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Case Report

Periodontal findings in a rare case of Takayasu arteritis

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ABSTRACT

Chronic periodontitis has been associated with many systemic diseases and many times chronic systemic diseases result in periodontal manifestations. An association between cardiovascular disease and periodontitis has been observed based on the current literature. The present case shows a rare finding of chronic periodontitis in a male patient as a result of an inflammatory disease of the blood vessel called Takayasu arteritis, which itself is a rare finding in a 31-year-old male patient. This case report attempts to highlight the role of diagnosis and understanding the etiology behind periodontal disease as a result of a rare systemic inflammatory disease, such as Takayasu arteritis.

1. Introduction

Periodontitis has been defined by the consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions as "a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms and characterized by progressive destruction of the tooth-supporting apparatus. Its primary features include the loss of periodontal tissue support, manifested through clinical attachment loss (CAL) and radiographically assessed alveolar bone loss, presence of periodontal pocketing and gingival bleeding."[1]

According to Albander et al.[2], numerous systemic diseases result in periodontal manifestations in the oral cavity. The systemic diseases/conditions which have an impact on periodontal disease range from diabetes mellitus, genetic diseases, immunologic disorders, neoplasm, and inflammatory disorders. Literature suggests that longstanding severe periodontal infection can affect endothelial function and worsen cardiovascular diseases such as ischemic heart disease ultimately resulting in myocardial infarction (MI). Evidence on periodontal manifestations as a result of cardiovascular disease is scarce and is limited to changes in gingival color or changes in the texture of the tongue due to congenital heart disease, Tetralogy of Fallot, and Eisenmanger syndrome.[3,4] Some rare diseases have some oral manifestations in the form of periodontal disease. An example of such a disease could be Takayasu Arteritis. Takayasu's arteritis (TA) is known by many names such as pulseless disease, aortic arch syndrome,[5] idiopathic arteritis, reverse coarctation,[6], occlusive thromboartopathy, and Martonell Syndrome.[7]

Named after an ophthalmologist Mikito Takayasu from Japan, TA is defined as a rare, chronic granulomatous inflammatory disease of arteries that are large and medium such as the aorta and its major branches.[6–9] It consists of segmental lesions which occur next to unaffected areas. These lesions include stenosis, occlusion, or dilation/aneurysm of the blood vessel.[6] The present case report highlights the findings of a male patient with advanced periodontal disease possibly as an indirect effect of Takayasu Arteritis.

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2. Case Report

A 31-year-old adult male patient reported to the Department of Periodontology with the chief complaint of loosening of the upper and lower teeth since 2 years. On anamnesis, the patient reported that he was on hypertensive medication 10 mg amlodipine (Amlog) for two years which was stopped 1 year back. The patient was on 150 mg aspirin 6 months back and currently was taking 75 mg ecosprin and 10 mg enalapril maleate (Dilvas). The patient was advised to bring his previous medical, dental reports and physician's consent to conduct further periodontal examination and provide the best possible treatment plan. The patient had no history of smoking. The patient belonged to rural area and and belonged to a rural area and socioeconomically he belonged to lower middle class.

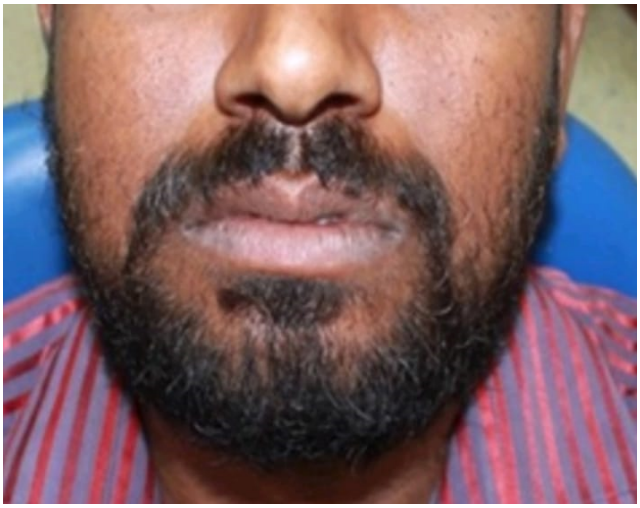

Figure 1: Extra oral image of patient showing extraoral swelling on the right side of face

