IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 3 Ser. 7 (March. 2019), PP 66-70
www.iosrjournals.org

The Profile of Occipital Neuralgia Patients Regarding Past/Current Use of Medication.

Omar F. Molina[1] Bruno R Simião[2] Marcus S. Peixoto[3] Zeila Coelho Santos[4]
Jamil Elias Dib[5] Daniella Mateus dos Santos[6] Fausto F. Junior[7]

[1]*Department of Orofacial Pain, University of Gurupi, Gurupi-TO, Brazil*
[2]*Department of Prosthodontics, University of Gurupi, Gurupi-TO, Brazil*
[3]*Department f Orthodontics, University of Gurupi, Gurupi-TO, Brazil*
[4]*Department of Orthodontics, University of Gurupi, Gurupi-TO, Brazil*
[5]*Department of Oral Surgery, University of Gurupi, Gurupi-TO, Brazil*
[6]*Private Practice in Orthodontics Gurupi-TO, Brazil*
[7]*Department of Pedodontics, University of Gurupi, Gurupi-TO, Brazil.*
Corresponding Author: Omar Franklin Molina.

Abstract
Introduction: *Occipital neuralgia is a very severe headache disorder which usually occurs concomitantly with temporomandibular disorders and facial pain.*
Aim: *Evaluate medication use: analgesics, muscle relaxants, anti-anxiety and antidepressant drugs in a group of subjects presenting with ON and craniomandibular disorders.*
Methods: *History of chief complaint, clinical examination, evaluation of past/current use o medication, palpation of joint and muscles, assessment of signs and symptoms of craniomandibular disorders and occipital neuralgia in 105 subjects with occipital neuralgia and craniomandibular disorders, 50 individuals with tension-type headache and 30 subjects with no craniomandibular disorders.*
Outcome: *The frequencies of analgesic use were about 95,2%, 76% and 43,4% in the subgroups with Occipital neuralgia, tension-type headache and no craniomandibular disorders, respectively (Chi-squared for independence p<0,0001, for trends p<0,0001): Occipital neuralgia versus tension-type headache (p<0,0003), Occipital neuralgia versus No CMDs (p<0,0001); tension-type headache versus No CMDs (p<0.003). Different analgesic types were used in different subgroups: Occipital neuralgia (mean 3,01), Tension-type headache (mean 2,3), No craniomandibular disorders (mean=1,5). Kruskal-Wallis statistics p<0,0001: Occipital neuralgia subgroup versus No Craniomandibular disorders subgroup p<0,001. Use of anti-anxiety drugs decreased from the Occipital Neuralgia subgroup (23,8%) to the Tension-type headache subgroup (10%) and to the No CMDs subgroup (3,3%). Chi-Squared for independence (p<0,009), for trends (p<0.003). Fisher´s exact test: Occipital neuralgia versus Tension-type headache subgroup (p<0,05); Occipital neuralgia versus No CMDs subgroup (p<0,009); Tension-type headache versus No CMDs subgroup (p=0,40). Antidepressants use decreased from the Occipital neuralgia (39%) to the Tension-type headache (32%) and to the No CMDs subgroup (20%). Chi-squared for independence (p=0,14), for trends (p=0,051), but the difference and the trend were not significant. The frequencies of muscle relaxation drugs were as follows Occipital neuralgia subgroup (60,9%); Tension-type headache subgroup (62%); No CMDs subgroup (26,7%). Chi-squared for independence (p<0,002), for trends (p<0,005): Occipital neuralgia versus Tension-type headache (p=1,000); Occipital neuralgia versus No CMDs (p<0,009), Tension-Type headache versus no CMDs (p<0,002).*
Conclusions: *Occipital neuralgia subjects more frequently used analgesics, anti-anxiety, antidepressants and muscle relaxation drugs as compared with the other two subgroups.*

Keywords: *Occipital Neuralgia. Analgesics. Muscle Relaxation. Antidepressants. Anti-anxiety dugs.*

Date of Submission: 25-02-2019 Date of acceptance: 11-03-2019

I. Introduction

Occipital neuralgia (ON) is a severe pain disorder usually described as deep, throbbing and aching pain occurring in the distribution of the second and third cervical dorsal root which manifests in the form of lancinating, paroxysmal pain, radiating to some anatomic areas innervated by the trigeminal nerve[1]. This severe pain usually occurs in the proximities of the nuchal line radiating to the ipsilateral hemicranium but may involve the whole head when it occurs in the bilateral form. Other characteristics of this pain disorder

include its distribution in the greater, lesser and/or third occipital nerves, severe tenderness over the affected nerve or pain generating zone which may be abolished immediately by a local anesthetic block of the nerve[2].

