Low bone mineral density as predictor factor for loss of teeth

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ABSTRAK
Rendahnya densitas mineral tulang merupakan kondisi klinis pada penderita osteoporosis. Osteoporosis adalah penyakit degeneratif metabolik yang ditandai dengan pengurangan massa tulang dan arsitek mikro tulang sehingga berisiko terjadi fraktur. Prevalensi terjadinya osteoporosis meningkat sejalan dengan peningkatan usia harapan hidup dan pertambahan jumlah penduduk wanita. Penulisan makalah ini bertujuan untuk mengkaji pengaruh penurunan densitas mineral tulang terhadap kehilangan gigi sehingga diharapkan ada pencegahan dini terhadap kehilangan gigi pada penderita osteoporosis maupun osteopeni. Metode yang digunakan untuk menilai penurunan densitas mineral tulang ialah dengan menggunakan *dual energy x-ray absorptiometry (DXA)* sedangkan kehilangan gigi dapat dilihat secara klinis dalam rongga mulut dan pemeriksaan radiologi dengan teknik panoramik. Kehilangan gigi merupakan kondisi akhir yang harus dialami oleh gigi yang mengalami penyakit periodontal yaitu adanya proses inflamasi secara progresif, hilangnya perlekatan jaringan periodontal dan menurunnya tulang alveolar pendukung gigi. Tingginya tingkat penurunan densitas mineral tulang dapat memnyebabkan kehilangan gigi lebih cepat oleh karena defisiensi hormon pada wanita atau pria usia lanjut. Kondisi ini mempengaruhi aktivitas osteoklas dan osteoblas sehingga terjadi *abnormality bone turnoever* pada seluruh tubuh termasuk pada tulang alveolar. Hal ini menunjukkan bahwa adanya hubungan antara penurunan densitas mineral tulang dengan kehilangan gigi pada manula. Kesimpulannya ialah adanya defisiensi hormon dalam proses interaksi osteoblas dan osteoklas menyebabkan terjadinya penurunan densitas mineral tulang pada tubuh termasuk pada rahang dan menyebabkan kehilangan gigi secara cepat.
Kata kunci: densitas mineral tulang, kehilangangigi, defisiensi hormon

ABSTRACT
Low bone mineral density is a clinical condition in patients with osteoporosis. Osteoporosis is a degenerative metabolic disease characterized by reduced bone mass and bone microarchitecture; so it risks for fracture. The prevalence of osteoporosis increases with the increasing life expectancy and preponderance of females. This paper aims to determine the influence of decreased bone mineral density of the missing teeth terhadap tooth loss which contributes to early prevention of tooth loss in people with osteoporosis and osteopenic. The method used to assess bone mineral density reduction is by using dual energy x-ray absorptiometry (DXA) while the loss of teeth can be seen by clinical and radiological examination of the oral cavity with a panoramic technique. Tooth loss is a condition that must be experienced by the end of dental periodontal disease which is present in a progressive inflammatory process, periodontal attachment loss and decreased alveolar bone supporting the teeth. The high rate of decline in bone mineral density can lead to faster loss of teeth because of hormone deficiency in elderly women or men. This condition affects the activity of osteoclast and osteoblast, causing abnormalities bone turnover on the whole body including the alveolar bone and showed relationship between decreased of bone mineral density with tooth loss in the elderly. The conclusion is the presence of hormone deficiency in the process of interaction osteoblast and osteoclast leads to decreased bone mineral density in elderly include in jaw bone and it causes rapid tooth loss.
Key word: *bone mineral density, tooth loss, hormone deficiency*

INTRODUTION

Oral health plays an important role in general health and quality of life for elderly. The population of elderly increases with increasing number of life expectancy in the elderly population.[1] In 2013, amount of elderly (age above 60 years) reached 554 million which is estimated to increase to 3 times in 2050, namely to 1,6 billion (21% of total world population).[2]