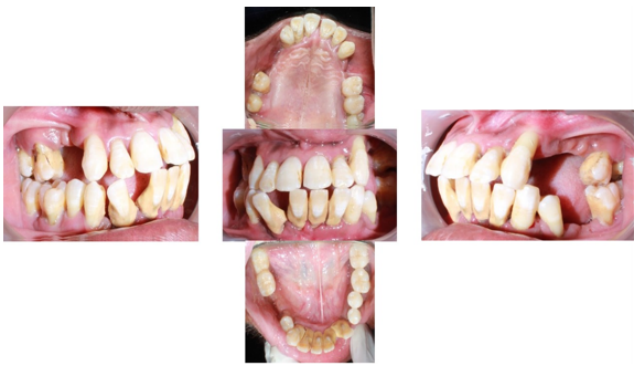

Figure 2: Intraoral view showing number of teeth present and the oral hygiene status. Teeth present 18,17,16,13,12,11,21,22,23,27,28,31,32,33,34,37,38,41,42,44,45,46,47,48 Missing 14,15,24,25,26,35,36,43.

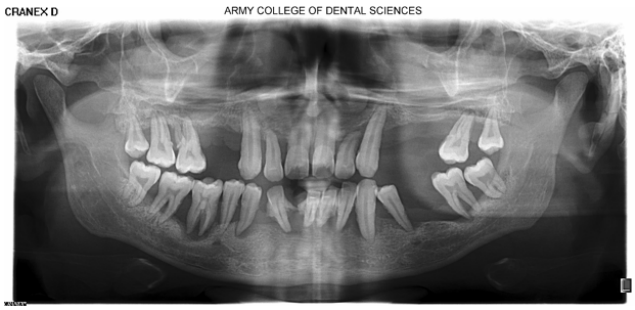

Figure 3: Orthopantomograph showing advanced bone loss irt 17,16,12, 27, 31,32,34 35,37,41,42,44,45,46,47 (the bone level at the apical third of root) and 16, 22,23, 27 (the bone level at the junction of the middle and apical third of root).

On physical examination, the patient appeared well-built and healthy, exhibiting no abnormality in gait. There was no sign of icterus or anemia. The pulse was measured as 87 beats/min. Extra-oral examination revealed an enlarged lymph node on the right side. (Figure 1) On clinical assessment, the patient was partially edentulous with missing 14,15,24,25,26,35,36,43 that were extracted previously due to loosening of teeth. The patient had generalized calculus deposits. (Figure 2) Most of the teeth were mobile with Grade III mobility irt (in relation to) 12, 46, 42, 41, 31, 32, 34, 37, Grade II mobility irt 17, 16, 22, 23, 27, 38, 45, 44, 33, 48 and Grade I mobility irt 18, 11, 21. On further questioning, the patient reported having been diagnosed with Takayasu arteritis a few years back.

The patient was instructed to undergo blood tests and radiographic examination. The patient's random blood sugar (RBS) was 115 mg/dl. Orthopantomograph (OPG) revealed generalized advanced bone loss. (Figure 3)

