Journal of Research in Health Sciences
ISSN: 2228-7809 | https://jrhsjournal.com

Glossopharyngeal Neuralgia secondary to basilar artery compression precipitated by hyperglycemic episodes- A Case Report

[1*]Mohammad Zeya Ansari, [2]Nibedita Mishra, [3]Sarita Kumari, [4]Murtaza Ahmed Khan, [5]Kunal Priyadarshi, [6]Shadab Shafi

[1*,2,3,4,5,6]Tata Main Hospital, Jamshedpur-831011, Jharkhand, India

Abstract:

Glossopharyngeal neuralgia (GN) is a rare cause of severe throat pain and odynophagia. It is rarer than trigeminal neuralgia (TN) and is often associated with vascular conflict. While there are well known non-compressive causes of such neuralgias but impact of hyperglycemia in precipitating or exacerbating its symptoms remains under-explored. Here, we present a case of a-67-year-old male who presented with acute, severe odynophagia and painful chewing. He was diagnosed with GN after excluding other possible causes. Investigations revealed poorly controlled diabetes mellitus (DM) and neuroimaging showed a vascular conflict with the basilar artery abutting the glossopharyngeal nerve. Interestingly, the neuralgic episodes were noted to get precipitated by hyperglycemia. Intensive glycemic control alleviated the neuralgic pain significantly. Hyperglycemia can worsen focal demyelination making it hyperexcitable. This case highlights the role of hyperglycemia in precipitating the GN in a patient with a rare vascular conflict. Although, we need large scale studies to prove such association but the rarity of GN is a speed breaker to such executions.

Key words: Glossopharyngeal neuralgia, hyperglycemia, trigeminal neuralgia, vascular conflict.

Introduction:

Facial and throat pains are commonly encountered by clinicians globally. It may be either neurogenic or non-neurogenic in origin. There are many differentials to such pains which include Eagle syndrome, pharyngo-tonsillar pathology, dental issues, temporomandibular joint disorders, cranial neuralgia, myofascial pain syndrome etc. Out of these disorders, cranial neuralgia is an uncommon entity which can be misdiagnosed leading to delay in accurate treatment. This causes physical, mental and financial misery and burden. Out of all the cranial neuralgias, trigeminal neuralgia is the commonest with an incidence of 4.7 cases per 100,00

person--year. Glossopharyngeal neuralgia (GN) is rarely reported as it accounts for only 0.2-1.3% of all cranial neuralgias. It is mostly seen in patients above 50 with no gender predilection. **[1,2]**

The glossopharyngeal nerve exits the brainstem from the lateral aspect of medulla and exits the skull through jugular foramen. It has five branches (the tympanic, the stylopharyngeus, the pharyngeal, the tonsillar and the lingual) which carry out its sensory and motor functions. **[3]** The low incidence of GN in comparison with trigeminal and facial is probably related to the proximal position of the transition zone and the short central myelin portion in glossopharyngeal nerve. Although GN is misdiagnosed and treated as trigeminal neuralgia, an individual can have concomitant dual neuralgia. **[4]**

It is basically either otalgic or oropharyngeal in type depending upon the area of pain and can be idiopathic (81%) or secondary (19%) to specific causes like vascular or nonvascular compression, multiple sclerosis, medullary tumors etc. The pain can be severe sharp, stabbing, shooting, or electrical shock-like sensations lasting from few seconds to two minutes. **[5]** It can be triggered by coughing, yawning, swallowing, or talking.

GN can have vagal nerve involvement manifesting as bradycardia and syncope which is called vagoglossopharyngeal neuralgia. The main difficulty in GN diagnosis is its rarity and similarity with trigeminal neuralgia that leads to diagnostic dilemma. There are patients who have diabetes and its complications that include anatomical and functional neuronal derangement both centrally and peripherally. Peripheral neuropathies are quite common while cranial neuropathies and neuralgias are uncommon diabetic complications. Hyperglycemia leads to a metabolic cascade which damages the nerves as there are advanced glycation-end products, oxidative stress and protein kinase C activation. **[6]**

Case presentation:

A-67-year-old male with diabetes and hypertension for 5 years and hypothyroidism for 3 years was admitted with complaints of difficulty in chewing and swallowing of food since a week. He felt intense episodic pain which used to become worse while taking food. He was anxious and fearful of taking food for the same reason of pain which he scored between 7 to 10 on the visual analog scale. He denied recent fevers, rashes, colds or cough. There were no nasal stuffiness or earaches. He had no symptoms of giddiness, syncope or seizure. His medications included tablets of metformin 1 gm twice daily, glimepiride 2 mg twice daily, pioglitazone 30

mg once daily, losartan 50 mg twice daily, metoprolol 25 mg twice daily and levothyroxine 75 microgram daily. There was no history of addictions or substance abuse. He behaved noncompliant to treatment of his comorbidities which made him have uncontrolled diabetes mellitus (HbA1c-10.3%).

