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Abstract

Weak oral bacteria such as periodontal bacteria have been observed in various arterial and venous lesions with epidemiological data reported prior to the discovery of bacterial invasion into vessels. Rich lymph vessels easily bring the bacteria from the mouth to the neck and the venous angle, which is directly open to the blood vessels. Periodontal bacteria travel within platelets. Periodontal bacteria, especially P. gingivalis aggregates platelets and forms thrombus. At the same time, secretions such as serotonin, various cytokines, and adhesion factors also appear in the blood. The characteristic of the arterial lesions are dependent on the age of the

discrepancy between vascular diseases. We were able to emphasize the collaboration study in this study with dentists and vascular surgeons.

Keywords: Dentist; Vascular surgeon; Weak oral bacteria; Buerger disease; Transportation of oral bacteria; Oral care

Introduction

Since 1999, various weak oral bacteria have been identi ed in atherosclerotic lesions [1]. Among these bacteria, Chlamydia pneumoniae which resides in the mouth, pharynx, or bronchus, has been thoroughly investigated and con rmed to be transported to vessel walls by monocytes [2]. is invasion mechanism appears to be a factor in the development of atherosclerosis. Additionally, cytomegalovirus can be absorbed from the oral cavity resulting in opportunistic infections. Recently, the so called in ammatory abdominal aortic aneurismal walls revealed the presence of cytomegalovirus [3]. Helicobacter pylori is well-known bacteria that resides in the stomach and may also appear in the oral cavity. is one was also identi ed in vessel walls. Over the past 13 years, the periodontal bacteria group that includes several species has been shown within vessel walls [4-6]. We, dentists and surgeons, started a study about periodontal bacteria invading the vessels in which we clari ed the relationship between periodontal bacteria and vascular diseases, especially Buerger disease, which is still major vascular disease in south and western Asia [7].

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Plasma (PRP) to stimulate good wound healing. When we accidentally added periodontal bacteria (P. gingivalis) to the sample and saw the mixed uid through a stereoscopic microscope, we could nd active movement. A er examining the sample by electron microscopy, we observed that periodontal bacteria (P. gingivalis) were engulfed by platelets and morphologically there were no change observed in the bacteria for one hour. Additionally, platelet aggregation was also observed. ese observations con rmed that P. gingivalis bacteria aggregate strongly in the platelets, and they don't die (Figure 1) [12].

P. gingivalis induced platelet aggregation reached the maximum in a few minutes and the mass became more than 20 microns larger than the size of the small artery in vitro. Additionally, as periodontitis itself expresses in ammatory substances such as IL-6, and TNF , it should be considered a systemic disorder [13]. A strong relationship with diabetes mellitus control is coming from the abovementioned mechanism. Fortunately, for our healthcare is now associated with good social manners.

Serum Bacterial Antibody Titer Changes in Periodontal Disease and Buerger Disease

Chen et al. [14] who are dentists, reported that the antibody titer for periodontal bacteria is signi cantly elevated in Buerger disease patients, recon rming that Buerger disease patients have very poor periodontal conditions [5] (Figure 2). In Buerger disease the antibody titers may actually be changing related to the severity of periodontitis.

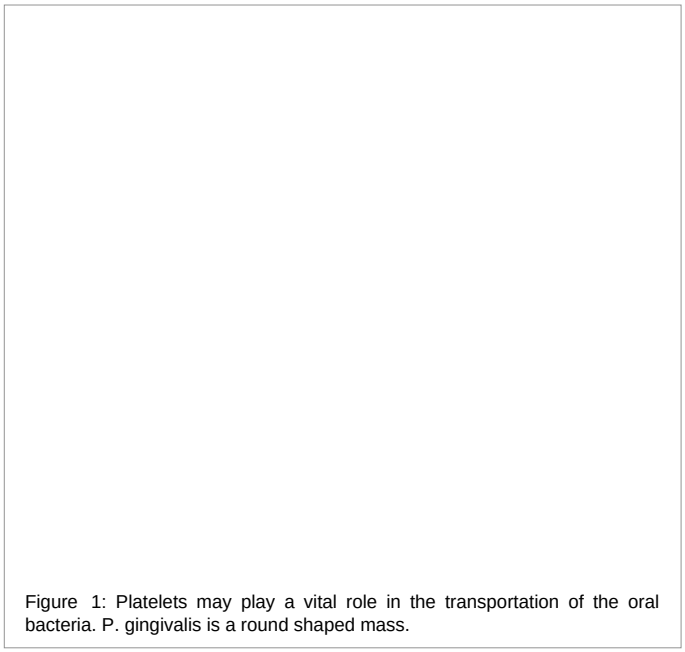

Figure 1: Platelets may play a vital role in the transportation of the oral bacteria. P. gingivalis is a round shaped mass.