ON, tension-type headache (TTH) and even migraine can be moderate or mild at some time during their course. However, when these pain disorders become chronic, pain episodes can last hours or even days being described as very severe[3]. When the causes of the headache are not tackled properly and when medication is the only mode of treatment used by the affected patient, pain episodes may become more chronic, frequent and severe in intensity[3]. In ON pain, a moderate headache may be exacerbated by psychiatric disorders including anxiety, depression, somatization and panic disorders[4]. Clinically, ON is considered a chronic form of headache characterized by severe, persistent and unbearable pain which leads patients to use many different forms of medication including antidepressants, non steroidal analgesics and even opioids [5].

Longer duration and greater intensity of pain in ON may cause depression and thus, encourage patients to use antidepressants more frequently as compared with other types of headache disorders. Even though antidepressants are widely used to treat both depression and severe pain, there are no epidemiological studies about current use of antidepressants in large samples of patients presenting with ON and concomitant craniomandibular disorders (CMDs). Thus, this investigation is aimed at:

Test the hypothesis that there is a higher frequency of analgesics, antidepressants, anti-anxiety and muscle relaxation drugs use in those presenting with ON as compared with two control subgroups.

II. Methods

Sample

Subjects participating in this study were those referred consecutively to an Orofacial Pain facility at the University of Gurupi-TO (Brazil) in the period 2010-2018. Subjects were evaluated comprehensively gathering data from their pain history, gentle palpation of muscle and joints, use of comprehensive questionnaires to collect information about signs and symptoms of bruxing behavior (BB), ON, CMDs and use of past and current medication. During clinical evaluation the principles of the Helsinki Declaration were followed: Patients were informed that their clinical evaluation and use of questionnaires had no absolute risk for their health, that any physical and psychological discomfort warranted the discontinuity of the evaluation, that confidentiality was absolute, that accurate evaluation was necessary in order to obtain accurate data and diagnosis before planning any treatment, that the principal examiner was scientifically experienced and qualified and that his or her data would provide practical and clinical benefits in future studies and treatments. Patients signed a formal consent allowing to use their records with demographic and clinical data for research purposes. Patients and those in the control groups were evaluated in the same period of time. The research involved a total of 185 charts retrieved and evaluated retrospectively: 105 with ON and CMDs, 50 with TTH and CMDs and 530 with no TH and no CMDS. Once the clinical examination and collecting data procedure were carried out, all those with a diagnosis of BB, CMDs and ON were stored in a database with the name "Occipital neuralgia patients". Once we observed that there was a large sample of ON, TTH and No CMDs subjects, their chart were reviewed retrospectively in order to carry out a comparison study about use of medications.

Inclusion criteria for BB: Patient´s report of catching himself/herself clenching and/or grinding the teeth at daytime and/or during the night, a friend, spouse or relative´s report of grinding the teeth at night, patient´s report of fatigue in the masticatory muscles following eating and/or speaking, awakening with facial, TMJ and/or head pain, dental pain and/or jaw locking on awakening in the morning.

Inclusion criteria for CMDs: Patient´s report of joint noises, a complaint of TMJ, facial pain, tenderness to palpation of the TMJ and/or masticatory muscles, impaired jaw movements and headache of neuromuscular origin

Inclusion criteria for TTH: A report of temporal and/or frontal headache, pain described as dull, pressing, steady, band-like type, moderate in intensity in both sides, nausea reported occasionally, absence of vomiting, presence of trigger point and muscle tension in the cervical region directly associated with referred pain to the anterior part of the head.

Inclusion criteria for those in the Non CMDs subgroup: Patients in this subgroup were included as controls if they not fulfilled criteria for CMDs, presented with some type of painful complaint and BB, they had no clinical signs and symptoms of ON, but some reported a complaint of headache.

Exclusion criteria for subjects in the experimental and control subgroups: Subjects were not evaluated comprehensively and their charts were not retrieved for research purposes if they demonstrated severe psychological or psychiatric disorders, cognitive impairment, difficulties to respond properly to questionnaires, presence of neuromuscular disorders, for instance, some type of epilepsy or tremor in the orofacial region. Their charts were not included in the current retrospective study.

III. Statistical analysis

Kruskal-Wallis statistics, Chi-Squared for independence and trends and Fisher´s exact test were used to analyze data in the current investigation.