Clinical examination did not reveal pallor, icterus, cyanosis, pedal edema, cervical or axillary lymphadenopathy. There was no thyromegaly or visible neck swelling. He had systolic blood pressure staying between 120-130 and diastolic between 60-80 mmHg and pulse was mostly between 44 and 60 beats per minute (as expected from his consumption of metoprolol). Systemic examination did not reveal cranial neuropathy or any other deficits. Otorhinolaryngologist did not find any visible oropharyngeal or laryngeal pathology.

His blood investigations revealed poor glycemic control without clinical and biochemical evidence of diabetic ketoacidosis. (table-1)

Investigations	**Reports**	**Normal values**
Haemoglobin	12.7 gm/dl	12-15 gm/dl
Total Leucocyte count	8500/cmm	4000-11000 per cmm
Platelet count	151000	150000-400000 per cmm
Random blood sugar	350	70-140 mg/dl
Glycated haemoglobin (HbA1c)	10.3	4-6%
Serum creatinine	0.96	0.5-1.5 mg/dl
Serum Sodium	137	136-146 mmol/L
Serum Potassium	4.2	3.5-5.5 mmol/L
Serum Calcium	8.56	8.5-10.2 mmol/L
C-reactive protein	0.2	0.08-0.79 mg/dl
TSH	4.5	0.3-6 µIU/mL
Serum protein	7.6	6.6-8.3 gm/dl
Urine for ketone bodies	Negative	Negative
Arterial blood gas analysis pH PCO2 PO2 Bicarbonate	 7.39 37 81 22	 7.35-7.45 35.0-45.0 mmHg 80-100 mmHg 22-26 mmol/L

Table-1: Blood instigation reports

Further evaluations included roentgenogram of skull (Towne's view) and contrast enhanced computed tomogram of neck which excluded elongated styloid process and other neck pathologies.

A neurologist expert opinion was sought who advised magnetic resonance imaging of brain with constructive interference in steady state (CISS) which revealed basilar artery which was abutting the glossopharyngeal nerve and indenting medulla(figure-1). It also showed age related cerebral atrophy with chronic micro vascular ischaemic changes and bilateral frontal, maxillary and ethmoid sinusitis.

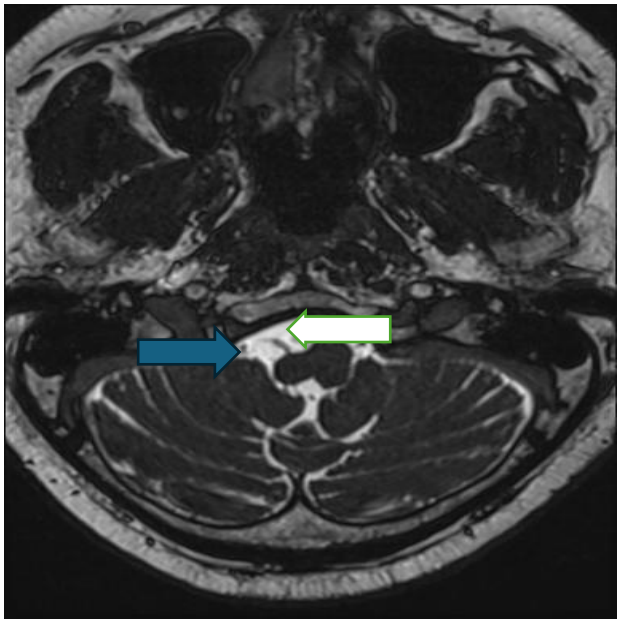

Figure-1: CEMR BRAIN with CISS (Constructive Interference in Steady State) White Arrow-tortuous basilar artery indenting medulla Green Arrow-Glossopharyngeal nerve abutment by basilar artery.

