Case Report

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Glossopharyngeal Neuralgia Leading to Sinus Pause: A Rare Entity

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Abstract

Glossopharyngeal neuralgia is in itself a rare entity and often remains undiagnosed. Asystole, convulsions, and syncope are associated with glossopharyngeal neuralgia in many patients described in the literature, and this condition is called vagoglossopharyngeal neuralgia. These reactions occur due to the complex anatomical relationship between the intermedius, vagus, and glossopharyngeal nerves leading to difficulties during neurosurgical assessment. Here we report a case of 66 year old male, known case of glossopharyngeal neuralgia, presenting with seizure followed by syncope and later on diagnosed to have prolonged sinus pause.

Keywords: Glossopharyngeal Neuralgia; Sinus Pause.

Introduction

Glossopharyngeal neuralgia is a rare facial pain syndrome, accounting for 0.2–1.3% of facial pain syndromes. Approximately 10% of patient are misdiagnosed as trigeminal neuralgia because both syndromes are manifested with facial pain. However in case of Glossopharyngeal neuralgia is located unilateral and extends to the ear and throat.

The first description of severe pain in the distribution of the glossopharyngeal nerve is credited to Weisenberg, in 1910, in a patient with cerebellopontine angle tumor. The term glossopharyngeal neuralgia was coined in 1926 to describe this rare condition characterized by paroxysms of excruciating pain located laterally at the back of the tongue, soft palate, throat, and lateral and posterior pharynx, radiating to the ear. Swallowing, coughing, yawning or chewing may trigger pain, which usually lasts from seconds to minutes.

The association between glossopharyngeal neuralgia and syncope is very rare, being identified by brief episodes of bradycardia, asystole, and hypotension. Such an association, with this same pathophysiology, was first described by Riley et al in 1942.

Onset is sudden and is usually characterized by severe, unilateral, paroxysmal pain along the glossopharyngeal nerve course. Syncope in Glossopharyngeal neuralgia related to neuralgic pain is most likely caused by activation of the dorsal motor nucleus of the vagus nerve by abnormally enhanced input from afferent or ischemic lesions of the glossopharyngeal nerve. The reflex arrhythmia could be explained from the fact that afferent nerve impulses from the glossopharyngeal nerve may reach the tractus solitarius of the brainstem and via collateral fibers reach the dorsal motor nucleus of the vagus nerve. One afferent branch of the glossopharyngeal nerve supplies the somatosensorial information to the nucleus ambiguus, while another afferent branch of the glossopharyngeal nerve, the carotid sinus nerve (Hering nerve), conducts impulses from the body of the carotid sinus to the nucleus dorsalis of the vagal nerve. It has been hypothesized that by artificial synapses in the glossopharyngeal nerve the impulses from the somsatosensorial branches stimulate the carotid sinus nerve and thereby the nucleus dorsalis. Activation of this abnormal loop during severe neuralgic pain would be responsible for bradycardia/

asystole, with cerebral hypoperfusion, slowing of electro-encephalographic activity, syncope, and convulsions in proportion to the duration of asystole. Individual differences in the susceptibility of the dorsal motor nucleus to the pain impulse may explain why not all cases are associated with syncope.

Case Report

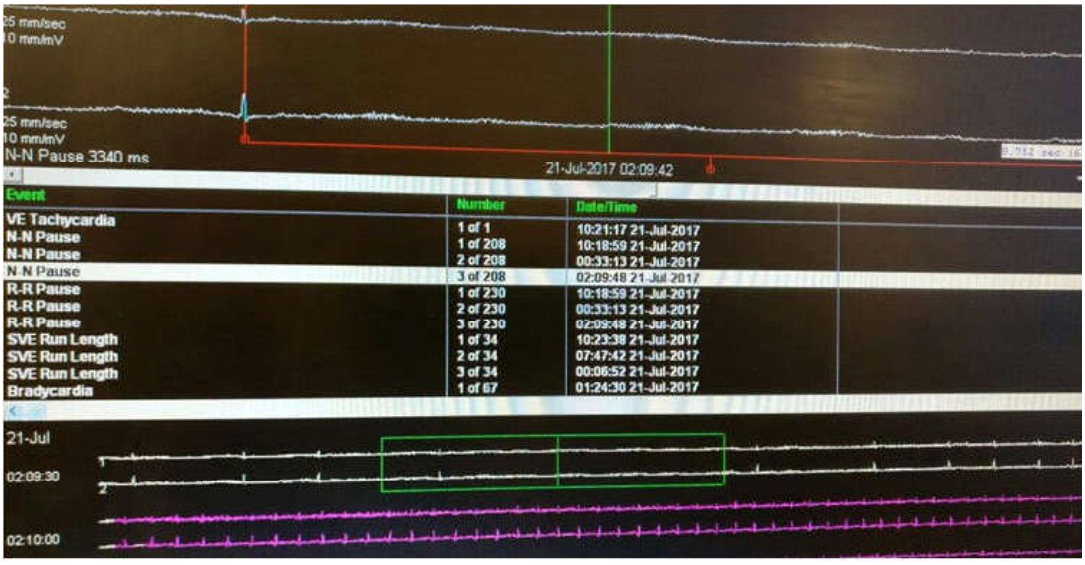
66 year old male, known case of glossopharyngeal neuralgia, recently diagnosed as seizure disorder, on anti epileptics, K/C/O hypertension presented to ER with 1 episode of seizure followed by one episode of vomiting after which the patient developed respiratory distress and eventually drowsy. There was no history of fever, cough, urinary/bowel disturbance, chest pain, palpitations.

On examination, patients airway was compromised and low GCS, in view of which patient was intubated and ventilated in order to protect the airway. Vitals-BP-160/110mm Hg, HR-74/min, SpO_2- 99% on ventilator, RBS-112mg/dl. Systemic examination was unremarkable except decreased air entry on right side. An initial differential diagnosis of ?Breakthrough seizure, ?CVA with aspiration with type-2 respiratory failure was made. Patient was started with anti-epileptics, antibiotics, nebulization, other supportive management and admitted in ICU under neurology department.

MRI brain was suggestive of right parietal small subacute infarct. Patients investigation were suggestive of hypocalcemia and hypomagnesemia and was managed accordingly. Patient responded well to the treatment, was extubated after one day, improved symtomatically and was shifted to HDU after three days.

In HDU, patient had 1 episode of siezure which was managed accordingly. On the same night, patient developed bradycardia and eventually asystole, hypotension and became drowsy. Inj atropine 0.6mg iv stat followed by fluid bolus was given after which patient become responsive and vitals stabilized. Patient was again shifted to ICU, Holter monitoring was planned.

Patient was advised lidocaine spray for his pain and later USG guided glossopharyngeal nerve block was done. Holter monitoring showed intermittent prolonged sinus pause of 13.5 seconds. Following this, patient was taken up for PPI. The patient remained free from syncope after placement of PPI in the hospital as well as on follow up.



Holter Monitoring

Final Interpretation

- Base rhythm is sinus
- Normal Heart rate variability
- Episode of prolonged sinus pauses seen (Maximum 13.5 sec)
- No tachyarrhythmia (AF/PSVT/VT).
- Occasional Supraventricular ectopics.
- Occasional/Frequent VPCs

Discussion

As far as treatment is concerned, the medical literature supports the use of carbamazepine in the management of idiopathic neuralgia. Temporary pacemaker implantation to treat the reflex cardiac syncope until therapeutic levels of carbamazepine are reached was first described by Khero in 1971.

However permanent pacemaker implantation, the available literature is quite controversial but our patient responded well to the permanent pacemaker and remained free from symptoms.

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