While going through his previous records (supplementary Diagram 1) we found that the patient was diagnosed with Takayasu Arteritis in 2017 at the age of 28 years. Based on his symptoms of chronic and intermittent fever, and asymmetric pulses present bilaterally (b/l) he was advised coronary angiogram (CAG) and Carotid Angiography which showed coronaries with carotid diffuse disease with left subclavian; 100% disease with b/l renal artery stenosis. (supplementary Diagram 2) The patient was treated with anti-hypertensive drugs and beta blockers. The patient was advised CT pulmonary angiogram (CTPA) followed by percutaneous transluminal angioplasty (PTA) in the renal artery and risk factors of hypertension and alcohol intake were considered. Doppler study reports of the left upper limb revealed circumferential wall thickening noted in the Subclavian artery, proximal axillary artery, common carotid. Doppler study of carotid and vertebral artery done in 2018, revealed features of arteritis of bilateral carotid arteries causing significant luminal narrowing. (supplementary Diagram 3)

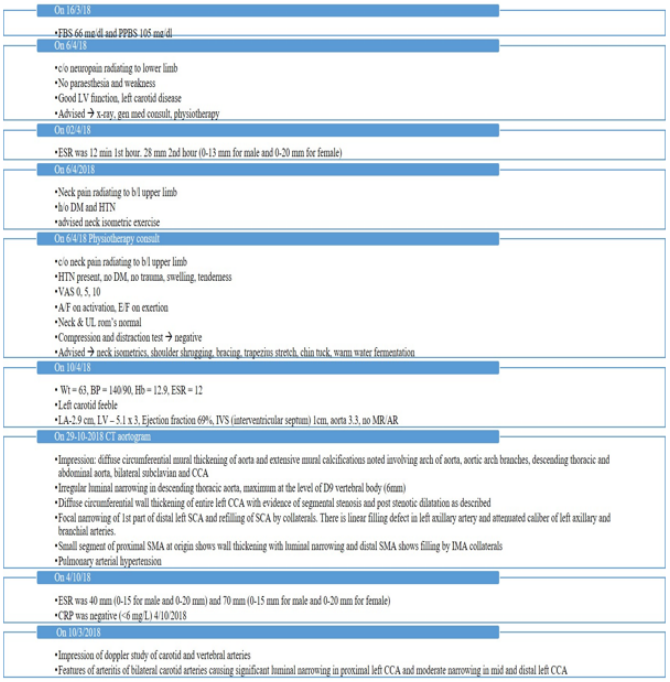

Diagram 1: Supplementary Figure 1

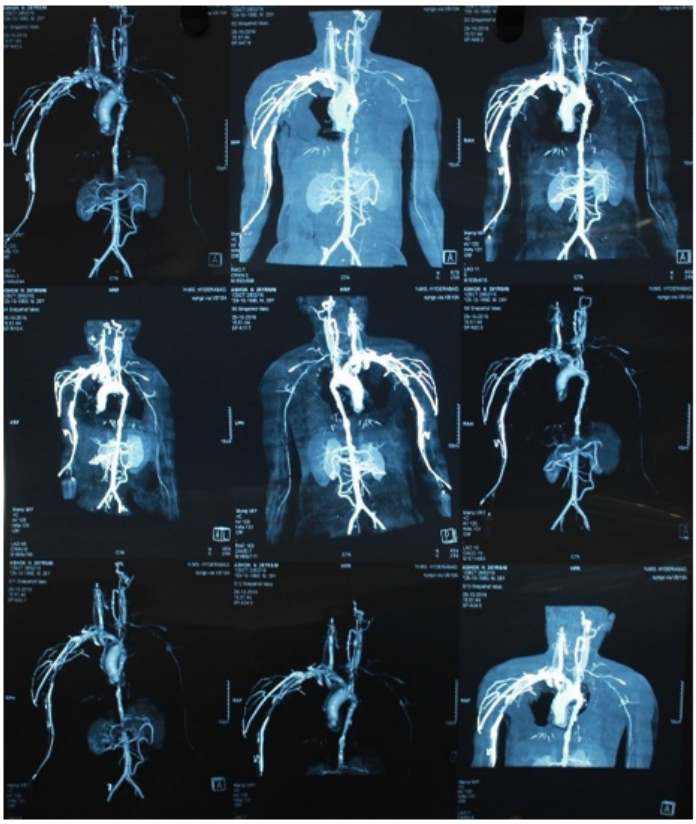

Diagram 2: Supplementary, Doppler study images of carotid and vertebral artery showing luminal narrowing

