CLINICAL VIGNETTE

Bradyarrhythmia and Syncope Due to Trigeminal Neuralgia Pain Crisis

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Case Vignette

A 56-year-old male with Type 2 diabetes and a seven-year history of trigeminal neuralgia presented to the Emergency Department with increasing episodes of severe left hemi-facial pain and headache. As part of routine emergency department protocol, he was connected to a telemetry monitor. He was found to have intermittent periods of sinus bradycardia with a rate of 30 beats per minute, punctuated by asystolic pauses lasting 3-20 seconds that coincided with bursts of electric pain on the left side of his face. These episodes occurred several times within a thirty-minute period and were usually preceded by diaphoresis, nausea, and vomiting.

The patient had been previously evaluated by a neurologist but had never been monitored on telemetry at the time of a painful episode. Despite being on an outpatient regimen of daily carbamazepine, his headaches were becoming more frequent, often occurring several times in a day. Additionally, his family noted that during particularly severe headaches, the patient would often "pass out" for ten seconds or less.

During hospitalization, the patient had several more episodes of left-sided headache associated with sinus bradycardia. Carbamazepine was discontinued due to concerns that it might be contributing to the arrhythmia. His electrolytes, cardiac enzymes, and chest radiography were normal. Imaging revealed no structural lesions of the heart or nodal disease; the brain, cerebellum, brainstem, and spinal cord were all normal. Various pain regimens were offered, including combinations of oral hydrocodone; intravenous hydromorphone; topical lidocaine cream; and anticonvulsants, such as topiramate and gabapentin, as well as baclofen and indomethacin. On the first two days of his admission, his pain improved moderately with a combination of oral and topical agents but did not resolve completely. He continued to have symptomatic bradyarrhythmias including sinus bradycardia and junctional escape rhythms but no further episodes of asystole. Peripheral nerve block and trigeminal nerve compression were considered by anesthesiology and otolaryngology consultants but deferred given there was no evidence of trigeminal nerve compression on MRI. The patient was continued on several days of oral and topical analgesics and anticonvulsants while on cardiac monitoring. After four days of around-the-clock medical therapy, he reported an improvement in pain control, and the arrhythmias ceased. After several days of monitoring, the patient was discharged with a complex regimen of oral pain medications, topical anesthetics, and anticonvulsants. Several months later, he spontaneously experienced a significant improvement in symptoms and no longer required intensive therapy to control his trigeminal neuralgia. His family also reported no further episodes of syncope.

Discussion

Trigeminal neuralgia (TN) is a well-described facial pain syndrome, characterized by episodes of paroxysmal electric shock-like pain limited to the distribution of one or more divisions of the trigeminal nerve. Prevalence is 0.015% in the general population[1] and is more often found in people over the age of 40, increasing in incidence with advancing age.[2] Classically, symptoms are unilateral, although bilateral involvement can occur in 5% of cases. Due to its varied response to medical therapy, there are many theories about the pathophysiology of the syndrome, the most common of which is trigeminal nerve root compression. As with our patient, despite medical therapy, patients often experience debilitating disease that affects their daily activities, quality of life, and in rare cases, results in excess vagal tone resulting in sinus bradyarrhythmias or asystole.

This is an infrequent phenomenon but has been described as the trigeminocardiac reflex (TCR). TCR is defined as the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnea, or gastric hyper-motility during stimulation of any of the sensory branches of the trigeminal nerve.[3] The anatomy of the TCR reflex arc is thought to be similar to the better known oculocardiac reflex (OCR), whose afferent input is carried by the ophthalmic (V1) division of the trigeminal nerve. Reported cases of TCR typically involve surgical manipulation of tissues adjacent to the course of the trigeminal nerve, most notably during surgical resection of schwannomas, tumor resection at the cerebello-pontine angle, and transsphenoidal resection of pituitary adenomas.[4] To our knowledge, there have been no case reports of spontaneous vagal stimulation resulting in bradycardia or self-limited asystolic pauses in a patient with known trigeminal neuralgia.

Management of cardiovascular events associated with stimulation of the trigeminal nerve parallels management of the oculocardiac reflex. Although the literature describes the antimuscarinic agents, atropine or glycopyrrolate, as being somewhat effective at preventing oculocardiac reflex in the operating room, it is unproven whether vagolytics are useful in preventing intraoperative trigeminocardiac reflex.

In our patient, it would be impractical to consider long-term treatment with anticholinergics. Instead, given the paroxysmal nature of his bradycardia and relationship to episodes of pain, the most practical approach was to control the stimulus, which in his case was trigeminal neuralgia. Although a specific etiology of TN was not identified, we suspect that his longstanding diabetes played some role in the pathophysiology. In some cases, periodic peripheral nerve blocks have been performed with moderate success in patients with a strong oculocardiac reflex. Although our patient's pain improved with medical therapy alone, had his pain and cardiac events persisted a trial of peripheral trigeminal nerve block or even trigeminal nerve decompression would have been considered.

This case illustrates an unusual case of self-limited episodes of severe bradycardia and asystolic pauses as a result of the trigeminocardiac reflex. We describe the anatomy of the trigeminal reflex arc and potential interventions aimed at truncating the sensory input that initiates the reflex. Although uncommon, it is important for practitioners to be aware of and prepare for potentially serious cardiac events in patients who have severe and refractory trigeminal neuralgia.

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