It is reported reported tooth loss is experienced by the elderly when compared with other age groups. According to WHO the prevalence of tooth loss for the Asian continent is India 19% (age 65-74 years), Srilanka 37% (age $\geq$ 65 years), Thailand 16% (age 65-74 years), and for Indonesia is 24 % (age $\geq$ 65 years). Tooth loss will affect the masticatory system and aesthetic for elderly groups. The number and

condition of teeth will affect selection of food and diet which can impact on elderly's quality of life.[3]

One cause of tooth loss is periodontitis which is an inflammatory process in the periodontal tissues including the gingiva, alveolar bone, cementum, and periodontal ligament. This condition occurs due to inflammation of the supporting tissues of the teeth, caused by specific microorganisms, and may further cause progressive damage to the periodontal ligament and alveolar bone with pocket formation, recession or both. This condition can be exacerbated by systemic diseases of the elderly, one of which is osteoporosis.[4]

Osteoporosis is a systemic bone disease which is characterized by bone tissue microarchitecture changes, low bone mass and will continue with the fragility and fractures. The disease is becoming one of the major problems in oral health in Indonesia.[5]

Several studies have described a relationship between decreases in bone minerals density of osteoporosis with tooth loss due to periodontitis. There are similaritiseens in the pathogenesis of osteoporosis and of periodontitis, which is an increase of systemic regulation, the production of *interleukin-1* (IL-1), *tumor necrotisis factor-alpha* (TNF-α), and IL-6 stimulates osteoclast activity and increases the rate of turn over thus causing a decrease in bone mineral density and bone osteoporosis.[5]

Osteoporosis is more common in women than in men. It is due to the influence of hormone estrogen which is expected to start to decline from the age of 35 years while in the male hormone testosterone is down at age of 65 years. According to world statistics, 1 in 3 women is vulnerable to osteoporosis.[5,6]

Passo et al found that the influence of osteoporotic/ osteopenic in the early osteoporosis is against periodontitis disease progression in women with menopause. Research result showed that women with osteoporosis or osteopenia have a 2-fold opportunity to experience the disease periodontitis compared with women with normal bone mineral density. It also proved the hypothesis that estrogen deficiency during menopause is linked to bone mineral density and contributes to the imbalance activator level of nuclear factor receptor-β ligant (RANKL), activator of nuclear factor receptor-β (RANK), osteoprotegerin (OPG) on the structure of periodontal tissues through increased stimulation of serum inflammatory mediators (IL-1, IL-6, TNF-α), so the impact on the network attachment loss and alveolar bone resorption. The level of periodontal tissue destruction depends on the balance of inflammatory mediators and protective destructive inflammatory mediators. When periodontal bacteria invade periodontal tissues causing inflammation then the inflammatory response of the body's defense will increase. Based on labrolatorium test individuals in the inflammatory response caused by genetic signals that affect the process of inflammatory mediators in bacterial lipopolysaccharide (LPS).[7]

Lipopolysaccharide produces antibodies as a defense mechanism, and also activates osteoclasts, causing bone loss. T-lymphocyte produces RANK- L that can play a role in osteoclast activation and bone resorption caused destruction OPG inhibits the release of inflammatory and tissue metaloprotein.[8]

The above explanation suggests that osteoporosis affects the condition of the periodontal tissues causing tooth loss. This includes alveolar bone resorption and periodontal attachment loss. This situation is related to the fact that systemic hormone deficiency due to deterioration in the elderly will affect the process of bone remodeling, causing rapid tooth loss, periodontal ligament attachment loss and alveolar bone.

Periodontal tissues

Periodontal tissues refer to a functional system tissue that surrounds and attaches the tooth to the jaw bone, thus supporting the teeth from its socket. Periodontal tissue is composed of gingiva, alveolar bone, periodontal ligament, and cementum.[9]

Gingiva is a part of the mucous membranes in the mouth that are attached to the alveolar bone and the neck cover and surround the teeth. On the surface of the oral cavity, gingiva marginal gingiva extends from the top to the mucogingival linkage. Gingiva is often used as an indicator of periodontal disease. The clinical features of the inflammed gingiva are changing of color from pink corel, including the presence of ethnic factors, but this varies from person to person; an increase in size of gingival; loss of texture on the surface of the gingiva stipling; and the tendency of bleeding on probing or palpation.[9,10]

