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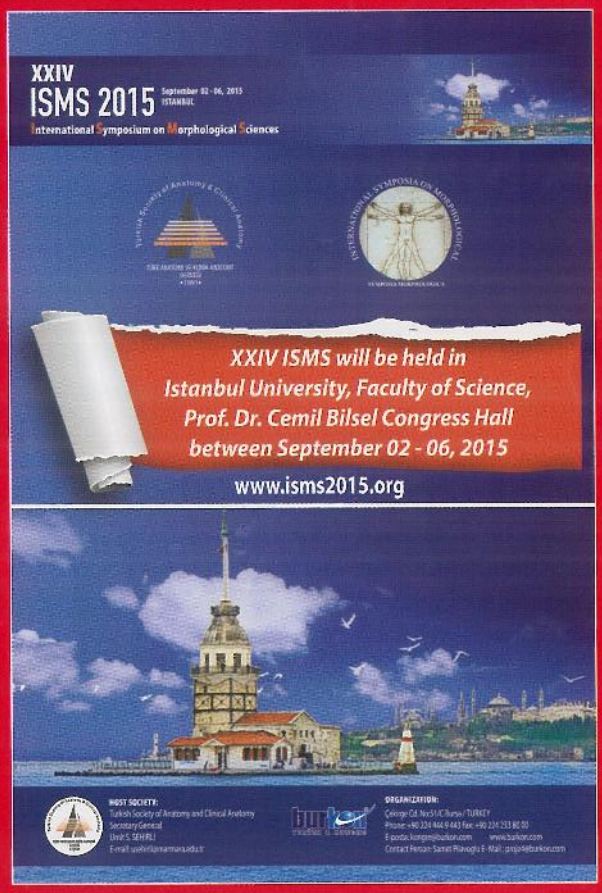

Special Issue includes abstracts for the XXIV International Symposium on Morphological Sciences 2nd–6th September, 2015, Istanbul, Turkey

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O-15

Relationship between consentration of alkaline phosphatase with bone destruction in periodontal disease patients

Kasuma N*, Darwin E**

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Periodontitis is a chronic inflammatory process which affect connective tissues surrounding the tooth (gums, periodontal ligaments,and alveolar bone) leading to attachment loss. Periodontitis may progress to bone destruction and tooth loss if it is left untreated. Clinical characteristics of periodontal disease include bleeding and friable gums, gingival recession, deepening pockets surrounding the tooth (indicating loss of anchoring attachments), and eventual tooth loosening. Alkaline phosphatase is a hydrolase enzyme, which is synthesized and secreted by polymorphonuclear neutrophils during inflamation and by osteoblast during bone formation and also by periodontal ligament fibroblast during periodontal regeneration. Creating a local bone environment of alkalinity to help bone mineralization. Acute infection causes bone destruction mechanism. Chronic periodontal inflammation increase levels of acid and alkaline phosphataseand by all products from bacteria and the destruction of tissues that support the teeth. When the inflammation spread along the transeptal fribres, it will shows a resorption of the alveolar bone crest. Due to the severity of the periodontal inflammation and bone turnover rate will increase ALP concentration. In severe periodontitis, the increasing bone turnover intensifies bone destruction by osteoclast. The purpose of this study is to examine the relationships between consentration of alkaline phosphatase with bone destruction in periodontal patients disease. This research involved 60 people with 20 healthy samples, 20 mild gingivitis samples, and 20 mild periodontitis samples. To see a normal distribution, Kolmogorov Smirnof Test is used ($p>0.05$). Post-hoc Bonferroni test is taken to test the differences each variables. Conclussion of this research is there are significant differences in the levels of Alkaline Phosphatase on the terms.

