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The Effects of Cr(VI) in the kidney of experimental rats and utilization of longanpeel fruit (*Dimocarpuslongan*) as renal protector in dentistry

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ABSTRACT

The present study investigated the effect of Cr(VI) in the kidneys of experimental rats and the protective effect of Dimocarpuslongan peel fruit as renal protector. Administration of 1 mLCr(VI) 1000 mg/L in experimental rats intraperitoneally increased all the biochemical parameters that were observed, including: MDA, urea, creatinine, SGOT and SGPT. Histologically, administration of Cr(VI) leads to swelling of tubule and severe necrosis in kidney. Pre-treatment with D.longan peel fruit antidote reduced the levels of biochemical parameters and oxidative stress parameters. The decreased levels of MDA, urea, creatinine, SGOT and SGPT were 51, 74%; 30,26%; 31,37%; 26,74% and 67,19% respectively. The pre-treatment with D.longan peel fruit also reduced the damage in kidney tissue, although there was still swelling in the tubules.

Keywords: *Dimocarpuslongan peel fruit*, Cr(VI), lipid peroxidation, kidney

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INTRODUCTION

Chromium is a widespread heavy metal in the earth's crust. Soil erosion is the major cause of chromium release into water, whereas the main source to air, soil, and water comes from fossil fuel combustion and industrial processes [1]. Ubiquitous, chromium and its compounds are persistent contaminants released into environment from a variety of anthropogenic sources including these: electroplating industries, tanneries, wood preservation, alloy industries, and dyes industries [2]. Chromium has several valence state, hexavalent chromium (Cr(VI)) and trivalent chromium (Cr(III)), with Cr(III) being the most prevalent form in nature. Cr(III) is an essential trace element. Cr(VI), in contrast to Cr(III), istoxic and is rarely found in nature, coming instead from industrial processes [1]. Several toxic effects result from Cr(VI) exposure in human, including these: mouth ulcers, acute tubular necrosis, vomiting, abdominal pain, kidney failure and even death [3]. Animal studies reported that Cr(VI) given to the experimental rats through drinking water can lead to cellular infiltration in the liver, pancreas, and small intestine [4]. In dentistry, chromium is one of the materials used in manufacturing orthodontic appliances like brackets, bands and arch wires. Previous studies reported that orthodontic therapy can induce DNA damage in oral mucosa cells as a result of metal ion release by fixed orthodontic appliances [5]. Sfondrini et al [6] reported that the greatest amount of chromium was released from new stainless steel brackets (0,52 ±1,083 µg/g) whereas the recycled brackets released 0,27±0,38µg/g; so the release of chromium has potential for inducing toxicity. Because the main route for chromium excretion is through the kidneys, increasing the content of Cr will lead to nephropathy [7]. Longan

(*Dimocarpuslongan*) is widespread in China, Taiwan, Vietnam and Thailand. This fruit not only possess a sweet taste, but also positively benefits health. Longan fruit is used as a traditional medicine in China to improve metabolism of blood and to relieve insomnia [8]. Longan peels contain high amounts of active compounds such as phenolic acids, flavonoids and polysaccharides, and also exhibit antibacterial, antiviral and antioxidant properties [9,10]. Kurniawan et al [11] proposed the peel of *Dimocarpuslongan* as a potential low cost biosorbent of heavy metals. The study reported that longan peel had a potential as an adsorbent for Cu(II), with a maximum capacity for Cu(II) of 8,2175 mg/g. Florenly et al [12] reported that the seeds and peel of longan have adsorption maximum capacities for removing Cr(VI) in aqueous solution of 8,9237 mg/g and 8,1021 mg/g respectively. The study of using *D.longan*peel fruit against Cr(VI) induced nephrotoxicity has not been done; therefore, this study aims to investigate this matter both histopathologically and biochemically.

MATERIALS AND METHODS

Dimocarpuslongan Peel Fruit Material

The peel of *Dimocarpuslongan fruit* was collected from a local market in Padang, West Sumatera, Indonesia.