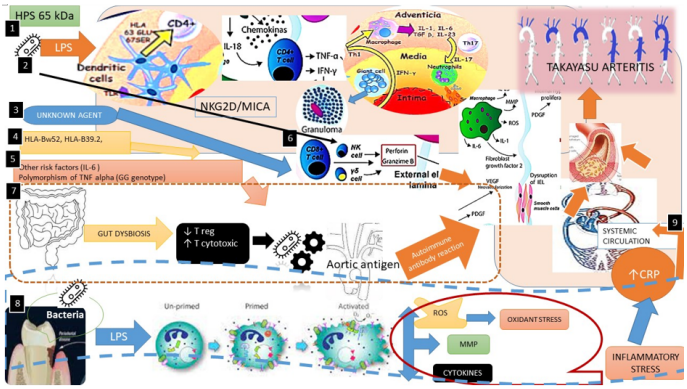

Figure 4: Pathogenesis of Takayasu arteritis and its link with other unknown and possible systems 1. HSP (Heat Shock Protein) 60 kDa present on endothelium of host is analogous to HSP kDa65 present on Mycobacterium tuberculae. This molecular mimicry triggers autoimmune response towards endothelial cells contributing to Takayasu arteritis. 2. Microbes (pathogenis/commensal) can act as antigens or lipopolysaccharide (LPS) released leads to TLR(toll like receptor) activation which is recognized by dendritic cell/antigen presenting cell, which activates T- cell and in turn B cell is activated producting autoantibodies which cross react with endothelial cell peptide which is similar to microbe. Hence, Anti-annexim-V (autoantibody) can directly cause apoptosis of endothelial cell or cause antibody dependent cytotoxicity via natural killer (NK) cell through NKG2D (natural killer activating receptor) and MICA (ligand for NKG2D) interaction. Microbes and stress can also stimulate MICA and NKG2D. 3.Unknown agents can cause autoimmune response against host endothelial cells. 4.HLA-Bw52 and HLA-B39.2 genotype also cause autoimmune reaction.5.Polymorphism of TNF alpha and specially GG genotype is associated with more production of TNF alpha in susceptible individuals increasing the inflammatory load on vasculature. 6.NKG2D activation on NK cells and T cells results in release of perforin which results in acute vascular inflammation and release of proinflammatory cytokines. 7. Dysregulation in the gut microbiota can increase the cytotoxic T cells and decrease the regulatory cells (Treg) which predisposes towards pathogenic microbiota. The microbes exhibit molecular mimicry with aortic antigens resulting in autoimmune antibody formation against aorta. 8.Periodontal microbes also secrete LPS which can lead to activation of macrophages and neutrophils to release reactive oxygen species (ROS) , pro inflammatory cytolines and matrixmetalloproteinases (MMP), resulting in periodontal tissue destruction and increasing the oxidant stress levels and inflammatory markers like C reactive protein (CRP). 9. It is a possibility that the locally produced oxidant stress and CRP levels may pass into the systemic circulation and affect the vasculature resulting in endothelial injury. 10. The TNF alpha and interferon gamma (IFNγ) act on monocyte resulting in formation of giant cell and granuloma formation. The resultant vascular inflammation results in tissue fibrosis of the intima of blood vessel. Neutrophil activation via Th17 results in vascular lesions.[10–12]