O-16

Effect of tryptophan on testosterone, estradiol and luteinizing hormone levels and on Leydig cells in male rats

Darwin E*, Putri A**, Aryaneta Y**

*Department of Histology, Faculty of Medicine, Andalas University, Padang, West Sumatra, Indonesia; **Biomedic Programe, Faculty of Medicine, Andalas University, Padang, West Sumatra, Indonesia

Introduction: Tryptophan is an essential amino acid found in many plant and animal proteins, that can be synthesized into serotonin and be converted to melatonin. Since tryptophan is the precursor of serotonin, its dietary amount has important effects on stress, mood, memory, and male sexual behavior. Melatonin is a hormone that regulates diurnal rhythms and influences the immune, gastro intestinal and reproductive systems.

Objectives: To determine the effect of tryptophan on testosterone, estradiol, luteinizing hormone levels and the number of Leydig cells in rats.

Methods: Male Rattus norvegicus were divided into four groups of seven rats, one served as control and three as treatment (P) groups. The treatment groups were given14 days intraperitoneal injection of 40, 50 and 60 mg/kg BW tryptophan for groups P1, P2, and P3. Blood was collected at day 15 to determine the level of testosterone, estradiol, and luteinizing hormone, and testis were excised and processed histopathologically to determine the number of Leydig cells

Results: Testosterone level of P1, P2 and P3 were no difference from control (12.95±1.55 nmol/l, 11.03±0.54 nmol/l, 13.57±1.79 nmol/l and 13.78±2.33 nmol/l), respectively). Estradiol level was significantly higher in P2 than control (10.17±0.85 pg/dl and 8.65±0.74 pg/dl respectively). Meanwhile, there was no difference between P1 and P3 from control (9.87±1.01 pg/dl, 8.08±0.53 pg/dl and and 8.65±0.74 pg/dl respectively). The level of luteinizing hormone was significantly lower in P1, P2 and P3 than control (3.78±0.29 nmol/l, 3.32±0.35 nmol/l, 2.96±0.28 nmol/l and 5.60±0.30 nmol/l respectively). The number of Leydig cells was significantly lower in P3, but no difference between P1 and P2 from control (17.66±0.81%, 21.00±1.09%, 19.66±1.03%, and 22.50±1.22% respectively).

Conclusion: Higher dose of tryptophan in the diet led to an increase of serotonin and melatonin, which led to an effect on the level of estradiol and luteinizing hormone. However, there were no effect on testosterone and on the number of Leydig cells.

O-17

Uric acid induces glomerulosclerosis, tubular injury and renal fibrosis through transforming growth factor, 1 elevation and fibroblast expansion

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*Department of Anatomy, Faculty of Medicine, Universitas Gadjah Mada, Yogyakarta, Indonesia; ** Department of Surgery, Faculty of Medicine, Universitas Gadjah Mada, Yogyakarta, Indonesia

Uric acid (UA) is an independent factor of cardiovascular diseases and induces renal damage. Transforming Growth Factor β1 (TGFβ1) is well known as a profibrotic factor in kidney and associated with fibroblast expansion. Here, we elucidate TGFβ1 modulation of hyperuricemia induced renal fibrosis in mice. Hyperuricemia is induced in Swiss Background mice (3-4 month, 30-35 gram, n=21) using intraperitoneal injection of 125 mg/kg of uric acid daily. NaCl injection was used in control mice. Mice were sacrificed in 7 (UA7) and 14 days (UA14) injection. Uric acid and creatinine serum is measured from retro-orbital blood serum before renal harvesting. Paraffin section is made, deparaffinized, then stained for Periodic Acid Schiff (PAS) and Sirius Red for glomerulosclerosis, tubular injury and fibrosis quantification. We extracted RNA and made cDNA, then run Reverse Transcriptase PCR (RT-PCR) for nephrine, podocine, MCP-1 and ICAM-1. PDGFR, immunostaining was done for quantification of fibroblast number. TGF,1 was measured using ELISA. $p<0.05$ was used as significant difference during data analysis. Injection of UA induced significant elevation of uric acid and creatinine level after 7 and 14 days followed by significant increase of glomerulosclerosis and tubular injury score in uric acid group compared to control ($p<0.05$). Both UA7 and UA14 groups also

