The 7th Temu Ilmiah Nasional And The 4th Join Scientic Meeting In Dentistry

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FACULTY OF DENTAL MEDICINE

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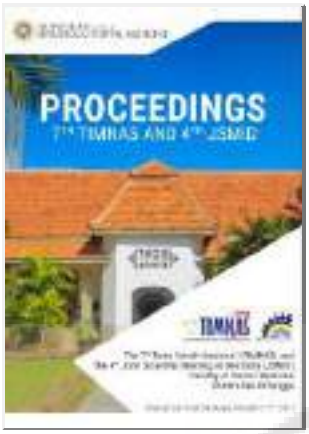
Holistic Oral Health Achievement Through Basic Medical and Clinical Science

1st Announcement



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Foreword: Come and join us in the 7thTemu Ilmiah Nasional (TIMNAS) - the 4th Joint Scientific Meeting in Dentistry (JSMiD)! We extend you a warm welcome in 2017, to the 7thTemu Ilmiah Nasional (TIMNAS) - the 4th Joint Scientific Meeting in Dentistry (JSMID) held in Shangri-La Hotel 5th – 7th October 2017. The 7th TIMNAS – 4th JSMiD is a global initiative of the internationally renowned in all aspect of Dentistry sharing and exchange of technical know-how among dental professionals and conducted with the main purpose to contribute in shaping the world of Dentistry. This event also has a mission to provide a stage for researchers and clinicians that are at the cu ng edge of life science and a clinical innovation whereby they can share their work and discuss its future application potential in a non-conventional se ng. Dental education in Indonesia has entering a new paradigm. The education is conducted in concerning with a human health. It stresses prevention over intervention. It **(More)**

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The Relationship Between Oral Cavity Infections and the Occurance of Stroke

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Keywords: Oral Cavity, Infection, Stroke.

Abstract: One of the main factors causing stroke is atherosclerosis i.e. narrowing of the arteries due to deposition of cholesterol and other substances or substances called atherosclerotic plaques. Atherosclerosis can be caused also by infection from odontogen and its supportingtissue. *P. gingivalis* bacteria are often associated with a risk factor for atherosclerosis. Bacteria along with its products in the form of exotoxin and endotoxin enter into the systemic blood circulation and stimulate blood clot to the brain, causing a stroke. Infection in the tooth root and its supportingtissue involves a lot of bacteria and microorganisms, infection is very close to the blood vessels, toxins, free radicals, bacteria themself, a cytokine releasedtogether with inflammation entering into the bloodstream and can cause inflammation of plaque in blood vessels, plaques block blood vessels to the brain causing a stroke. **Purpose:** Analysis of the mechanism of stroke caused by infection odontogen and its supportingtissue. **Review:** Bacterial products in the form of exotoxin and endotoxin, free radicals, cytokines enter into the systemic circulation and stimulates the occurrence of thrombogenesis by inducing platelet aggregation and increasing blood clotting factors. Bacteria may also enter into endothelium and stimulate inflammatory processes in the arteries, inflammatory stimulation secretes a specific enzyme i.e. metalloproteinase which can cause plaque rupture and be a risk factor for stroke. **Conclusion:** There is relationship between oral cavity infection and stroke. Poor oral health becomes one of risk factor for atherosclerosis. Atherosclerosis is one of the main factors triggering the occurrence of stroke. Some pathogenic bacteria causing chronic inflammation have been considered as the basic mechanisms of atherosclerosis, inflammation plays an important role in the development of atherosclerosis.

1 INTRODUCTION

Stroke is a non-contagious disease that causes deaths in urban areas. Approximately 28.5% of stroke patients die and the rests are paralyzed. 15% of the paralyzed patients could be totally recovered. Stroke is a brain nerve disorder caused by damage of the blood vessels in the brain that occurs within about 24 hours or more. Stroke occurs because of inhibition of blood supply to the brain caused by a blood clot (atherosclerosis) or due to rupture of blood vessels in the brain leading to the death of brain tissue. Stroke may be ischemic or bleeding (hemorrhagic). In ischemic stroke, the flow of blood to the brain stops because of atherosclerosis or blood clots blocking blood vessels. In hemorrhagic stroke, blood vessels rupture and inhibit normal blood flow in body, penetrating into an area in the brain and damage it [.1,2.3.19]

Atherosclerosis is an aggressive disease that involves large-sized muscular arteries and large elastic arteries continued to microcirculation consisting of a central core with a large central core of which middle core experiences necrosis. Inflamed microcirculation prone to clogging (thrombosis) because of the sticking plaque. The lysed cells contain of cholesterol esters, lipid-containing foam cells as well as fibrins and fibrinogen on the surface of the blood vessel walls [.4.5.6]

Oral cavity and teeth can be a place bacteria and other microorganisms that may spread to other organs in human body. Gingivitis and periodontitis have been proven as risk factors for systemic diseases, especially heart disease and stroke. The relationships between periodontal disease and stroke is the release of exotoxin and endotoxin bacteria in periodontal tissue. Proteolytic enzymes affect the immunologic response. The release of inflammatory mediators is one of the risk factors for atherosclerosis. The damage of endothelium cells,

