IJBPAS, August, 2015, 4(8): 5731-5738

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

ATYPICAL ODONTALGIA, CURRENT KNOWLEDGE ON PATHOPHYSIOLOGY, DIAGNOSIS AND TREATMENT-A REVIEW

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ABSTRACT

Atypical odontalgia(AO) is a chronic orofacial pain condition which may be confusing for clinicians to diagnosis and frustrating for patient because it may lead to several wrong treatments without any effective achievement. This review tries to discuss new findings in etiology, diagnosis and treatment options for better understanding and management of AO. Recent studies emphasizeon neuropathic origin of AO based on reliable results of QuantitativeSensory Tests(QST).These tests in conjunction with appropriate history also provide a well diagnostic tool for clinicians.It is also important to evaluate patient`s psychological background because it`s accepted that psychologic disorders have high prevalence among AO patients and they can complicate the pharmacologic and behavioral management of patient for clinicians.

Keywords: Atypical odontalgia, Chronic pain, Neuropathy, Psychology, Antidepressant, Anticonvulsants

INTRODUCTION

Atypical odontalgia (AO) was first reported by McEllin and Horton in 1947[1]. It was defined as a dull throbbing pain localized to dentoalveolar tissues or teeth [2]. There may or may not be a history of dental

treatment prior to onset. The pain may move from one area to another [3].

Atypical odontalgiahas been reported with different incidences from 2.1% [4]to 12%[5] in the literatures. It is frequently reported that females have high tendency to AO[6] and upper molars are more susceptible to it[7, 8].Several diagnostic criteria and classification have been proposed for diagnosis of AO[9, 10].Based on IHS(International Headache Society) classification atypical odontalgia is "Persistent facial and/or oral pain, with varying presentations but recurring daily for more than 2 hours per day over more than 3 months, in the absence of clinical neurological deficit."

The IHS diagnostic criteria is:

A. Facial and/or oral pain fulfilling criteria B and C

B. Recurring daily for >2 hours per day for >3 months

C. Pain has both of the following characteristics:

1. Poorly localized, and not following the distribution of a peripheral nerve

2. Dull, aching or nagging quality

D. Clinical neurological examination is normal

E. A dental cause has been excluded by appropriate investigation

F. Not better accounted for by another ICHD-3 diagnosis[11].

There are lots of case reports and reviews of literature in this topic. This article tries to review the recent data in pathophysiology, diagnostic tools and treatment options of AO.

PATHOPHYSIOLOGY OF AO

Neuropathic Pain

The most acceptablepathophysiology for AO is a neuropathic pain condition[12]. In general Neuropathic pain may affect central nervous system(CNS) or peripheral nervous system(PNS)[13]. Trigeminal neuralgia, Atypical odontalgia, Burning Mouth Syndrome, PostherpeticNeuralgia have been stated to be peripheral neuropathic pain which occure in the orofacial region[14]. Although some authors classified AO as idiopathic facial pain[15], several studies have found correlation between AO and neuropathy. It has been reported that in 83% of AO patients pain started after invasive dental procedures such as root canal treatment or tooth extractions [16]. Gatchel et al reported Hyperactivation of Aβ, Aδ, and C fibersin AO[23].There was evidence of derangement in the periodontal plexus and disorganization like axonal sprouting in animal studies after loss of tooth pulp in an inflamed enviorment [17].Somatosensory abnormalities has been found in 87% of the included AO patients[16].There are also some reports of other pain conditions in

AO patients. High prevalence of episodic Tension-Type Headache (TTH), chronic TTH, and Myofascial temporomandibular disorder in AO patients were reported. There is no agreement on accompaniment between Burning Mouth Syndrome and (BMS) and AO [12, 14]. These findings all suggest the role of neuropathy in this disorder.

Psychologic Pain

It has been reported that AO patients havehigh prevalence of psychological problems. Baad-Hansen et al evaluated psychologic disorders of 46 AO patients and reported moderate to high depression and somatization scores[18].Anxiety[19],Somatoform pain disorder, Introversion[20] were also reported in patients with AO. However this cormobidity may not reflect the psychiatric origin of AO. Some authors believed that psychologic problems was not the causeof AO and itmay be a consequence of chronic pain condition[18, 21]. Takenoshita in psychologic assessment of AO patient found that 33% of AO patients had no specific psychiatric diagnosis[22].It seems that psychologic factors can influence the pain in AO patients regardless of it`s origin. Gatchel *et al.*, defined Psychogenic pain as a pain condition which is caused or prolonged by mental, behavioral or emotional factors [23].It is accepted that emotional status can change the perception of pain[24-26]. This highlight the importance ofassessing patient`s psychological background prior to treatment. It is preferable to benefit from team work to control all aspects of pain.