IV. Results

There were 100 females and 5 males in the ON subgroup,48 females and 2 males in the TTH subgroup and 23 females and 7 males in the Non CMDs subgroups: ON versus TTH (Fisher´s exact test p=1,000); ON versus Non CMDs subgroup (Fisher´s exact test p<0,005); TTH versus No CMD subgroup (Fisher´s exact test p<0,01). Mean age in the ON + CMDs subgroup was about 39,2 (SD=9,7 range 18-75), 37,3 (SD=12,8 range 18-72) in the TTH + CMDs subgroup and 32,7 (SD=14,2, range 14-70) in the Non CMDs subgroup (Kruskal-Wallis´ statistics p<0,003): ON versus TTH subgroup (p>0,05); ON versus No CMD subgroup (p<0,01); TTH versus No CMDs subgroup (p>0,05). 95,2%, 76%, and 43,4% in the ON + CMDs, TTH + CMDs and No CMDs subgroups respectively, reported use of analgesics to treat their pain (Chi-squared for independence p<0,0001, for trends p<0,0001): ON subgroup versus TTH subgroup (p<0,0003); ON versus No CMDs subgroup (p<0,0001); TTH versus No CMDs subgroup (p<0,003). Means in the use of different analgesics or anti-inflammatory drugs to treat painful symptoms were about 3,01, 2,3 and 1,5 in the ON, TTH and no CMDs subgroups, respectively (Kruskal-Wallis´statistics p<0,0001): ON versus No CMDs (p<0,001). 23,8%, 10% and 3,3% in the ON + CMDs, TTH + CMDs and No CMDs subgroups, respectively, reported the use of anti-anxiety drugs (Chi-squared for independence p<0,009, for trends p<0,003):Fisher´s exact test ON versus TTH subgroup (p<0,05), ON versus No CMDs subgroup (p<0,009), TTH versus No CMDs subgroup (p=0,40). Current/past use of antidepressants was reported by 39% in the ON subgroup, by 32% in the TTH subgroup and 20% in the Non CMDs subgroup (Chi-squared for independence p=0,14, for trends p=0,051). Even though past/current use of antidepressant was reported more frequently by the ON subgroup, the difference was no statistically significant when pairs of subgroups were contrasted. Use of muscle relaxation drugs were reported by 60,9, 62% and 26,7% of the ON, TTH and No CMDs subgroups, respectively (Chi-squared for independence p<0,002, for trends p<0,005): ON versus TTH subgroup (Fisher´s exact test p=1,000); ON versus No CMDs subgroup (p<0,001); TTH versus No CMDs subgroup (p<0,002).

V. Discussion

Data in the current investigation demonstrated a higher frequency of analgesics and or anti-inflammatory use among subjects presenting with ON and CMDs. These drugs do not constitute the most effective mode of treatment for ON. However, it may be that such individuals used such drugs as in the first stage of their disease process they were not aware of the severity and complications of their cranial neuralgia. Some researchers[6] consider that these medications tend to be the mainstay for neurologists when they do not perform local injections with anesthetics. Such drugs may be used as a initial treatment attempting to break the pain cycle[6]. A pharmacological approach to cervicogenic headache including ON is recommended as a potential treatment intervention including the use of non-steroidal, anti-inflammatory drugs, for instance, indomethacin, ibuprofen, naproxen and celecoxib[7]. One investigation[8] asserts that pharmacologic treatments used initially for ON pain include non-steroidal anti-inflammatory (NSAIDS), anti-epileptic and possibly opioid drugs. In the current study, ON subjects reported a mean of three different analgesic and/ anti-inflammatory drugs prescribed to alleviate ON pain. It may be that because analgesics were not effective alleviating such a severe headache, patients shifted frequently from one drug to the other attempting to get relief of their pain. In this regard, one investigation[9], reported that all patients in one series of ON patients had refractory pain with no significant relief from previous nonsurgical treatment consisting of both analgesics and anti-inflammatory drugs.

ON and CMDs individuals reported a higher frequency of use of anti-anxiety drugs. It may be that because ON pain is difficult to diagnose, treat and becomes associated with both peripheral and central sensitization, the pain becomes more recalcitrant, difficult to treat with time, and previous drugs provide only transient and or insufficient relief. Thus, patients become more anxious and depressed, increasing their use of both antidepressants and anti-anxiety medication. Support for this assumption comes from one investigation[12] and case presentation reporting that ON may be associated with depression, anxiety and other psychological disorder, including worry and apprehension associated with failure of previous medication for pain relief[13]. Chronic headache including chronic migraine and ON can lead to peripheral and central sensitization and aggravation of peripheral neuralgias, thus, seriously limiting the efficacy of medication[14]. The efficacy of simple analgesics tends to decrease with increasing frequency of headaches. Headaches are often associated with stress, anxiety and depression[15]. Thus, it is very likely that following the observation of the inefficacy of analgesics and anti-inflammatory drugs, patients become more anxious, depressed and shift to other types of medication including anti-anxiety and antidepressants.

