

A Study on the Association Between Refractory Trigeminal Neuralgia and Hypertension in a Nigerian Tertiary Hospital.

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ABSTRACT

Background: Poorly controlled hypertension can result in the refractory pain of trigeminal neuralgia (TN).

Objective: To determine the association between refractory TN and hypertension among patients seen at the Oral Medicine Clinic, University of Benin Teaching Hospital.

Methods: A 1-year prospective study in which patients diagnosed with refractory TN with poorly controlled blood pressure (BP) were managed with the cardiologist and followed up for 4 weeks, to determine the effect of BP control on the pain of TN. On each review, patient's BP was documented, along with their pain scores using the Numerical rating scale (NRS). These assessments were done at a 2-week and 4-week intervals following therapeutic intervention for the BP, and the values compared.

Results: Of the 25 cases of TN seen during the study period, 12 were diagnosed with refractory TN secondary to uncontrolled hypertension. The cases consisted of 6 (50%) males and 6 (50%) females. The mean age of the patients was 54 ± 9.1 years. The refractory pain was commoner on the left side ($n=7$, 58.3%) of the face; right side ($n=5$, 41.7%), with the mandibular division of the trigeminal nerve ($n=9$, 75%) more affected than the maxillary division ($n=3$, 25%). Upon institution of appropriate BP control, there was reduction in the BP. The median systolic BP dropped from was 161.5mmHg (16.75) to 136.0mmHg (20.5); the median diastolic BP dropped from 100.0mmHg (13.5) to 81.5mmHg (3.75). There was also a significant reduction in the NRS-scores from 8 (2.0) to 3.5 (1.75).

Conclusion: In conclusion hypertension should be taken into consideration as a triggering factor for TN, and also in the development of refractory pain in patients with TN associated with hypertension. As a result, regular blood pressure check and control is necessary for TN cases associated with hypertension.

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INTRODUCTION

Trigeminal neuralgia (TN) has been described by the International Association for the Study of Pain as "a unilateral disorder characterized by brief electric-shock like pains, abrupt in onset and termination limited to the distribution of one or more divisions of the trigeminal nerve (Truini, 2015). The pain is limited to the distribution of one or more divisions of the trigeminal nerve" (Truini, 2015) and can be triggered by activities like chewing, smiling, talking, shaving, tooth brushing and can occur spontaneously (Oyetola *et al*, 2018).

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Conventional first line therapy includes carbamazepine and oxcarbazepine (Cruccu *et al.*, 2008) with or without alternatives like baclofen, lamotrigine, pregabalin, gabapentin, pimozide, phenytoin, amitriptyline (Cruccu *et al.*, 2008; Pilitsis and Khazen, 2020). Even with these medications there may be intractable cases where patients do not achieve adequate pain relief.

These cases are termed "Refractory trigeminal neuralgia" (Crucchi *et al.*, 2008; Crucchi and Truini, 2013). It often results in patient dissatisfaction, depression, polypharmacy, multiple specialty referrals, frequent consultations and even frustration of the physician (Moran and Neligan, 2009). While alternative treatment modalities like surgical interventions exist for refractory pain of TN (Crucchi and Truini, 2013), it is of utmost importance to determine the possible cause(s) of the refractory pain before alteration of medication or use of alternative treatment modalities. A study by Abah *et al.* (Abah *et al.*, 2015) reported some causes of treatment failure to include drug tolerance, drug-drug interaction with Vitamin B, and fake drugs. Concurrent intake of carbamazepine with drugs like phenytoin, phenobarbital can induce the clearance of carbamazepine (Lynch and Price, 2007). Treatment failure can also be due to dose reduction to an insufficient level or treatment interruption, as a result of undesired side effects (Maarbjerg *et al.*, 2017). Most cases of TN are classical where TN is usually caused by vascular compression of the trigeminal nerve root at the brainstem by a tortuous vessel (Sahin *et al.*, 2017). Based on the theory of increased arterial tortuosity and pulse pressure, arterial hypertension has been stated as a risk factor for development of TN (Sahin *et al.*, 2017; Uniyal *et al.*, 2017). The underlying mechanism suggested is that hypertension [systolic blood pressure (SBP) values of 130mmHg or more and/or diastolic blood pressure (DBP) of more than 80mmHg] (Kimberly, 2025) can lead to vascular loops compressing the trigeminal nerve resulting in demyelination of the nerve root entry zone, leading to ectopic impulse generation, ephaptic transmission of impulses, with the resultant sharp, electric-like pain associated with TN (Pan *et al.*, 2011; Sahin *et al.*, 2017). Hypertension also accelerates atherosclerotic changes, causing the formation of ectatic vessels and resulting in the development of vascular compression (Uluduz *et al.*, 2007). Episodes of unstable BP can also result in acute neuralgia attacks in hypertensive patients with TN (Sahin *et al.*, 2017). Cranial MRI is an important diagnostic tool for identifying vascular lesions compressing trigeminal nerve or presence of other underlying aetiology (Sahin *et al.*, 2017). Few studies have suggested a positive association between the pain of TN and hypertension. This area, however, needs to be explored further for validation (Arnet *et al.*, 2000). Little information exists in literatures regarding the pain of TN triggered by hypertension and improved with the regulation of hypertension, This center has had several cases of refractory TN amongst patients being managed for TN. After ruling out other possible causes of the refractory pain, a common denominator found in the patients was poorly

controlled hypertension. This study thus aims to highlight uncontrolled hypertension as risk factor for development of refractory TN and the importance of blood pressure control in achieving favourable treatment outcome.