He was started on tablet pregabalin 75 mg and fluoxetine 20 mg once per day which the patient refused to take on second day due to sedation. With intensive insulin therapy random blood sugar came down to below 150 mg/dl. The pain showed significant improvement which was given a score of 3 to 4 on visual analog scale by the patient and made him able to take food without much pain. He was discharged on 7th day with a combination of rapid and intermediate acting (30/70) insulin with tablet metformin 500 mg twice daily. His blood sugar values came to 110-140 mg/dl during discharge. On follow-up visits he did not have recurrence of pain episodes with well controlled blood sugar values. He had to go to his far away house in a village

where he did not take insulin but continued other medications. He came back after 4 months with a similar quality and intensity of throat pain with difficulty in swallowing which he had in the previous episode. On routine investigations random blood sugar was 270 mg/dl with raised glycated haemoglobin (9%). He was counselled for drug compliance and restarted on previous insulin regimen with pregabalin 75 mg per day. He became drowsy after first dose of pregabalin 75 mg which led to refusal on taking further doses. With counselling it was changed to gabapentin 100 mg twice daily. But he developed giddiness with drowsiness after second dose of gabapentin and was apprehensive of any such drug. Therefore, considering abatement in pain severity following glycemic control in the previous hospitalization, a stringent glycemic control process was started. Review after a week showed good response to therapy with normalization of blood sugar level and significant improvement in throat pain.

Discussion:

There are different causes of throat pain like sore throat, tonsillitis, pharyngitis, epiglottitis, retropharyngeal abscess, elongated styloid process and rarely cranial neuralgia. Our patient had painful chewing and odynophagia of acute nature with an intensity severe enough to make him avoid food intake. He was diagnosed with glossopharyngeal neuralgia based on clinical features after excluding other causes of such symptoms. He had poor glycemic control with vascular conflict caused by tortuous basilar artery abutting glossopharyngeal nerve. The common vascular conflicts associated with GN are posterior inferior cerebellar artery (PICA), vertebral artery or anterior inferior cerebellar artery (AICA). There are rare reports of GN caused by vertebra-basilar dolichoectasia which improved after microvascular decompression. **[7,8]**

The vascular conflict is self-explanatory factor of neuralgia but the first and second episodes of GN in our patient were associated with poor glycemic status with abatement in pain after intensive glycemic control. Although, none of the previous reports and studies have mentioned such association between GN and hyperglycemia but there is evidence of trigeminal neuralgia precipitated by poor glycemic control. A higher prevalence of diabetes mellitus in classical trigeminal neuralgia patients was observed in few pilot surveys. **[9]** Hyperglycemia can lower the pain threshold and make trigeminal neuralgia less responsive to carbamazepine. **[10]**

It is a proven fact that diabetes mellitus causes definite anatomical and physiological nerve changes. There are many microenvironmental changes in diabetes in the brain and the nerves

like oxidative stress, inflammation, activation of microglia, excitotoxicity and neuronal damage leading to hyperalgesia. **[11]**

An analytical study on "association of comorbidities in the development of trigeminal neuralgia" showed a significant association with multiple sclerosis , glossopharyngeal neuralgia, hemifacial spasm and complex diabetes (all with p-value < 0.001). **[12]**

Cranial nerves with vascular conflict face repeated pulsatile impacts on it leading to focal demyelination which facilitates a crosstalk with adjacent nerve fibres, a phenomenon known as ephaptic transmission. Acute or chronic hyperglycemia is another factor which may augment or worsen demyelination further in such patients. **[13]**

In a case control study by Esposito K et al it was observed that a sudden increase in plasma pro-nociceptive tumor necrosis factor-alpha (TNFa) during acute hyperglycaemia boosted pain sensitivity. **[14]**

Hence, it is quite possible that hyperglycemia can be an etiology or a precipitant of cranial neuralgias. It is desirable to search for predisposing or precipitating factors of neuralgia even if there is a reached anatomical diagnosis as it is imperative to manage both the primary and the precipitating factors to get optimum and satisfactory response. **[15]**

Therefore, the link between cranial neuralgias and diabetes mellitus is considerable but we need to follow the future trends in such association to endorse it.

Conclusion:

This case emphasizes on searching for the precipitating factors in cranial neuralgias which can be managed conservatively and cost effectively. Although, a tortuous basilar artery was considered the aetiology of glossopharyngeal neuralgia in this case, but robust glycemic control significantly alleviated the neuralgic pain. Therefore, a keen observation and search for the precipitating factors should be practiced in such uncommon neuralgias which can avert the need of pain relievers or modulators and an invasive procedure like vascular decompressive surgery.

Reference:

1-Koopman JS, Dieleman JP, Huygen FJ, de Mos M, Martin CG, Sturkenboom MC. Incidence of facial pain in the general population. PAIN®. 2009 Dec 15;147(1-3):122-7. doi: 10.1016/j.pain.2009.08.023.