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Relationship Between Consentration of Alkaline Phosphatase with Bone Destruction in Periodontal Disease Patient

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Abstract

Periodontitis is a chronic inflammatory process which affect connective tissues surrounding the tooth (gums, periodontal ligaments,and alveolar bone) leading to attachment loss. Periodontitis may progress to bone destruction and tooth loss if it is left untreated. Clinical characteristics of periodontal disease include bleeding and friable gums, gingival recession, deepening pockets surrounding the tooth (indicating loss of anchoring attachments), and eventual tooth loosening.Alkaline phosphatase is enzyme synthesized and secreted by polymorphonuclear neutrophils during inflamation, osteoblast during bone formation, periodontal ligament fibroblast during periodontal regeneration creating a local bone environment of alkalinity to help bone mineralization.This research involved 60 people with 20 healthy samples , 20 mild gingivitis samples , and 20 mild periodontitis samples. . To see a normal distribution, Kolmogorov Smirnof Test is used ($p > 0.05$). Post-hoc Bonferroni test is taken to test the differences each variables.Conclussion of this research is there are significant differences in the levels of Alkaline Phosphatase on the terms of the PDI group, which is highest in the mild periodontitis with mean = 135,74 ± 15,08 ng / dl . The condition of mild peridontitis rose 5,5-fold compared to healthy conditions.

Keyword : Alkaline Phosphatase, periodontital disease, periodontitis, bone destruction

Introduction

Periodontitis is chronic inflammatory disease and infection of periodontal tissue begins with colonization and growth Gram-negative anaerobic bacteria and spirochetes. Periodontitis will cause attachment loss, bone destruction, mobility of teeth and loss of teeth. Periodontitis is innate and adaptive immune responses. Lytic enzymes with stimulated osteoclastogenesis causes host-mediated destruction of soft tissue (Graves and Cochran, 2003).

Diagnosis of periodontitis is generally simple; by measuring gingival pocket depth; measuring Periodontal Disease Index and dental X-ray , but to diagnose and to determine disease progression , treatment evaluation , researchers have been used biological marker which can be found in gingival crevicular fluid . One of the enzymes of tissue degradation is Alkaline Phosphatase (ALP). (Dabra, 2012).

ALP is intracellular enzymes which is found particularly in bones. Destructive processes in alveolar bone in advanced stages & acute phase increase ALP concentration. After periodontal therapy, ALP level turn into normal (Yoshie, et al, 2007). Humans have 4 types of ALP based on enzyme source : Tissue - nonspesific alkaline phospatase (TNAP); Placental Alkaline Phospatase (PLAP) ; Germ Cell Alkaline Phospatase (GCAP); Intestinal Alkaline Phospatase (IAP)(Milan, 2006) . TNAP works in bone mineralization in periodontitis. Acute infection causes the mechanism that causes bone destruction. Chronic periodontal inflammation resulting in increased levels of acid and alkaline phosphatase,all produced from bacteria and the destruction of the tissues supporting the teeth.When the inflammation has spread along the fibers transeptal, it will show the presence of resorption of the alveolar bone crest. Due to the severity of the periodontal inflammation, bone turnover rate and ALP concentration increase. In severe periodontitis, the increasing bone turnover intensifies bone destruction by osteoclast. The purpose of this study is to see relationship between ALP level and bone destruction in periodontal disease patients.

Method

This study is a cross sectional comparative study in 3 groups of sample based on Periodontal Disease Index (PDI) by Ramfjord. Samples were taken by consecutive sampling technique based on exclusion and inclusion criteria. The excluding criteria was consuming antibiotics and antiinflamatory during the last 3 months, smoker, pregnant, menstruation, have a systemic disorders such as diabetes melitus,and got a history of periodontal treatment during the last 3 months . All subjects were informed of the purpose and informed consent was obtained from all the individuals and local ethical committee approval that was in accordance with the last update of Helsinki declaration was obtained.

