Bruxism: An Interesting Presentation of Bilateral Basal Ganglia Lesions

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A 39 year old Native American female with a one week history of new onset jaw clenching and slow speech...
Patient had gone to an OSH for complaints of right cheek pain/swelling and chronic abdominal pain

CT of the neck revealed soft tissue swelling but no abscess. Incidental note of a density in right frontal lobe surrounded by edema.

Transferred to us for MRI of the head and further management of her known alcoholic liver disease
Past Medical History:

Alcoholic liver disease
Cirrhosis
Ascites
Recent SBP
Coagulopathy
Hepatic encephalopathy
Medications:

Lasix 80 mg po daily
Spironolactone 200 mg po daily
Lactulose 45 ml po tid
Rifaximin 550mg po bid
Pentoxifylline 400 mg po tid
Thiamine, Folate, multivitamin
Lansoprazole 15mg po daily
Propranolol 10 mg po bid
On presentation to our facility, patient complained mainly of *right cheek pain*. She also mentioned a one week history of *slow speech, jaw clenching and difficulty chewing*

Denied *problems with swallowing, gait ataxia, tremors or weakness, sensory disturbances or any other neurological deficits*

Did not know of any family members having the same problem
Family History: Multiple family members with diabetes

Social History: Reports drinking 24 cans of beer daily for the past 20 years. Quit drinking in November 2012 (six months before presentation). Daily cigarettes. No illicit drug use.
Initial physical exam:

VITALS: Temp 98.9, HR 102, RR 18, BP 118/75, SpO2 97% on RA

GENERAL: AAOx4. Speaking slowly, clenching her teeth throughout the entire interview.

HEENT: NC/AT. PERRLA. Sclerae icteric. No obvious signs of intraoral abscess or swelling.

CVS: S1S2 heard. RRR. No m/r/g noted.

LUNGS: CTAB. No wheezes, rales or rhonchi.


EXTREMITIES: Flapping tremor in upper extremities. 2+ pitting edema to the superior thighs bilaterally. Clubbing in upper extremities.
Neurologic exam:

*Mental status:* Fluent speech, but *mildly dysarthric* from teeth grinding. Comprehension intact, follows commands

*Cranial nerves II-XII:* Able to open jaw and mouth volitionally and control her face without difficulties.

*Motor:* No pronator drift. Spontaneous movements in all extremities. Good distal strength, 5/5 bilaterally.

*Cerebellar:* Mild *dysmetria* on finger-to-nose testing. Decreased rapid alternating movements globally. Prosody of speech was mildly irregular and somewhat *scanning.*

*Reflexes* were brisk throughout.
<table>
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Blood EtOH: Negative
Urine toxicology screen: Negative
Blood ammonia: 72 [25-55]
Next morning MRI... T1 weighted

Normal

Our patient

1. Putamen
2. Pallium
3. Caudate nucleus
4. Insula
5. Lateral ventricle
6. Thalamus
T2 weighted
MRI Official Read:

*Focal, well-defined, symmetric areas of increased T1 and T2 in the globus pallidus bilaterally. These are not the typical changes associated with alcoholism, nutritional abnormalities or other metabolic factors. Their exact etiology is uncertain but they might represent evidence of exposure to an extrinsic substance such as carbon monoxide.*

Normal gray-white matter interface, brainstem and thalami
Differentials for bilateral basal ganglia lesions...

Carbon monoxide poisoning
Heavy metal poisoning (lead, cadmium, arsenic, mercury)
Wilson’s Disease
Hepatic encephalopathy
Acute hypoxic/anoxic injury
Hypoglycemia
**Heavy Metal Screen:**
Cadmium 0.3
Arsenic: None detected
Mercury: <1 [0-9]
Lead: 1 [0-25]

Carbon Monoxide level: 4.7

HIV 1,2 NR

Ceruloplasmin 15 [20-51]
24 hr Urinary copper    23 [Normal  < 40]

Slit-Lamp examination: No Keyser Fleischer rings

Liver biopsy: Quantitative analysis reveals a liver copper content of 41 mcg/g dry weight, a finding that is well within the range expected for chronic liver disease of any etiology. As such, the quantitative copper studies do not provide supportive evidence of Wilson disease.
What about the bruxism?
Discussion

Bruxism refers to a nonfunctional clenching of the jaw and grinding of the teeth that can be either diurnal or nocturnal (more common and better studied).

Two theories for etiology: peripheral malocclusion and central motor pathway disturbance (especially dopaminergic pathways).

Role for smoking, alcohol, drugs, diseases, trauma, stress, personality.

Nocturnal bruxism appears to be part of arousal response (sudden change in depth of sleep) and autonomic changes not seen in diurnal bruxism.
Our patient evaluated by oral surgery. No malocclusion or TMJ problems. Recommended septic mouthwashes for superficial ulcers noted on cheek and tongue.
Diurnal bruxism reported in Huntington’s Disease and basal ganglia infarcts. More common in females.

Amphetamines and nicotine facilitate release of dopamine and users of these substances have higher rates of bruxism.

Interestingly, patients with long term neuroleptic use are also at increased risk of developing bruxism.

Parkinson’s disease patients have higher rates of bruxism than controls.
The basal ganglia are the deep cortical structures involved in regulating and coordinating complex movements.

Serve as a relay between the cortex and spinal cord.

The globi pallidi have a high level of oxidative metabolism, and therefore are the most vulnerable to hypoxia.
Decreased GP activity would INCREASE glutamate release from subthalamic nucleus → damaging substantia nigra.
Substantia nigra tends to inhibit the pontine parabrachial area, which controls orolingual and orofacial movements.

Thus damage to the globi pallidi and substantia nigra may disinhibit the PPA and lead to tonic contraction of orolingual muscles.
In our patient, we believe her bruxism was a manifestation of basal ganglia lesions secondary to chronic alcohol use.
References


Chih-Sung Liang, M.D., Ming-Kuen Chou, M.D., Fei-Wen Yang, M.D. “Delayed-onset diurnal bruxism, psychic akinesia and depression after carbon monoxide poisoning: a case report.” Department of Neuropsychiatry, Beitou Armed Forces Hospital, Taipei, Taiwan (R.O.C.)


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