MATERIALS AND METHODS

This was a 1-year prospective study between January 2024 and December 2024 in which patients diagnosed with refractory TN secondary to poor BP control were managed with the cardiologist, and followed up weekly for 4 weeks; to determine the effect of proper BP control on the pain of TN in the Oral medicine clinic of the University of Benin Teaching Hospital. Demographic data, information concerning history, and treatment outcomes were obtained from patients' medical records within 4 weeks of treatment. Following therapeutic intervention for the BP, patients were reviewed at a 2-week and 4-week intervals. On each review patient's BP was taken and documented, along with their pain scores using the Numerical rating scale (NRS) and the values compared. Data obtained from the study were analyzed using the IBM Statistical Package for Social Science (SPSS) version 26.0. Statistical significance levels of the treatment outcome were assessed through Chi-square distribution. $P \leq 0.05$ was considered significant.

RESULTS

There were 25 cases of trigeminal neuralgia seen in the Oral medicine clinic of the University of Benin Teaching Hospital between January 2024-December 2024. Of the populace, a total of 12 patients (6 males and 6 females) reported unremitting pain even with their medications. The age ranged from 35 to 68 with an average age of 54 ± 9.1 years. The left side of the face was observed to be mostly affected with the mandibular division of the trigeminal nerve being more implicated (Table 1).

At To, the median NRS score was 8 (2.0). It decreased to 5.5 (1) at T1 and 3.5 (1.75) at T2. The median systolic BP was 161.5mmHg (16.75) at To. This reduced to 148.0mmHg (11.5) and 136.0mmHg (20.5) at T1 and T2 respectively. At To the median diastolic BP was 100.0mmHg (13.5). A reduction to 86.5mmHg (7.75) and 81.5mmHg (3.75) at T1 and T2 respectively was observed (Table 2).

A cross-tabulation of the median NRS pain scores, systolic and blood pressure values across the review periods observed that the reduction in the systolic blood pressure was not statistically significant ($p > 1.00$). A similar trend was observed in the diastolic blood pressure ($p > 0.175$). The reduction in the NRS pain scores across the review periods was also not statistically significant ($p > 0.534$).

Table 1

Characteristic	N = 12 ¹
Age (years)	
Mean (SD)	54± 9.12
Gender	
Male	6 (50%)
Female	6 (50%)
Site	
Right	5 (41.7%)
Left	7 (58.3%)
Trigeminal nerve division	
Maxillary	3 (25%)
Mandibular	9 (75%)

Table 2

Characteristics	Median (IQR)
To	
NRS	8.0(2)
Systolic	161.5(16.75)
Diastolic	100.0(13.5)
T1	
NRS	5.5(1)
Systolic	148.0(11.5)
Diastolic	86.5(7.75)
T2	
NRS	3.5(1.75)
Systolic	136.0(20.5)
Diastolic	81.5(3.75)

Table 3:

	To (median)	P value
T1 (median)		
NRS (5.5)	NRS (8.0)	>0.625
Systolic (148.0)	Systolic (161.5)	>1.00
Diastolic (86.5)	Diastolic (100)	>1.00
T2 (median)		
NRS (3.5)	NRS (8.0)	>0.534
Systolic (136.0)	Systolic (161.5)	>1.00
Diastolic (81.5)	Diastolic (100)	>0.175

DISCUSSION

Trigeminal neuralgia (TN) has been referred to as "suicide disease" as it is one of the most painful conditions known to man (Sean, 2022). Having patients experience unremitting pain of this magnitude is definitely most worrisome to the clinicians and unbearable for the patients. The mean age of patients with refractory TN in this study was 54± 9.12. This aligns with reports in literature where TN had been seen more in patients above 50years (Omoriegie and Okoh, 2015; Oyetola *et al.*, 2018). Most people with hypertension are over the age of 50, so in line with the theory that hypertension is a possible triggering factor for TN attacks, it is not surprising that hypertension and TN may appear together at the same time periods.

TN has been observed to be predominant in females (Abal *et al.*, 2015; De Toledo *et al.*, 2016). A male predominance has also been reported (Omoriegie and Okoh, 2015; Oyetola *et al.*, 2018). This study observed that refractory TN had equal gender predilection indicating that gender may not be risk factor for development of refractory TN. The most affected side in this study was the left side of the face. This aligns with a report by Oyetola *et al.* (Oyetola *et al.*, 2018) where TN affected the left side of the face more. Omoriegie and Okoh (Omoriegie and Okoh, 2015) reported a slightly higher occurrence on the right side. The refractory pain was mostly associated with the mandibular division of the trigeminal nerve. This aligns

with an earlier report in the study center (Omoriegie and Okoh, 2015).

In this study, the TN patients all had pre-existing hypertension, were compliant with their use of carbamazepine with adequate control of the TN pain. The refractory neuralgia attacks were observed to be precipitated during poor BP control periods. These facts gives credence to the theory that hypertension is a risk factor for the clinical signs of TN (Sahin *et al.*, 2017; Uniyal *et al.*, 2017) Institution of ardent BP control by the cardiologist normalized the BP and relieved the refractory pain. This aligns with a report of Amlodipine-responsive TN by Uniyal *et al.* (Uniyal *et al.*, 2017) and hypertension-induced TN by Sahin *et al.* (Sahin *et al.*, 2017). Interestingly, upon follow-up of these patients, occasions of non-compliance with their BP control brought back their pain. Cranial MRI is an important diagnostic tool for identifying vascular lesions compressing trigeminal nerve. Though this imaging modality was not available for this study, the patients' positive response to antihypertensive medication justified the diagnosis of RTN secondary to poor BP control.

CONCLUSION

In conclusion proper BP monitoring is necessary for all TN cases, especially TN associated with hypertension as pain of TN can be exacerbated by elevated BP. While MRI is important for diagnostic accuracy, the efficacy of medical therapy for TN can be highly enhanced with proper control of the BP in all TN cases.

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