GCF was collected in sterile test tubes from each person between 8.00 and 12.00 am. All subjects were requested to avoid eating and drinking 1 hours before sampling. GCF was collected with Absorbing Paper Strip method. Patients sit on dental chair then to rinse with a solution of 2% khlorheksidin. Paper points is inserted by using the technique of superficial intracrevicular then left for 3 minutes. then each tube was frozen at –-20°C until sending to biochemistry laboratory. Another GCF collection was done only in patient group during 30 days. After 60 saliva samples were sent to biochemistry lab at one time to avoid the problems of several setting up of the unit. Samples were analyzed usingElisa Kit for ALP, homo sapiens (Human), sE91472Hu with detection range 3.12-200 ng/ml and sensitivity1.36 ng/ml USCN product by spectrophotometer variant hemoglobin testing analyzer called Bio rad. The measurement of ALP in GCF which collected from gingival pocket from three groups is tested using ELISA sandwich methode. Laboratory test performed on biomedical laboratory Faculty Medicine of Andalas University. SPSS statistically software using Post-hoc Bonferroni test is taken to test the differences each variables ($p > 0.05$).

Result

According to Table 1 there is significant difference between ALP level in mild periodontitis and mild gingivitis as well as healthy group based on Periodontal Disease Index (p<0.05).

Table 1. Difference of Alkaline Phosphatase levels (ng / dl) in Gingival crevicular Fluid with Periodontal Disease Based on PDI

Enzyme	PDI	f	Mean	SD	p
ALP	Healthy	20	24,68	19,74	0,00
	Mild Gingivitis	20	88,85	20,31	
	Mild Periodontitis	20	135,74	15,08	
	Total	60	83,09	18.38	

There are significant differences in the levels of ALP highest in the mild periodontitis with mean = 135,74 ± 15,08 ng / dl .Mild gingivitis patients likely to have elevated levels of ALP 3,6 –fold. Mild peridontitis rose 5,5-fold compared to healthy conditions.

Tabel 2. Differences of each group with Post-hoc Bonferroni test based on Periodontal Disease Index (PDI).

	ALP Level		
PDI	Healthy	Mild Ginggivitis	Mild Periodontitis
Healthy	-	0,00	0,00
Mild Gingivitis	0,00	-	0,00
Mild Periodontitis	0,00	0,00	-

There is significant difference of ALP level between healthy and mild gingivitis group, healthy and mild periodontitis, mild gingivitis and mild periodontitis group (p<0.05).

Discussion

The increased activity of ALP, indicates that the pathological destructive process had affected the alveolar bone, which means that periodontal disease had significantly advanced. Alkaline Phosphatase is bone metabolism enzyme . ALP is enriched in the membranes of mineralizing tissue cells (e.g. osteoblasts) and PMN granules. ALP is produced by some oral bacteria, (gram-negative microorganisms) in subgingival plaque (Dabra, 2012).

ALP is the main glycosylated protein present in bone whoch is bound to osteoblast cell surfaces *via* a phosphoinositol linkage within mineralized matrix , plays an as-yet-undefined role in mineralization of bone (Whyte, 1994 ; Clarke, 2008). Major source of ALP during inflammation has been proposed to be neutrophils , then cause bone loss . Neutrophil predominance in the pocket epithelium and the pocket itself. ALP regulate mineralization process . ALP increases the concentration of phosphorus , growth inhibitors of hydroxy apatite and phosphoprotein (Usal *et al* , 2008) . The source of ALP are polymorphonuclear neutrophils during inflammation, osteoblasts for bone formation , periodontal ligament fibroblasts during periodontal regeneration .

ALP bind and regulate calcium and phosphate mineral deposition by regulating the amount of hydroxyapatite formed. This enzyme is first identified to indicate the location of the inflammation . When osteoblasts form bone , and fibroblast regenerate the periodontal ligament , ALP production increased.Among the enzyme in GCF , ALP is one of the first identified , which are membrane - bound glycoprotein produced by the cells in the periodontium and gingival crevice . There is a double involvement in the process of periodontal inflammation and healing / regeneration . (Perinetti et al. , 2008)

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