



The Acquired form of Eagle Syndrome includes Dental Vascular Pathology

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Abstract

Periodontitis is a chronic inflammatory response to bacterial plaque that causes tooth mobility and loss by destroying the supporting soft tissues and anchoring bone. The infection that causes dental caries spreads from the dentine to the vascular dental pulp and periapical bony tissues before affecting adjacent soft tissues and causing sepsis to spread. Periodontitis is linked to increased cardiovascular, cerebrovascular, and peripheral artery disease, as measured by clinical disease, angiography, ultrasonography, and reduced flow-mediated dilation, according to a number of case-controlled, cross-sectional, and cohort studies. This review identifies the necessity of further investigation into these associations because some studies report a similar relationship between atherosclerosis and periapical infection and possibly also with coronal caries.

Keywords: Atherosclerosis; Epidemiologically; Chronic inflammatory; Otolaryngologist

Introduction

Atherosclerosis and periodontitis share epidemiologically confounding environmental risk factors, including smoking and exposure to cadmium. The risk factors for both atherosclerosis and periodontitis, with which periodontitis appears to have distinct positive feedback relationships, further complicate epidemiological studies. Diabetes, elevated plasma lipid levels, hypertension, and a low white blood cell count are some examples. Periodontitis may play a causal role in atherosclerosis through bacterial invasion of arteries, specific atherogenic properties of oral bacteria, the acute phase response, and cytokine polymorphisms, according to animal and human intervention studies [1].

The otolaryngologist at Duke University, Watt W. Eagle, who reported the first cases in 1937, gave the condition its name. It is a rare condition brought on by an elongated or disfigured styloid process that causes orofacial and cervical pain that is often brought on by movements of the neck. This pain interferes with the operation of the structures that are nearby and causes the condition. Some authors refer to Eagle syndrome as stylohyoid syndrome, styloid syndrome, or styloid-carotid artery syndrome [2].

Etiology

The cause of Eagle syndrome is up for debate. Dr. Watt Eagle proposed that reactive, ossifying hyperplasia was brought on by osteitis, periostitis, or tendonitis of the styloid process and the stylohyoid ligaments as a result of surgical trauma (a tonsillectomy). Later, in 1975, Lentini proposed the idea that appropriate traumatic or stressful events might cause persistent mesenchymal elements, also known as Reichert cartilage residues, to undergo osseous metaplasia. In 1962, Epifanio believed that the ossification of the styloid process was linked to endocrine disorders in women who were entering menopause and also had ossification of other body ligaments. C. Gokce et al. According to a 2008 study, patients with end-stage renal disease who had abnormal metabolisms of calcium, phosphorus, and vitamin D had heterotopic calcification, which led to styloid process elongation and the onset of Eagle Syndrome. Finally, in a 2015 retrospective study, Sekerci found a correlation between the presence of an elongated styloid process and an arcuate foramen. Using three-dimensional CT scan data from 542 patients, the results were derived [3].

Epidemiology

At a very early stage, Eagle reported that the normal styloid process measured less than 2.5 centimeters in length, and that any process that was longer than 2.5 centimeters could be deemed abnormally elongated. The length of the styloid process was later determined to range from 1.52 to 4.77 cm during a postmortem examination of 80 cadavers. About 4% of the general population has an elongated styloid process, but only about 4% of these people have symptoms that are related to the elongation of the styloid; Consequently, the actual incidence is approximately 0.16 percent, with a 3:1 ratio of females to males. Patients typically exceed 30 years of age, and although unilateral cases do occur, this is typically a bilateral process [4].

Pathophysiology previously

It was hypothesized that the neurovascular structures in the retro styloid compartment were compressed and stressed as a result of the formation of scar tissue around the styloid apex following tonsillectomy. However, patients who have never had a tonsillectomy can also develop Eagle syndrome. The pathogenesis of pain in Eagle syndrome has been linked to a number of different theories. The first hypothesis is that the elongated styloid process compresses cranial nerves, most frequently the glossopharyngeal nerve, resulting in pain in the neck and throat. Alternately, the internal carotid artery could be compressed by the styloid process, which could result in transient ischemic attacks or compression of the sympathetic nerves that run along the artery, which could cause a variety of symptoms. Although the pain in Eagle syndrome is typically more dull and constant than in glossopharyngeal neuralgia, cases with sharp, intermittent pain along the glossopharyngeal nerve path have also been reported [5].

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Received: 05-June-2023, Manuscript No: jdpm-23-104062; **Editor assigned:** 07-June-2023, PreQC No. jdpm-23-104062 (PQ); **Reviewed:** 21-June-2023, QC No. jdpm-23-104062; **Revised:** 23-June-2023, Manuscript No. jdpm-23-104062 (R); **Published:** 30-June-2023, DOI: 10.4172/jdpm.1000161

Citation: Zhaouju S (2023) The Acquired form of Eagle Syndrome includes Dental Vascular Pathology. J Dent Pathol Med 7: 161.