The frequency of use of antidepressant drugs was higher among individuals with ON and CMDs. Some types of pain disorders in the craniofacial region are very intense, incapacitating and do not respond to conventional pharmacologic treatment. In this clinical situation, patients become anxious, apprehensive and depressed. Thus, they may use more medication including anti-anxiety and antidepressant drugs to ameliorate their pain. Although not statistically significant, a trend to increased use of antidepressants was observed from the No CMDs to the TTH and then to the ON subgroup. No doubt, such increased of antidepressant use is related with more intense, chronic and frequent pain. A combination of drugs including ibuprofen, naproxen, indomethacin and tricyclic antidepressants is usually recommended for ON treatment[7]. Amitryptiline with a dose of 25 mg daily is one of the most common antidepressants used in the treatment of cranial neuralgias[10]. A recent review[6] about cranial neuralgias indicates that a combination of tricyclic antidepressants, oral steroids, and anti-epileptic drugs, is more effective in eliminating pain in cranial neuralgias. Any type of headache pain is best managed with a combination of amitryptiline, NSAIDs and anti-epileptic drugs[11]

Muscle relaxation drugs are more frequently used in individuals presenting with ON and CMDs. The frequency of muscle relaxation drugs in the current investigation was higher in the subgroups presenting with ON and TTH as compared with the second control subgroup It may be that many clinicians recognize the role of muscle tension, muscle hyperactivity and the presence of trigger points and a pain generating zone as an important pain mechanism in patients diagnosed with ON. This assumption is congruent with one investigation[7] reporting that muscle relaxation drugs including tizanidine, baclofen and cyclobenzaprine constitute potential treatment interventions in patients presenting with ON. Tricyclic antidepressants, amitryptiline, NSAIDS, muscle relaxants , anticonvulsants and even botulinun toxin constitute a set of prophylactic medication which has a role in the treatment of TTH and other severe headaches. Conservative treatment for ON includes the use of posture correction and reducing both neuralgic and muscle pain[17]. Spasm of the trapezius muscle may also be associated with chronic local contraction and muscle hyperactivity[18], thus facilitating the formation of trigger points which eventually may cause pressure on nervous structures

VI. Conclusion

ON subjects more frequently used larger amounts of analgesic/anti-inflammatory, anti-anxiety, antidepressants and muscle relaxation drugs, indicating the complexity of ON, patients´ suffering and difficulties to alleviate their pain and psychological suffering. More studies are mandatory using similar methods to further substantiate findings in the current research.

References

[1]. Kapural L, Sable J. Peripheral nerve stimulation for occipital neuralgia: Surgical leads. J Prog Neurol Surg 2011; 24: 86-95.
[2]. Jung SJ, Moon S, Kim T, Eom K. A case of occipital neuralgia in the greater and lesser occipital nerves treated with neurectomy by using transcranial doppler sonography: Technical aspects. Korean J Pain 2011; 24: 48-52.
[3]. Beran R. Management of chronic headache. Aust Fam Phys 2014; 43: 106-10.
[4]. Kaniecki RG. Tension-type headache. Continuum Lifelong Learning Neurol 2012; 18: 823-34.
[5]. López PJ, Bretones JM, Garcia M, Sánchez E, Rodríguez MA. Occipital neuralgia: a noninvasive therapeutic approach. RLAE 2018; 26: 1-7.
[6]. Taylor MT. Treatment options or cranial neuralgias. Pract Neurol 2016; May: 26-28.
[7]. Biondi D. Cervicogenic headache: D iagnostic evaluation and treatment strategies. Pain Management Rounds; 2004; 1: 1-6.
[8]. Manolitsis N, Elahi F. Pulsed radiofrequency for occipital neuralgia. Pain Phys 2014; 17: 709-17.
[9]. Kapoor V, Rothfus W, Grahovac SZ, Kassam SZA, Horowitz M. Refractory occipital neuralgia: Preoperative assessment with CT-guided nerve block prior to dorsal cervical rhizotomy. AJNR 2003; 2105-10.
[10]. Gadient PM, Smith JH. The neuralgias: Diagnosis and management. Curr Neurol Neurosci Rep 2014; 14: 459-66.
[11]. Zhang GQ, Meng Y. Oral and craniofacial manifestations of multiple sclerosis: implications for the oral health care provider. Eur Rev Med Pharmacol Sci 2015; 19: 4610-20.
[12]. Skaribas I, Calvillo O, Delikani-Skaribas E. Occipital peripheral nerve stimulation in the management of chronic intractable occipital neuralgia in a patient with neurofibromatosis type 1: a case report. J Med Case Rep 2011; 5: 174-79.
[13]. Ducic I, Hartmann EC, Larsson EE. Indications and outcomes for surgical treatment of patients with chronic migraine headaches caused by occipital neuralgia. Plast Reconstr Surg 2009; 123: 1453-61.
[14]. Elsayed AA, Kreuer L, Wheeler S, Robillard J, Seeger S. Radiofrequency ablation of pericranial nerves for treating headache conditions: A promising option for patients. Ochsner Journal 2018; 18: 59-62.
[15]. Bendtsen L. Drug and nondrug treatment in tension-type headache. Ther Adv Neurol Disord 2009; 2: 155-61.
[16]. Bendtsen L, Jensen R. Pharmacological and non pharmacological management of tension-type headache. Headache
[17]. Choi I, Jeo SR. Neuralgias of the head. JKMS 2016; 31: 479-88.
[18]. Trescot A. Headache management in a interventional pain practice. Pain Phys 2000; 3: 197-300