Periodontal ligament is the connective tissue that fills the space between the tooth surfaces with wall socket. The function of the periodontal ligament is to maintain the biological activity of cementum and bone, cleaning nutrition and waste products through the blood and lymph flow, tactile pressure and deliver the sensation of pain by the trigeminal nerve. Most diseases of the periodontal ligament, if not treated properly, will ultimately lead to tooth loss.[9,10]

Periodontal ligament is derived from the *dental sac*, a circular fibrous layers of connective tissue surrounding the tooth germ. When developing the eruption, the *dental sac* of loose connective tissue will be differentiated into three layers', the outer layer is close to the bone, cementum layers in all cementum side, and a layer made up of irregular fibers. The main fibers of the ligament is derived from intermediate

layer which is thickened and arranged according to the needs of the function when the teeth to be occluded.[9]

Cementum is a thin layer of calsification tissue and covers the surface of the tooth root. Dentin and cementum are bordered by email and the periodontal ligament. The structure has many similarities with the bone structure, the content of the organic matter is 45-50%. Its function is to bind the tooth to the alveolar bone, namely the presence of periodontal ligentum main fiber embedded in sementum.[9,10]

Alveolar bone is a part of the maxilla and mandible that forms and supports the tooth socket. Alveolar bone is anatomically divided into two parts, namely alveolar proprium and supporting alveolar bone. Supporting alveolar bone consists of two parts, the bones that make up the puck oral compacta located on the vestibular and bone spongius, located between the cortical plate and alveolar proprium.[9]

Pathogenesis of periodontitis

Periodontal disease is an inflammatory process that lasts for a certain period with the occurrence of periodontal attachment loss, which may result in a loss of teeth.[8]

Inflammatory process in the periodontal tissues occurs due to inflammation of the tissues supporting the teeth. Biofilm on the tooth surface, known as plaque, grow and develop within a period of several weeks. The initial growth is the presence of aerobic bacteria at supragingival area. Furthermore, the flora changes the shape of gram-positive into negative and aerobic facultative into anaerobes. Subgingival biofilm growth takes more than 12 weeks.[8]

Inflammatory response to periodontal disease includes the activation of leukocytes, neutrophils, T-lymphocytes, plasma, the release of antibodies, LPS and chemical inflammatory mediators that include cytokines, chemokines, and C-RP. LPS is present in the cell wall of gram-negative bacteria and act as the strongest stimulation in the host response complex.[8]

The earliest sign of an increasing presence of neutrophils is followed by the release of cytokines by nutrophil and macrophages. The release of chemical mediators TNF-α attacks PG and IL-1. Inflammatory process affects the stimulation of fibroblasts by IL-1 and the secretion of matrix metalloprotein (MMP), the most prominent collagen, with PMN neutrophils. MMPs play a role in increasing activity of collagen release. TNF-α fully responsible for the increase in osteoclast activity that can lead to fractures. MMP also activates cytokines and the chemokines, exacerbation destructive bones process.[8]

Lipopolysaccharide produces anti-bodies as a defense mechanism, and also activates osteoclasts that cause bone loss. T-lymphocytes produces RANK-L that can play a role in osteoclast activation and bone resorption. Media destruction OPG inhibits the release of inflammatory and tissue metaloprotein.[8]

Initiation and progressive periodontal disease depends on the presence of pathogenic bacteria, periodontal tissue response, and risk factors. The risk factors are linked to systemic disease factors in the body, which include extrinsic factors, intrinsic factors, and factors lokal.[8]

Extrinsic factors which include smoking, drinking alcohol, are stressed by the burden of thinking, socioeconomic status, and the status of systemic disease. Instrisic factors that may affect periodontal disorder is oral hygiene, gender, ethnicity, and age. Local factors include the presence of certain lesions in the marginal gingiva, overhanging fillings, fillings of damage, and interdental areas where food can be impaction.[8]