DIAGNOSTIC TOOL FOR ATYPICAL ODONTALGIA

In assessment of AO patient it is important to rule out any pathologic lesion in the pain site.It has been shown that Cone beam CT(CBCT) can provide good information about probable problems which periapical radiograph couldn`t reveal it.Pigg et al examined the diagnostic yield of the Cone Beam CT(CBCT) and periapical radiograph the affected area of pain in AO patients.He found that sixty percent of patients with AO didn`t show any periapical bone destruction in CBCT.Overall, CBCT rendered 17% more periapical bone destructions than conventional radiography[27]. In another study, this author used MRI in AO patients and examine the signal changes in affected area.He has reported that MRI examination failed to show any changes in painful region. There was correlation between findings of radiographic and MRI examination [25].

QST have been shown reliable results in AO patients.These tests are used to assess the presence and severity of neuropathic

pain[22].The AO patients showed multiple somatosensory abnormalities in Quantitative Sensory Tests[10, 20, 21]. Although no simple or pathognomonic QST profile has been detected in AO patients[10]. There are reports of hyper and hyposensitivity to mechanical stimuli of the face[11], increased heat pain sensitivity[11, 12] and increased sensitivity to topical application of capsaicin12].It should be keep on mind that findings of QST can be interpreted in presence of other findings such as patient medical and dental history, radiographs and other paraclinical tests(if there is any indication).

TREATMENT OF PATIENTS WITH ATYPICAL ODONTALGIA

A serious challenge in the treatment of patients with AO that they may notbe convinced that CNS plays a rolein stimulating of peripheral tissues. This may lead to patients` lack of cooperation with physician and their early drop out of the treatment to seek for another dental procedures[15].

Several treatments have been suggested for pain relief in AO patients.The effectiveness ofTricyclic antidepressants(TCAs) in neuropathicpain is well documented[28, 29]They act in a multimodal manner, with contribution ofmonoamine reuptake inhibition and blockade ofN-methyl-D-aspartate (NMDA)receptors, sodium channels and calcium channels[30]. Application of these drugs in orofacial pain has been also documented[27-29].Vickers reported pain relief after application of Nortriptyline in orofacial pain condition[31].Anticonvulsantshave been also used for chronic neuropathic pain.These drugs have fewer side effects than TCAs so they can be used alone or in conjunction with TCAs to reduce their side effects[18].Howevere, Recent data shows that anticonvulsants may be not as effective as antidepressants in treatment of atypical odontalgia[32].SNRI(Serotonin Norepinephrine Reuptake Inhibitors) family of antidepressants are newer medicaments which have less side effects than TCAs. Duloxetine, Venlafaxine, Milnacirine, Desvenlafaxine are in this category. The mechanism of these drugs are different based on their dosage[33].There are some reports of successful management of AO with Duloxetine[34], Milnacipran[28]butForssell et al reported that Venlafaxine didn`t have considerable effect on reducing facial neuropathic pain[35].Other approach in treatment had been also examined.Administration of local anesthesia significantly reduced patients pain but it couldn`t remove the pain completely[36].Attempts to pain control withintra-venous infusion of fentanyl (a l-

opioid agonist) and S-ketamine werefailed[37].When peripheral blockade of AO pain with lidocaine showed some degree of pain relief[38],sympathetic blockade showed different results from 0% to 80% pain relief.This may be due to combination ofperipheral and central factors influence this type of pain[39].There is also a report of pain relief with subcutaneous application of sumatriptan in AO patients[40].In addition to prescribe medicaments Zakrzewska suggested Cognitive Behavioral Therapy as a part of treatment procedure[34].

CONCLUSION

The exact mechanism in pathophysiology and etiology of AO is still unknown. It seems that neuropathic mechanisms are involved. Psychological disorders have high prevalence between AO patients which complicate the pain condition and also management of patients.It is recommended that evaluation of psychologic background of AO patients be a part of treatment procedure. This may help the clinicians to better pain control and decrease the number of unnecessary dental treatments.

ACKNOWLEDGEMENT

None declare

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