Table 1: Social and demographic data in Occipital neuralgia (ON subgroup n=105), Tension-Type headache (TTH subgroup n=50) and Non Craniomandibular subjects (Non CMD subgroup n=30).

	SUBGROUPS					
	ON=105		TTH=50		NO CMDs=30	
GENRE	n	%	n	%	n	%
Females	100	95,2	48	96	23	76,7
Males	5	4,8	2	4	7	23,3
Totals	105	100	50	100	30	100
AGE						
Mean	39,2		37,3		32,7*	
SD	9,7		12,8		14,2	
Range	18-75		18-72		13-70	

*Kruskal-Wallis´ test p<0,003: ON versus TTH (p>0,05); ON versus No CMDs (p<0,01); TTH versus No CMDs (p>0,05).

Table 2: Use of analgesics in the subgroups presenting Occipital neuralgia (ON=105), Tension-Type Headache (TTH=50), and No Craniomandibular Disorders (No CMDs=30).

Use	Analgesics Anti-inflammatory					
	ON	%	TTH	%	No CMDs	%
YES	100	95,2	38	76	13	43,4*
NO	5	4,8	12	24	17	56,6
TOTALS	105	100	50	100	30	100
MEAN	3,01		2,3		1,5**	
SD	1,6		1,7		2,4	
RANGE	0-8		0-7		0-9	

*Chi-squared for independence (p<0,0001), for trends (p<0,0001). Fisher´s exact test: ON versus TTH (p<0,0003); ON versus No CMDs (p<0,0001); TTH versus No CMDs (p<0,003).
**Kruskal-Wallis test (p<0,0001): ON versus TTH (p>0,05); ON versus No CMDs (p<0,001); TTH versus No CMDs (p>0,05).

Table 3: Use of anti-anxiety, antidepressants and muscle relaxation drugs in ON (n=105), TTH (n=50) and No CMDs (n=30) subjects .

	SUBGROUPS						
	ON=105		TTH=50		NO CMDS=30		
Anti-anxiety	n	%	n	%	n	%	
Yes	25	23,8	5	10	1	3,3*	
No	80	76,2	45	90	29	96,7	
Totals	105	100	50	100	30	100	
Antidepressants							
Yes	41	39	16	32	6	20**	
No	64	61	34	68	24	80	
Totals	105	100	50	100	30	100	
Muscle Relaxants							
Yes	64	60,9	31	62	8	26,7***	
No	41	39,1	19	38	22	73,3	
Totals	105	100	50	100	30	100	

*Chi-squared for independence (p<0,009) for trends (p<0,003).
Fisher´s exact test: ON versus TTH (p<0,05); ON versus No CMDs (p<0,009); TTH versus No CMDs (p=0,40).
**Chi-Squared for independence (p<0,14) for trends (p>0,051)
***Chi-squared for independence (p<0,002), for trends (p<0,005).
Fisher´s exact test: ON versus TTH (p=0,90); ON versus No CMDs (p<0,0009), TTH versus non CMDs (p<0,002).

Omar Franklin Molina.. "The Profile of Occipital Neuralgia Patients Regarding Past/Current Use of Medication." OSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 3, 2019, pp 66-70.