Hormone deficiency and osteoporosis

The occurrence of osteoporosis due to cell number and activity of osteoclasts exceeds the amount and activity of osteoblats cells (bone forming cells). This is due to a deficiency of the hormone estrogen testosteron and cytokines as well as the factors that lead to cell differentiation osteoclasts.[11]

Estrogen is a steroid sex hormone that plays an important role in bone metabolism, but it can affect the activity of osteoblasts and osteoclasts, as well as maintain the balance of the work of both cells through paracrine regulation by cell production osteoblast.[11]

In normal circumstances estrogen in circulation reached osteoblasts, and moved through a receptor that is present in the cell cytosol. This will result in a decrease in the secretion of cytokines such as IL-1, IL-6, and TNF-α. The function of cytokines in bone resorption. On the other hand estrogen increases the secretion of transforming growth factor β (TGF-β), which is one of the growth factors. This growth factor is a mediator attractive target cells of estrogen to release several growth factors and cytokines as mentioned above, even if the growth factor indirectly or directly also affect osteoclasts.[11]

The differentiation and activity of estrogen suppress the expression of RANK-L, MCSF of osteoblast stromal cells, and prevent the occurrence of complexs bond between RANK-L and RANK, the receptor produces OPG, RANK competent with. Indirectly inhibit estrogen production of cytokines that stimulate osteoclast differentiation, such as IL-1, IL-6, TNF-α, IL-11, and IL-7. The apoptosis of osteoclasts, indirectly produce estrogen and stimulate osteoblasts to TGF-β, while the TGF-β

will induced the osteoclasts faster to undergo the apoptosis.[11]

In men, testosterone plays an important role in bone metabolism. Similar to the role of estrogen and progesterone in women, testosterone has a role in this type of formation and bone remodeling in men. Deficiency of androgen receptor has a bearing on receptor activator of RANK-L genes, which marked influence osteoklastogenesis.[12]

Although the men did not have a sign of hormonal changes as experienced by women, but hormonal changes have an important role in the pathogenesis of osteoporosis. The level of sex hormone binding globulin (SHBG) increases with age, while the biologically available serum by the body (serum non-SHBG) estrodiol and testosterone decrease with age. Bone mineral density apparently is associated with high levels of steroids, especially bio-available levels estradiol.[12]

Tooth loss and osteoporosis

Osteoporosis and periodontitis are diseases that have a systemic change to the bone. The change in the process of bone turnover in long bones in the body. It will also be experienced by the alveolar bone. So, someone who has low bone mineral density will also have a bone mineral density in jaw.[13]

Osteoporosis mainly affects bone trabecular and cortical bone to continue. This is due to the more cortical in the composition of the maxillary bone. So osteoporosis is more common in bone, especially in the posterior maxilla where bone density is lower than in the anatomy section anterior.[14]

Some studies have also linked the influence of systemic disease of osteoporosis against tooth loss in postmenopuse women. Nicopoulou-Karayianni et al state that women who have osteoporosis have fewer teeth than the number of women who had normal bone mineral density or early osteoporosis.[15]

Talo dan Tuba-Filiz stated that women who are at postmenopouseal phase with osteoporosis have lower bone mass of the mandible, and thinning cortical bone on bone gonion. They also have lost more teeth than postmenopousal women who have normal bone mass density.[13] Suresh et al also shows the same thing in comparing the condition of periodontitis and osteoporosis in pre and post menopausal women. The result is an increase in periodontitis with an increasing proportion of osteoporosis and osteopenia.[16]

Other studies also suggested that the absence of a significant correlation between low bone mineral density and tooth loss in postmenopausal women than women with normal bone mineral density. It is associated with the prevalence and incidence of caries in children and adults who are so high and the low of socioeconomic that lead to choose extraction as the best method of treatment. Thus, a less significant correlation was obtained from the research.[14]

It is concluded that deficiency of hormone in the process of interaction osteoblast and osteoclast leads to decreased bone mineral density in elderly include in jaw bone and it causes rapid tooth loss.

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