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Potential Therapy for Damaged Kidney Tubule Cells Due to Aluminium Chloride Induction Using Human Wharton Jelly Mesenchymal Stem Cells

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ABSTRACT

Exposure to aluminium chloride (AlCl3) can occur through many different methods. Most of the aluminium will be excreted by the kidneys. Still, if the amount entering the body is high enough, it can lead to kidney dysfunction. This study uses AlCl3, which is nephrotoxic, so that it can damage renal tubular cells. The damage caused by AlCl3 can be overcome by human Wharton jelly mesenchymal stem cells (HWJMSCs), which are currently being developed as a therapy for tissue and cell repair because they have repair and anti-inflammatory agents against body tissues and cells.

This experimental study used 18 rats as experimental animals, divided into three groups. The first group received no treatment (negative control). The Second group was induced with AlCl3 (positive control). The third group was induced with AlCl3 and then given HWJMSCs (treatment). Renal tubular cell damage was analyzed using a one-way ANOVA, followed by a post hoc Bonferroni test. In the data obtained, the average percentage of cell damage was 40,71% (negative control), 72,89% (positive control), and 57,58% (treatment). Data analysis showed a significant difference between the positive and negative control groups ($p = 0,001$ ($p < 0,005$)), but no significant difference between the treatment group and positive control group ($p = 0,061$) and negative control group ($p = 0,009$). This study concludes that HWJMSCs play a role in repairing renal tubular cell damage, but this effect is not optimal.

Keywords: *renal tubular cells; aluminium chloride ($AlCl_3$); human Wharton jelly mesenchymal stem cells (HWJMSCs)*

INTRODUCTION

Exposure to AlCl3 can occur through many different media and methods, such as exposure from antacids, water use, food additives, deodorant use, and pharmaceutical products.[1] Some aluminium enters the human body from food, and smaller amounts enter through the skin as antiperspirants. Most of the aluminium is excreted by the

kidneys, but if the amount of aluminium entering the body is high enough, it can lead to aluminium accumulation in the kidneys, which can cause kidney dysfunction.[2] The Food and Drug Administration (FDA) has approved the use of AlCl3 as an active ingredient at up to 15% calculated in the form of hexahydrate, in aqueous solutions in non-aerosol preparations.[3]

Aluminium accumulation in internal organs can be prevented by the kidneys through the process of urine excretion, but the kidneys are also very susceptible to the nephrotoxic effects of aluminium.[4] Aluminium accumulation in the kidneys can occur through the amplification effect of the food chain cascade, which induces nephrotoxicity. Aluminium can induce changes in renal histopathology, such as tubular necrosis, glomerular atrophy, organelle vacuolation, and mitochondrial degeneration, leading to decreased glomerular filtration rate and renal tubular reabsorption rate, which in turn can cause renal dysfunction.[5]

The toxicity of AlCl3 has been reported to cause renal tubule cell degeneration by increasing the production of reactive oxygen species (ROS), thereby causing oxidative stress (OS) in cells, decreasing glutathione (GSH) concentrations, and causing oxidative damage to deoxyribonucleic acid (DNA).[6] Under physiological conditions, ROS are produced and inactivated by cellular and extracellular defense mechanisms because, under normal circumstances, there is a balance between oxidants and the antioxidant defense system. When an imbalance occurs between oxidants and antioxidants, it leads to increased ROS and OS activity, causing tissue damage.[7] The accumulation of ROS directly contributes to biomacromolecular damage and influences various inflammatory responses.[8]

Research on AlCl3 exposure in rats found that OS was induced by ROS formation, which disrupted the normal antioxidant system, significantly reducing GSH and glutathione s-transferase (GST) concentrations in kidney cortex homogenates. Disruption of the normal antioxidant system also significantly increases the concentration of lipid peroxidation (LPO) in rat kidneys. In a study conducted with AlCl3 exposure (25 mg/kgBW intraperitoneal) in rats, an increase in malondialdehyde (MDA) concentration was found in rat kidneys. Malondialdehyde is one of the products of LPO, indicating that AlCl3 exposure increases LPO concentration and decreases the activity of antioxidant enzymes, namely glutathione peroxidase (GSH PX) and catalase (CAT).[9]

Renal tubule cells can be used as a parameter in this study due to significant changes, namely, structural damage to the renal tubules due to exposure to AlCl3. In a study conducted by inducing AlCl3 (30 mg/kgBW) intraperitoneally every 5 days for 15 days, histopathological damage to the kidneys was observed. In this study, dilation of the renal Bowman's capsule, atrophy and destruction of the glomerulus, obstruction of the renal tubules, and degeneration of tubule cells with pyknotic nuclei were observed.[4]

Stem cells are undifferentiated cells, which allows them to develop into new cells whose properties will depend on the microenvironment in which they develop.[10] Mesenchymal stem cells (MSCs) are a type of cell with great potential, originating from the mesoderm, which plays an important role in various stem cells. Mesenchymal stem cells have strong proliferation capacity and diverse differentiation potential. Mesenchymal stem cells can differentiate into osteoblasts, cartilage, or adipocytes under appropriate in vivo or in vitro induction conditions. In addition, MSCs have the capacity to differentiate into heart, liver, endothelial, hematopoietic, nerve, and other cell types. Mesenchymal stem cells can maintain their differentiation potential even after repeated subculturing and cryopreservation, thereby producing new cells.[11]

Previous studies have shown that MSCs can increase transforming growth factor-beta (TGF-β) and decrease tumor necrosis factor alpha (TNF-α), in addition to the ability of