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vascular smooth muscle migration, and matrix protein tissues are factors associated with atherosclerosis.[7,8]

Periodontal disease directly contributes to atherosclerosis and blockage of blood vessels (thromboembolic) i.e. by systemic vascular thickening on LPS and inflammatory cytokines. The released LPS will bind LBP (LPS-binding protein) i.e. high-affinity protein in the plasma protein that would be unfavorable to CD14 receptor in endothelium, soluble monocytes and macrophages will activate cellularly, thereby causing the release of cytokin.[9]

Pathogenic bacteria in the periodontal tissue are detected in carotid plaque and play a role in atherogenesis by damaging endothelium and stimulating a inflammatory process of large artery. Periodontal tissue bacteria can also stimulate thrombogenesis through aggregation and increase blood clotting factors.[10]

2 LITERATURE REVIEW

2.1 Stroke

Stroke is a sudden, quick disorder of the brain's anatomical function because of bleeding in the brain. According to the World Health Organization (WHO), stroke is a rapidly growing clinical sign of focal or global brain dysfunction with the symptoms that last for 24 hours or more that may cause death, with no cause other than vascular. Stroke is a syndrome marked quickly i.e. a functional disorder of the focal and global brain lasting more than 24 hours (leading to death, unless there is surgical intervention) that is not caused by any causes other than vascular.[11]

Stroke includes ischemic stroke (due to brain infarction) and hemorrhage (non traumatic intracerebral bleeding). Intra ventricular hemorrhage and some cases of subarachnoid hemorrhage. Stroke is catagorized as cerebrovascular disease (brain blood vessels) characterized by brain tissue deaths due to reduced blood flow and oxygen into the brain. Reduced blood flow and oxygen can be caused by blockage, narrowing or the rupture of blood vessels.[12]

2.1 Pathology of Stroke

Stroke infarction is caused by lack of blood flow to the brain. Normal blood flow to the brain is 58 ml / 100 g of brain tissue per minute, if coming down to 18 ml / 100 g of brain tissue per minute, electrical activity of neurons will be halted even though the cell structure is still good, clinical symptoms are still reversible. If cerebral blood flow down to <10 ml/100g of brain tissue per minute, it will trigger a series of irreversible biochemical changes of cell and membrane forming infarction region.[1] Approximately 10% of strokes are caused by intracerebral hemorrhage, hypertension (especially the uncontrolled hypertension). Another cause is the rupture of aneurysms, arteriovenous malformation, angioma cavernosa, alcoholism, blood dyscrasias, anticoagulant therapy and subarachnoid amyloid. Subarachnoid bleeding, most cases are caused by the rupture of aneurysm in the branching of large arteries. Another cause is malformation of the venous arteries or tumors.[11]

One of the main factors causing stroke is atherosclerosis which is a condition of narrowing arteries due to deposition of cholesterol and other substances including immune cells and fibroblasts referred as atherosclerotic plaque. It can rupture and cause a thrombus or blood clot migrating to artery blockage. It may result in myocardial infarction or stroke.[13]

2.2 Atherosclerosis

Atherosclerosis is a disease caused by a progressive inflammatory response of the blood vessels (large and medium arteries) marked by a deposit of collagen, fat, cholesterol, disposed products of cells and calcium along with the proliferation of myocytes leading to thickening and hardening of the artery walls resulting in stiffness and fragility of arteries.[15] Atherosclerosis is affected by high cholesterol levels (LDL), smoking, high blood pressure, diabetes mellitus, lack of physical activities, high levels of blood homocysteine, fibrinogen, lipoproteins, some bacteria causing infection or transformation of myocytes or endothelial will trigger atherosclerosis.[13]

Atherosclerosis develops as a vascular wall reacts to increased lipid filtration and blood plasma proteins. While on thrombogenic, atherosclerosis occurs as a result of recurrent episodes of thrombosis resulting in spot formation. Atherosclerosis begins during an injury to the endothelium as a result of various risk factors with various intensity. One of the main causes of endothelial injury is high LDL plasma. LDL will be oxidized into LDL- oks that are easy to stick and

accumulate on the walls of blood vessels, and become lipid deposits. This accummulation causes the endothelial lesion. In injured endothelium will be hyperpermeable indicated by the occurrence of various processes of exudation and infiltration into blood vessel layers due to increased activity against lipoprotein, leukocytes, platelets and other plasma content. Injured endothelium also has more procoagulant compared to anticoagulants, and also experiences acceleration of leukocyte adhesion molecules e.g. L-selectin, integrin, platelet-endotheleal-cell adhesion molecule (PECAM-1) and endothelial adhesion molecules e.g. E-selectins, P-selectins, intraceluler cell adhesion molecule (ICAM-) and vascular-cell adhesion molecule (VCAM-1). This situation causes macromolecules more easily stuck to the blood vessel wall resulting in injury to the endothelium.[14]