2-Singh P. M., Kaur M., Trikha A. An uncommonly common: Glossopharyngeal neuralgia. Annals of Indian Academy of Neurology. 2013;16(1):1–8. doi: 10.4103/0972-2327.107662.

3- Chhabra A, Bajaj G, Wadhwa V, Quadri RS, White J, Myers LL,et al. MR neurographic evaluation of facial and neck pain: normal and abnormal craniospinal nerves below the skull base. Radiographics. 2018 Sep;38(5):1498-513. doi:10.1148/rg.2018170194.

4- Han A, Montgomery C, Zamora A, Winder E, Kaye A, Carroll C, et al. Glossopharyngeal neuralgia: epidemiology, risk factors, pathophysiology, differential diagnosis, and treatment options. Health Psychology Research. 2022 Jun 28;10(5):36042. doi: 10.52965/001c.36042. PMID: 35774913; PMCID: PMC9239365.

5- Blumenfeld A, Nikolskaya G. Glossopharyngeal neuralgia. Current pain and headache reports. 2013 Jul;17(7):343. doi: 10.1007/s11916-013-0343-x. PMID: 23709235.

6- Yagihashi S, Mizukami H, Sugimoto K. Mechanism of diabetic neuropathy: where are we now and where to go?. Journal of diabetes investigation. 2011 Feb;2(1):18-32. doi: 10.1111/j.2040-1124.2010.00070.x. PMID: 24843457; PMCID: PMC4008011.

7-Zhong, J., Dai, Z., Chen, F., Wang, G., Shi, H., Song, W. et al. (2014) Glossopharyngeal Neuralgia Due to Vertebrobasilar Dolichoectasia Coexisting with Fusiform Aneurysm of the Basilar Artery. Case Reports in Clinical Medicine, 3, 545-548. doi: 10.4236/crcm.2014.310118.

8-Chen JY, Takizawa K, Ozasa K, Otani N, Young A, Noma N. Glossopharyngeal neuralgia due to vertebrobasilar dolichoectasia: a case report. Quintessence Int. 2024 May 30;55(5):420-425. doi: 10.3290/j.qi.b5031815. PMID: 38415995.

9- Xu Z, Zhang P, Long L, He H, Zhang J, Sun S. Diabetes mellitus in classical trigeminal neuralgia: a predisposing factor for its development. Clinical neurology and neurosurgery. 2016 Dec 1;151:70-2. doi: 10.1016/j.clineuro.2016.10.015. Epub 2016 Oct 22. PMID: 27816028.

10-Zhang A, Zhang W, Xu H, Guo C, Yuan L, Xu Y, et al. Diabetes mellitus contributes to carbamazepine resistance in patient with trigeminal neuralgia. Neurosurg Rev. 2021 Apr;44(2):1119-1125. doi: 10.1007/s10143-020-01304-4. Epub 2020 Apr 24. PMID: 32333283.

11-Džidić-Krivić A, Fajkić A, Farhat EK, Lekić L, Ejubović A, Vukas SK, et al. Linking metabolic syndrome to neurodegeneration mechanisms and potential treatments. Molecular neurobiology. 2025 Nov;62(11):14344-66. https://doi.org/10.1007/s12035-025-04947-w

12-Tang M, Devarajan A, Huo L, Chung T, Vasan V, Zhang JY, et al. Identifying associated comorbidities in the development of trigeminal neuralgia: A propensity-matched analysis of the National Inpatient Sample. Clinical Neurology and Neurosurgery. 2025 Aug 1;255:108985. DOI: 10.1016/j.clineuro.2025.108985. PMID: 40451122.

13- Gambeta E, Chichorro JG, Zamponi GW. Trigeminal neuralgia: An overview from pathophysiology to pharmacological treatments. Molecular pain. 2020 Jan;16:1744806920901890. doi: 10.1177/1744806920901890. PMID: 31908187; PMCID: PMC6985973.

14- Esposito K, Nappo F, Marfella R, Giugliano G, Giugliano F, Ciotola M,et al. Inflammatory cytokine concentrations are acutely increased by hyperglycemia in humans: role of oxidative stress. Circulation. 2002 Oct 15;106(16):2067-72. . DOI:10.1161/01.cir.0000034509.14906.ae.

15- Gaitour E, Nick ST, Roberts C, Gonzalez-Toledo E, Munjampalli S, Minagar A, et al. Glossopharyngeal neuralgia secondary to vascular compression in a patient with multiple sclerosis: a case report. Journal of Medical Case Reports. 2012 Jul 19;6(1):213. Doi: 10.1186/1752-1947-6-213. PMID: 22812720; PMCID: PMC3459714.