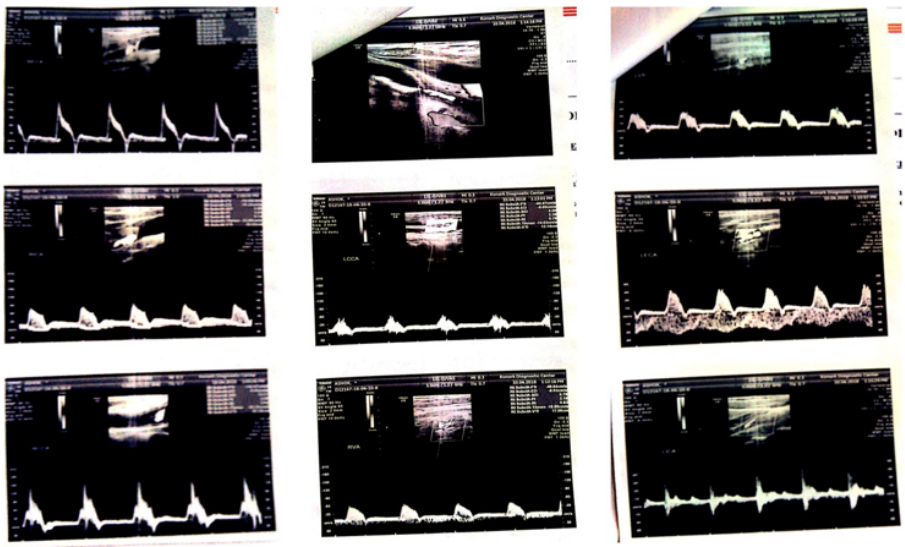

Diagram 3: Supplementary, Coronary angiogram showing thickening of aorta and subclavian artery