Preparation of peel fruit of Dimocarpus as antidote

The *D.longan peel fruit* was shredded and washed with tap water. Then, it was air dried for 2 weeks at room temperature. Dehydrated, the *D.longan peel fruit* was grinded into powder form using a blender. 2 g of the powder was boiled with 120 mL distilled water for a few minutes, then filtered and stored in a sealed bottle

Chemicals

All chemicals used in this study, including potassium dichromate, are analytical grade and obtained from Merck(Darmstad, Germany).

Experimental Rats

Adult rats used in this study weighing between 140-160 g were purchased from the Faculty of Pharmacy, Andalas University. The rats were placed in proper cages and maintained on ad libitum diet and water. All treatments and protocols in this study are in accordance with protocol of the animal ethics committee of Andalas University.

Experimental Design

3 groups of 3 rats were formed in this study. The first group (Group 1st) was the control, in which the rats were given only distilled water. The second group (Group 2nd) was the Cr(VI) treated-group, in which rats were given 1 mL Cr(VI) 1.000 mg/L intraperitoneally. The third group (Group 3rd) was the group with pre-treatment of 5 mL*D.longan*peel fruit solution antidote given orally for a week, followed by administration of 1 mL of Cr(VI) 1.000 mg/L intraperitoneally. The experimental rats were left for 5 hours, then the rats were sacrificed using chloroform anesthesia. The blood of rats was collected for analysis of serum biochemical parameters and oxidative stress. The rats were then dissected, kidneys removed and fixed in Bouin's solution for histological analysis.

Analysis of Serum Biochemical Parameters and Oxidative Stress

Serum biochemical parameters examined in this study include levels of liver function enzymes SGOT and SGPT, kidney function parameters, urea and creatinine, and oxidative stress, which was measured in the form of the lipid peroxidation product, malondialdehyde (MDA).

Histopathology Analysis

The kidneys were sliced using a microtome, stained with hematoxylin and eosin, and analyzed using a light microscope.

Statistical Analysis

The data, particularly the level of serum biochemical parameters and oxidative stress, were analyzed statistically using "Statistical Package for Social Science Program ver.16 Software" (SPSS ver.16). The analysis was conducted using analysis of variance (ANOVA) followed by Tukey Test.

RESULTS AND DISCUSSION

The Level of Liver Function Enzyme, Kidney Function Parameters and Malondialdehyde.

The result of the SGOT, SGPT, urea, creatinine and MDA level was showed in table.1

TABLE 1 THE MEAN LEVEL OF SGOT, SGPT, UREA, CREATININE AND MDA IN SERUM

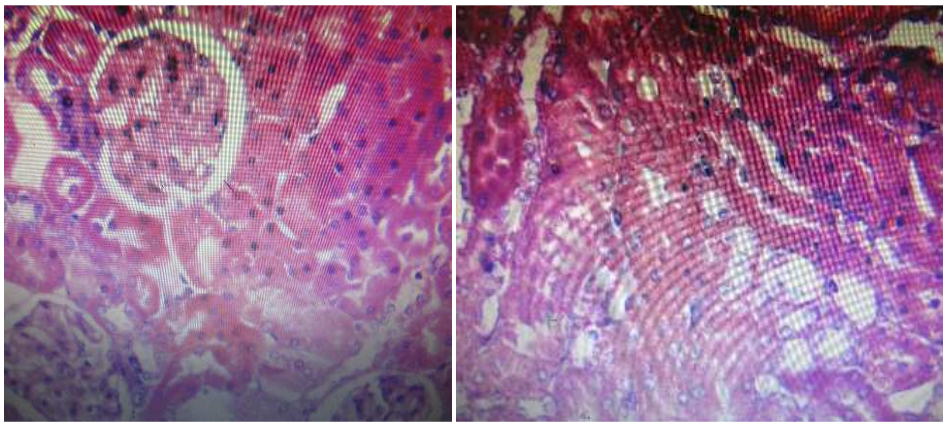
No	Parameters	Group 1st (Control)	Group 2nd (Cr(VI) treatment)	Group 3rd (pre treatment with antidote)
1	Malondialdehyde (MDA) (mg/dl)	3,61	5,74	4,11*
2	Urea (mg/dl)	38,36	32,98	23,00*
3	creatinine (mg/dl)	0,21	0,51	0,35*
4	SGOT (U/L)	111,987	56,53	41,41*
5	SGPT (U/L)	25,88	46,03	15,1*