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Pathophysiology

Reactive hyperplasia and reactive metaplasia that link the elongation to either trauma-induced ossification of the stylohyoid ligament complex or overgrowth of the styloid process itself. Eagle syndrome, as it was initially described by Eagle, may be explained by this phenomenon in patients who have had their tonsils removed. The irritated adjacent musculature or mucosa caused by the abnormal angulation of the abnormally long styloid process is another potential cause. The fifth, seventh, ninth, and tenth cranial nerves stretching and fibrosis in the post-tonsillectomy period could also be a possible cause. Last but not least, it's possible that the symptoms are the natural course of aging. Degenerative and inflammatory changes in the tendinous portion of the stylohyoid insertion, a condition known as insertion tendinosis, may cause pain in the glossopharyngeal nerve distribution resembling Eagle syndrome, as normal aging is associated with a decrease in soft tissue elasticity. This better manifestation is referred to as a pseudo-stylohyoid syndrome to avoid confusion [6].

Material and Methods

Assessment of the periodontium

An orthopantomogram was used to check for caries and other dental infections in the patients; Additionally, a standard periodontal screening examination was performed. Those who met the dental inclusion criteria and had Armitage's description of severe periodontitis were invited to participate in the study. A single examiner used a standardized, pressure-calibrated periodontal probing device (Florida Probe) with 1-mm markings to conduct a comprehensive periodontal and clinical assessment of all teeth following randomization. This one examiner was blinded to the treatment allocation at all times during the study [7].

For each and every tooth, the following parameters were frequently evaluated: periodontal pocket depth (PPD), which is defined as the distance from the marginal gingiva to the depth of the pocket, and clinical attachment level (CAL), which is defined as the distance from the cemento-enamel junction or restorative margin to the depth of the pocket, are all indicators of periodontal health. Six locations were used to test each parameter, with the exception of PI, which was tested at four locations. Periodontal inflammation was expressed as a measure of the periodontal inflamed surface area (PISA), as was suggested at the joint workshop on periodontitis and atherosclerotic cardiovascular diseases held by the EFP and AAP. PISA is the total number of sites with BOP that show periodontal pockets. Both at the follow-up (FU) sessions and the baseline (BL) sessions, all clinical assessments were carried out [8].

Groups treated without surgery and those treated with antibiotics

Patients were randomized to one of the two non-surgical treatment groups and given instructions on how to control plaque and underwent a session of supragingival scaling and polishing. Both groups did not include antibiotic therapy. The primary endpoint (PET/CT analysis) was not assessed or reassessed during the periodontal treatment by a single, experienced periodontist. Patients underwent a one-stage full-mouth disinfection within a week.

In the first OSFMD session, antibiotic therapy and daily chlorhexidine rinses were started for the PT1 group. Systemic antibiotics (500 mg of amoxicillin, 125 mg of clavulanic acid, and 500 mg of metronidazole) were administered to the PT1 group of patients. For seven days, they were instructed to take the two antibiotics three

times daily. The patients' oral hygiene standards were reviewed and oral hygiene procedures were reinforced from the post-treatment phase until the follow-up [9].

Result and Discussion

Prevalence of periodontal diseases

Between March 2013 and December 2015, 421 consecutive patients with PAD were referred to our vascular outpatient clinic and considered eligible for the study. After that, they were asked to come in for a periodontal exam. Twelve patients rejected further dental assessment, leaving a pool of 409 patients (114 ladies) who partook in additional periodontal assessment. 270 of these patients failed to meet the dental inclusion criteria for the following reasons: lacked at least two teeth with a probing depth greater than 5 millimeters and/or at least two teeth with a clinical attachment loss greater than 5 millimeters (n = 134), lacked at least twelve natural teeth, including third molars (n = 127), lacked more than 20% of probing sites that displayed bleeding (n = 5), or had received periodontal treatment within six months of the study (n = 3) [10].

After the screening, 14 patients were left out because they were participating in another clinical trial, had a life expectancy of less than six months due to carcinoma or massive aortic abdominal aneurysm, or were allergic to penicillin (n = 4). After receiving a detailed introduction to the study protocol, 35 patients chose not to participate in the RCT for personal reasons. 0.2% of the patients had no clinical signs of periodontal disease. 125 patients had gingivitis at a prevalence of 30.6%. 64 patients had moderate periodontitis, with a prevalence of 15.6%, and 219 patients had severe periodontitis, with a prevalence of 53.5%.

Conclusion

Although the current study characterized dental and TMJ pathology in Arctic foxes primarily from Alaska's coastal regions, the use of museum specimens to examine disease and abnormalities in wild populations has a few limitations. First, because many of the specimens were likely collected from trappers and/or from locations that are simple for humans to access, the frequency of pathology such as tooth fractures and trauma in the study population is probably higher than what is observed in wild populations [11]. Additionally, the majority of specimens were collected in the 1950s and 1960s, so data collection was inconsistent over time. Despite the fact that the majority of abnormalities did not differ significantly between mid-century foxes and modern foxes, the frequency of the abnormalities that were found may differ from that of the live populations that are currently in existence. Finally, the quality of specimens and, ultimately, the precision and accuracy of the data collected are impacted by museum storage methods and specimen care. It is possible to mistake artefact changes caused by processing and storage for ante-mortem pathology.

Acknowledgement

None

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