Atherosclerosis can also be caused by an infection originating from odontogen. Anaerobic bacteria are often associated with risk factors for atherosclerosis, bacteria and their products in the form of exotoxins and endotoxins enter to the systemic circulation and stimulate the occurrence of trobogenesis dam damage endothelium.[15]

2.3 Relationship of Odontogenic Infection With Stroke Occurrence

Odontogenic infections can come from three paths i.e. periapical pathways, periodontal pathways, pericoronal pathways. Odontogenic infections begin from tooth surfaces i.e. caries that becomes infection in the dental apex expanding to the surrounding tissue. It spreads through contamination via haematogen and lymphogen. Tooth and oral tissue is the center of infection, there are 3 pathways of infection in the oral cavity i.e. metastatic infections of the oral cavity as a result of bacteremia. This infection is due to dental procedures and dental infections. Oral cavity infection may cause bacteria dwell in an organ in the body. Metastatic wound infections is caused by the effects of bacterial toxins circulating. Metastatic inflammation occurs by the presence of dissolved antigens in the bloodstream reacting with circulating and forming an immunocomplex macromolecule complex allowing various acute and chronic reactions in the colonized bacterial region.[15]

Periodontal infection is potentially systemic leading to hypercoagulation and increased blood viscosity. In patients with periodontal disease, it is frequently found increased fibrinogen and white blood cells. Individuals with poor oral hygiene significantly increase in coagulation factor VIII *von willebrand factor*, this may increase risk for thrombus formation. Periodontal infection may also increase blood viscosity and thrombogenesis, this condition increases the risk for peripheral and central vascular disease.[16]

Gram-negative bacteria and lipopolysaccharides in the oral cavity cause infiltration of inflammatory cells into the artery wall, arterial smooth muscle proliferation and intravascular coagulation. These changes are identical to those observed in atheromatosis. Periodontal disease leads to chronic systemic infection, the bacterimia condition initiates the body's response by affecting coagulation, endothelial and vascular wall integrity, platelet function leading to atherogenic changes and the occurrence of thromboembolism.[15]

3 DISCUSSION

Periodontal infection affects the occurrence of atherosclerosis. The formation of atherosclerosis begins with circulating monocytes sticking to the endothelium mediated by multiple adhesion molecules on the surface of endothelial cells i.e. intercellular adhesion molecule-1 (ICAM-1) in endothelial leukocyte adhesion molecule (ECAM-1) and vascular cell adhesion molecule -1 (VCAM-1). Molecule adhesion is regulated by a number of factors i.e. the product of bacterial lipopolysaccharide and cytokin. After prostaglandins bind to endothelium, subsequently penetrating in the deeper under the intimal layer, enlarging monocytes and forming atheromatous plaque.[18]

Atheromatous plaque formation and thickening of blood vessel walls causes narrowing of blood vessel lumen, resulting in reduced blood flow. Trombosis often occurs after rupture of atheromatous plaque, an activation of platelets and the coagulation pathway. The accumulation of platelets and fibrins may block blood vessels and lead to ischemia e.g. angina myocardial infection. A research on animals shows that gram-negative bacteria and lipopolysaccharide cause infiltration of inflammatory cells into the artery wall, intravascular arterial smooth muscle proliferation. This change is identical with atheromatosis occurance. Chronic periodontal diseases cause systemic infections, bacteremia initiates the body's response by affecting coagulation, endothelium, and the integrity of blood vessel walls, platelet function. All of those cause atherogenic changes and thromboembolic occurence.[4,15]

The microorganisms in the oral cavity and its products may spread systemically through the circulatory system, the pathogenic bacteria in the oral cavity can be found in the artery. It roles as a direct mediator of vascular disease e.g. hypercoagulation, atherosclerosis progression, or both. Undirect mediator, on the atherosclerosis found inflammatory components and epidemiological evidence of an increased level of systemic inflammation. Another mechanism that connects oral infections with ischemic is the target of antibodies to the bacteria. Bacterial pathogens in periodontal tissues can be detected in the carotid plaque and play roles in atherogenesis by damaging the endothelium and stimulating the inflammatory process of major arteries.[7]

On the damage of endothelium, the cells showed endothelial adhesion molecules called vascular cell adhesion molecules (VCAM) and intracelulermoleculer adhesion (ICAM) that cause leukocytes stickable. Leukocytes sticking is also supported by selectins and integrins. If leukocyte sticking is deposited more, it will occur atheroma accumulation in the form of lipids and cause the production of some chemical mediators and growth factors which then causes the stimulation of the release of monocytes and macrophages. Chemical mediators migrate into the muscle cells that will respond to the occurrence of inflammatory stimuli by secreting metalloproteinases that may break down the fibrous capsule along with cholesterol plaques and cause rupture of the plaque that will increase stroke risk factor for myocardial infarction.[4]

4 CONCLUSIONS

There is a relationship between oral infections and stroke. Poor oral health is one of the major risk factors for atherosclerosis. Atherosclerosis is one of the major factors causing stroke. Some pathogenic bacteria that cause chronic inflammation have been considered as a basic mechanism of atherosclerosis, inflammation has an important role in the development of atherosclerosis.

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