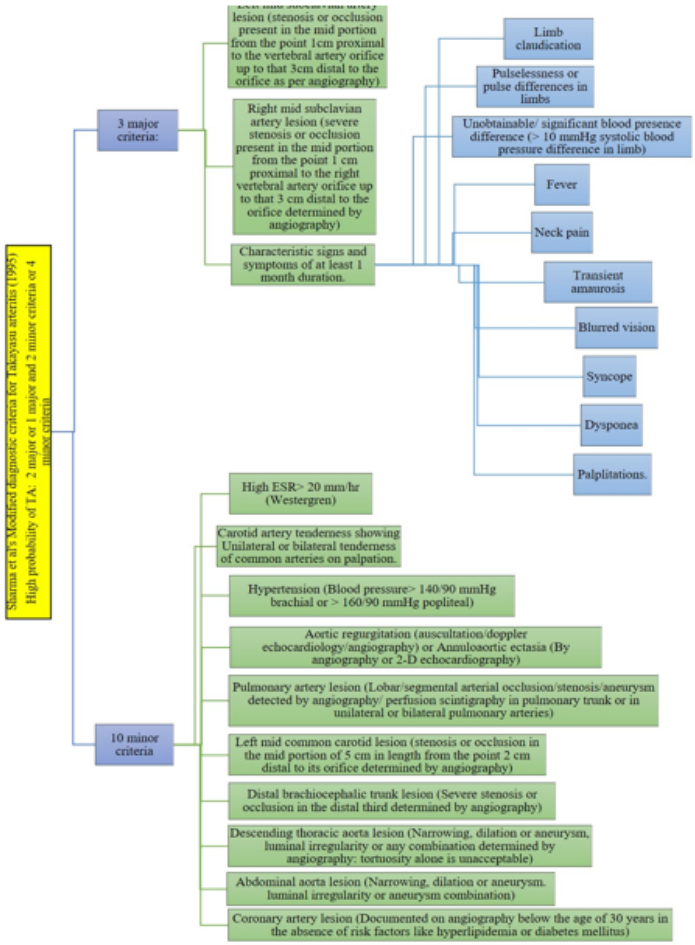

Diagram 4: Supplementary

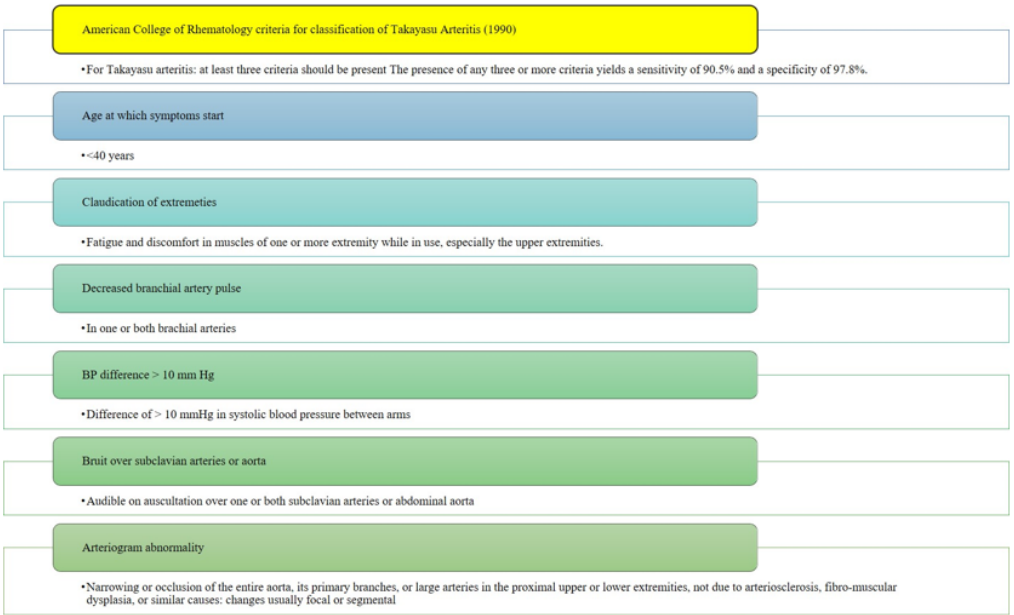

Diagram 5: Supplementary

Based on the presence of local factors (plaque and calculus), advanced destruction of the periodontal tissues, mobility of teeth, and bone loss. The patient was diagnosed with generalized chronic severe periodontitis occurring as a manifestation of systemic disease (Takayasu arteritis).

The treatment plan included extraction of hopeless teeth 17,16,12,22,23,27,31,32,33,34,37,41,42, 43,44,45,46, phase I periodontal therapy (scaling and root planning) of remaining teeth followed by maintenance visits to check if the patient can maintain oral hygiene. The missing teeth would be replaced with removable prosthesis as an interim measure followed by implant placement at a later stage.

In the present case, immediate Phase I periodontal therapy was not started, and the patient was advised to get the physician's consent regarding the same. Simultaneously, a review of the literature was carried out to know how this disease could affect the periodontal tissues and whether periodontal therapy could have any deleterious effect on the underlying medical condition of Takayasu's arteritis.

Unfortunately, the patient was lost to follow-up due to the COVID-19 pandemic. This report is to apprise dentists about the presence of this disease, the periodontal manifestations seen in such patients, possible linking mechanisms between the two diseases and guidelines to be followed for treating such patients as per the literature evidence.

3. Discussion

TA can affect ages ranging from 6 months to less than 40 or 50 years. The prevalence is seen more in Asia than in North America.[13–15] It is seen predominantly affecting the female population in the European and Asian countries, while male predominance is seen in the Indian population.[9] The male-to-female ratio (M: F) is 1:8 in adults and 1:2 in children.[6,13] In females, the disease is more common during the childbearing age of 20-30 years.

TA presents itself as acute and chronic phases and progresses through stages of constitutional symptoms of fever, weight loss, malaise, anemia, myalgia, arthralgia, and arthritis. This is followed by a vascular inflammatory phase, which includes claudication of the jaw and extremities and neurological manifestations such as headache, and seizures following transient ischemic attack or stroke. Treatment of TA is required due to the risk of developing cardiovascular complicationsfor which immunosuppressants and antihypertensives are given.[6]