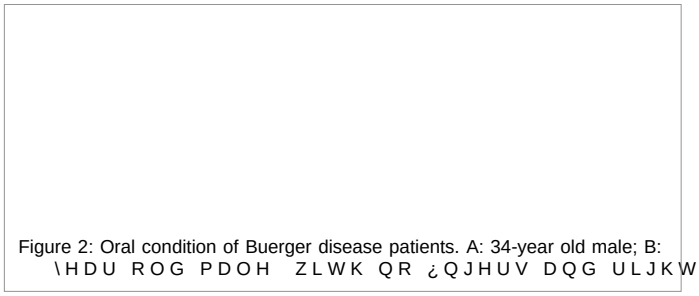

Figure 2: Oral condition of Buerger disease patients. A: 34-year old male; B: \HDU ROG PDOH ZLWK QR ¿QJHUV DQG ULJKW EHORZ NQHH DPSXWDWLRQ

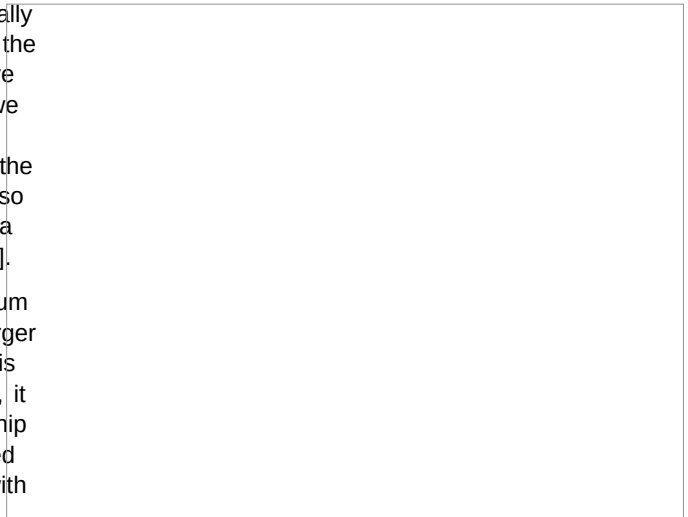

Figure 3: Hypothesis of Buerger disease development. This is different from the developing mechanism of atherosclerosis. From the literature 17.

E ective antibiotic treatment against the bacteria can decrease the titer level rapidly.

Do All Persons with Serious Periodontitis Develop Buerger Disease or another Vascular Arterial Disorder?

Recent studies on Buerger disease have shown a speci c HLA locus and infection susceptibility for basilar bacteria, such as periodontal bacteria [15]. Varicose veins seem to occur in mothers and daughters. Interestingly approximately 50% of varicose veins contain periodontal bacterial DNA by our study suggesting that pregnancy may be linked to varicosity development when the woman su ers from periodontitis during pregnancy. However it is questionable that the periodontal bacteria may transmit from mothers to daughters.

Hypothesis and Future Views of Buerger Disease Development

Pathogens in Buerger disease are likely to be mainly periodontal bacteria. Among periodontal bacteria, T. denticola and P. gingivalis are main member of red complex, bacterial group characteristic of periodontitis patients. P. gingivalsis is inevitably moved as an initiator of platelet aggregation, and from the venous angle of the neck, the bacteria group can enter the blood stream and stimulate platelet aggregation a er uptake into platelets. It is suggested that aggregation reaches a maximum level when the platelet thrombi passes through the lung, a er which the thrombi starts to move in the arterial blood stream. When the arterial wall is young but spastic from cigarette smoking, the platelet thrombus containing the oral bacteria do not adhere to the arterial wall but form a small arterial embolism. It is suggested that the digital arterial obstruction in Buerger disease patient angiography may be initial ndings. is change will grow to the proximal arterial regions due to packing. Microorganisms that pass through capillaries can be caught at the venous valves, resulting in phlebitis migrans or deep vein thrombosis formation in the extremities. e literature shows small arterial changes are very common all over the body, but are symptomatic only in the extremities (Figure 3). is mechanism is speci c in Buerger disease and considerably di erent from atherosclerotic disease including aneurysms [16,17].

References

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