained, has been reported with frontal lobe and midbrain locomotor region pathology. However, gait ignition failure

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syndrome secondary to lumbosacral spinal stenosis has not 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 L5 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 gaitreturns 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 lumbosacral pathology.

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Burning Mouth Syndrome After Hemicolectomy and Hyperalimentation

Jasmine M. Campbell[1]; Jasir T. Nayati, CNMT[2]; and Alan R. Hirsch, MD[3]

[1] Caribbean Medical University School of Medicine, Des Plaines, IL / Curacao Campus
[2] St. James School Of Medicine, Park Ridge, IL / Anguilla Campus
[3] Smell and Taste Treatment and Research Foundation, Chicago, IL

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.

RESULTS: Abnormalities in neurological examination: Motor Examination: abductor pollicis brevis 4/5 bilaterally. Drift testing with bilateral cerebellar spooning and bilateral abductor digiti minimi signs. Cerebellar Examination: rapid alternating movements are decreased in the left upper extremity. Reflex Examination: Deep Tendon Reflexes: Brachioradialis: 3+ bilaterally. Biceps: 3+ bilaterally. Triceps: 3+ bilaterally. Ankle Jerk: 2+ bilaterally with delayed return. Hoffman reflex: positive bilaterally. Serum Thiamine level: 66 nmol/L (normal 70-180 nmol/L).

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

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