Diagnosis is based on following certain established criteria given by Ishikawa (1988), 1990 criteria given by the American College of Rheumatology (ACR), and Criteria given by Sharma et al (1995). (supplementary Diagrams 3, 4 and 5) For diagnosis, there is no specific test and clinicians generally rely on signs and symptoms, clinical angiography, CT, and MRI.[6] Due to nonspecific clinical presentation diagnosis is delayed.[14] Surgical procedures should be avoided during the active phase of the disease.[16]

Takayasu arteritis patients have been shown to exhibit a bone loss in lumbar vertebrae and hip region. This hints towards a plausible relationship between inflammation, lipid metabolism, and bone metabolism.[17] Such evidence can suggest a possible influence of the systemic disease (TA) on the alveolar bone surrounding the teeth resulting in advanced bone loss as seen in the present case.

Exact etiology of disease is still unexplored and unknown. Nevertheless etiologic factors which could lead to TA include an autoimmune response, eg: HSP 65 M is common to BCG vaccine and mycobacterium tubercule; infection from TB and genetic predisposition due to HLA association. Tuberculosis as an infectious etiology has been reported in more than half of cases examined during autopsy.[18] Studies have suggested an association between HLA types A9-10, BW52, DHO, and DW12 in Japanese patients and B5 and B21 in Indian patients and TA.[9,10,13] Some have suggested the possible effect of hormones due to the susceptibility of the disease in pregnant females.

Based on the present case report, the severe bone loss and loss of attachment seen could be attributed to the initiation of the disease by periodontal pathogens associated with dental plaque combined with systemic inflammatory stress generated as a result of Takayasu arteritis. Inflammation is a key player linking periodontal disease and many chronic diseases. Thus, we hypothesize that the severe destruction seen in the present case could be attributed to the indirect influence of systemic diseases like Takayasu arteritis. The possible mechanism that could contribute to a link between periodontal and systemic disease can be seen explained in the figure based on literature review.[11,12,19]. (Figure 4).

Duque et al[6] have reported a case of Takayasu arteritis in a pediatric patient in whom dental treatments including scaling, extractions, pulpotomy, and composite restorations were performed successfully. Gupta et al[5] have reported a case of a female patient with TA, who was successfully treated for pericoronitis and space infection. In both the above case reports, extraction of teeth had to be performed as one of the dental procedures due to mobility of teeth occurring probably due to localized periodontal destruction. This finding could suggest that the severe destruction of periodontal tissues may have been exacerbated by systemic diseases such as Takayasu arteritis. Moreover, treating such patients require complete knowledge of the disease. Goa et al have reported a case of a female Takayasu arteritis patient who was diagnosed with chronic periodontitis. A decrease in the levels of Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level was observed following nonsurgical therapy and the authors hypothesized about the potential relationship between periodontitis and TA concerning inflammatory factors, bacteria, and medication.[19]

Based on supporting studies,[5,6] dental treatments in TA should be carried out preferentially in a hospital setting with strict cardiac monitoring. Antibiotic prophylaxis and steroid supplementation are recommended. Treatment should be planned during inactive phase of disease. Care has to be taken for possible interaction between steroids and any other prescribed drugs, such as anticoagulants, antihypertensives, and immunosuppressants.[6]

4. Conclusion

A relationship exists between severe chronic periodontitis and systemic diseases, which many times include disruption in inflammation, lipid metabolism, and bone metabolism as the linking pathophysiological mechanism. As complete etiology of TA is unknown and as it comes under the umbrella of diseases affecting the vasculature, it could be possible that the effect of severe periodontal infection may indirectly affect Takayasu arteritis by causing endothelial dysfunction or increasing CRP levels or vice-versa. Inflammation or autoimmune response could be the linking mechanisms. Longitudinal studies may be required to establish a plausible connection between these two entities resulting in a piece of stronger evidence for perio-systemic interlink. Another possibility is that the patient suffered chronic periodontitis which was due to indirect effect of TA on the patient's general health, related to systemic stress, but which does not affect all patients with TA.

5. Source of Funding

None.

6. Conflict of Interest

None.

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