**P<0,05 compared to group 2nd*

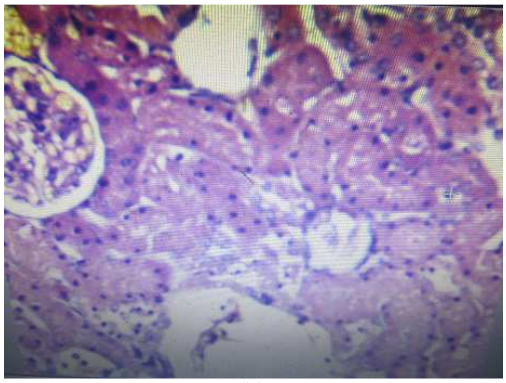
The administration of Cr(VI) in rats increased almost all the biochemical parameters that were observed. In group 3, there were significant decreases of the parameters compared to group 2. The decrease of MDA, urea, creatinine, SGOT and SGPT in group 3, compared with group 2 are 51,74%; 30,26%; 31,37%; 26,74% and 67,19% respectively. Cr(VI) induces the production of free radicals through various mechanisms that cause peroxidation, which is measured by an increase of MDA, a lipid peroxidation product. The peroxidative damage also occurs in the liver and kidney. Balakrishnan et al [13] reported that administration of potassium chromate (10 mg/kg b.wt) in experimental rats can lead to elevated levels of MDA and reduced hepatic and nephronic function. These was reflected by significant increase of serum ALT levels, indicating hepatotoxicity and increased serum BUN and creatinine levels, indicating nephrotoxicity. Similar results were reported by Mehany et al [14]. Mehany et al [14] reported that administration of potassium dichromate intraperitoneally at a dose 15 mg/kg to rats, resulted in renal tubular damage and increased markers of renal malfunction (blood urea, nitrogen, and creatinine). Pre-treatment with vitamin E for 2 weeks could reduce urea levels. Oxidative stress markers such as MDA also decreased. Jhong Huang [8] reported that the water extract of longan peel showed DPPH scavenging activity and high levels of both total polyphenols and protection liposome. Total polyphenols, DPPH scavenging activity, and protection liposome of longanpeel was respectively, 18,5 mg/mL, 70,0 mg/mL and 20,6 mg/mL. These results imply that *D.longan* peel antidote could protect lipid molecules against oxidative damage.

Histopathology Analysis

The protective effect of *D.longan* peel fruit antidote against Cr(VI) induced nephrotoxicity described in Figure.1



(a) **(b)**



(c)

Figure 1 : (a). photomicrograph of kidney in control group (group 1st) (b). Photomicrograph of kidney in group 2, normal glomerular, there are swelling of tubules and severe necrosis. (c).Photomicrograph in kidney in group 3, there are no necrosis, but the tubules are swelling

Molina Jijon et al [15] reported that exposure with Cr(VI) will resulted in tubular damage and acute tubular necrosis. It is also reported that oxidative stress is a main cause of the cell and tissue damage resulting from Cr(VI). ROS will attack free fatty acids in membrane lipids which will cause lipid peroxidation and affect structure and cellular function. Pre-treatment with *D.longan* peel antidote could reduce damaging effects that result from lipid peroxidation, although swelling tubules will still prevail. Yang et al [16] reported that crude extract of longan pericarp possess high phenolic content and shows higher significant scavenging activities of free radicals such as DPPH and ABTS radicals. The result showed that *D.longan* peel antidote has the potential to reduce the toxicity from Cr(VI)exposure, so it has a potential to be used as antidote in Cr(VI) toxicity induced by the appliance of orthodontics therapy.

CONCLUSION

The administration of Cr(VI) in experimental rats resulted in elevated levels of serum biochemical parameters including MDA, urea, creatinine , SGOT and SGPT. Histologically, administration with 1 mLCr(VI) 1000 mg/L will induce damage of kidney tissues such as swelling tubules and necrosis. The pre-treatment with 5 mL *D.longan* peel fruit solution antidote everyday for 1 week could reduce the elevated levels of serum biochemical parameters caused by Cr(VI) toxicity, thereby reducing kidney damage. The protective effect of *D.longan* peel antidote in kidney of experimental rats may be due to the anti-oxidant mechanism that could prevent lipid peroxidation.

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