MSCs to reduce MDA, ROS, and increase GSH. Human MSC injections also show that not only are inflammatory factors reduced, but also inflammatory cell infiltration and extracellular matrix deposition. This also indicates that MSCs can replace damaged kidney cells by increasing TGF-β, a multifunctional cytokine that plays an important role in regulating several cellular processes, including self-renewal and cell differentiation.[11,12,13]

Mesenchymal stem cells have been shown to secrete bioactive molecules such as cytokines/chemokines and growth factors, including granulocyte colony-stimulating factor, leukemia inhibitory factor, macrophage colony-stimulating factor, prostaglandin E2 (PGE2), interleukin-10 (IL-10), TGF-β, vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and insulin-like growth factor-1 (IGF-1). Mesenchymal stem cells do not directly replace damaged epithelial cells. Instead, the cells modulate the kidney's regenerative response, which in turn has been shown to accelerate the recovery phase, indicating that the cells replacing the kidney epithelium are known to originate from within the kidney itself. Surviving kidney cells differentiate and replicate to restore the damaged epithelium.[14]

Wharton's jelly is a type of special mucous connective tissue that protects the umbilical cord. Wharton's jelly is an amorphous substance rich in glycosaminoglycans, proteoglycans, and mostly hyaluronic acid. Wharton's jelly is surrounded by myofibroblasts, which are mesenchymal cells with characteristics of fibroblasts and smooth muscle cells. Wharton's jelly functions as an adventitial layer, which is not found in the umbilical cord, and binds the fibers surrounding the umbilical blood vessels.[15]

Human Wharton jelly mesenchymal stem cells (HWJMSCs) have high proliferation potential. This type of mesenchymal stem cell does not transform into teratogenic or carcinogenic cells upon transplantation. Bone marrow and adipose tissue are used as sources of MSCs. However, bone marrow as a source of MSCs has many disadvantages. The first disadvantage is that only a small portion of the body's bone marrow cells are MSCs. The second disadvantage is that bone marrow collection requires an invasive and very painful procedure. On the one hand, although adipose tissue is abundant, its collection also requires an invasive procedure. Human Wharton's jelly mesenchymal stem cells are a better source for MSCs because the tissue is globally accessible, and its collection does not pose ethical concerns, as it is usually discarded as human waste.[16,17]

Based on the above description, this study is interested in examining the potential of human Wharton jelly mesenchymal stem cells on the histopathological appearance of rat kidney tubule cells damaged by aluminium chloride induction. This study will focus on analyzing the impact of human Wharton's jelly mesenchymal stem cell administration on the histopathological appearance of kidney tubules damaged by aluminium chloride induction. It is hoped that this study will contribute further knowledge about the use of human Wharton's jelly mesenchymal stem cells as a therapy for cell damage.

MATERIALS & METHODS

This study is an experimental study with a posttest-only control group design. This study was conducted at the Biomedical Animal House Laboratory and the Anatomical Pathology Laboratory of the Faculty of Medicine, Andalas University, from November 2022 to December 2023. This study has passed ethical review from the Ethics Committee of the Faculty of Medicine, Universitas Andalas (No: 456/UN.16.2/KEP-FK/2023).

Two-month-old male Wistar rats (Rattus norvegicus) weighing between 200 grams were selected for the study. The rats were kept in cages at a stable temperature of about 26°C and given a standard pellet diet every day for a 7-day acclimatization period. After 7 days, they were weighed and randomly

divided into three groups. Group I (negative control) received no AlCl3 administration, Group II (positive control) received AlCl3, and Group III (treatment) received AlCl3 and HWJMSCs. Sample size was calculated using the WHO formula, resulting in 5 rats per group. To compensate for possible dropouts, 1 extra rat per group was included, totaling 18 rats.

AlCl3 (cat no 1.01084.1000, Milipore Merck, USA) was given orally at a dose of 300 mg/kg body weight per day for 5 days. On the 13th day, after the AlCl3 treatment, Group II was terminated, and their kidneys were removed. Group III received an intraperitoneal injection of HWJMSCs. HWJMSCs were obtained previously from the IMERI FKUI laboratory and identified using CD73, CD105, and CD90 markers. The dose used was 1×10^6/mouse in 500 µL of complete medium (a-MEM high glucose, penicillin/streptomycin, and fetal bovine serum) that had undergone passage 3^6. On the 30th day, group I and group III were terminated, and their kidneys were removed. The kidneys were immediately washed using a cooled 0.9% physiological NaCl solution. The rat kidneys were then immersed in 10% formalin. Subsequently, the rat kidneys were removed, and histological preparations were made using paraffin blocks and hematoxylin eosin (HE) staining. The kidneys were cut transversely at the posterior third, 3-5 mm thick. Each kidney specimen will be observed microscopically using a light microscope. The part observed is the tubule cells in the cortex that have undergone cell damage. The image selected shows the area with the most tubule cell damage, and 5 overlapping areas are then selected at 400x magnification. Each image is counted using the ImageJ 2018 version 1.52a application, where the number of damaged cells is multiplied by 100%.

Normality of the data was evaluated with the Shapiro-Wilk test. When normal distribution was confirmed, One-way ANOVA was used for parametric analysis, and a Bonferroni post hoc test was applied.

RESULT

The results of this study can be seen from the histopathological description of the renal tubule cells in each group. The percentage of renal tubular cell damage was calculated using ImageJ.

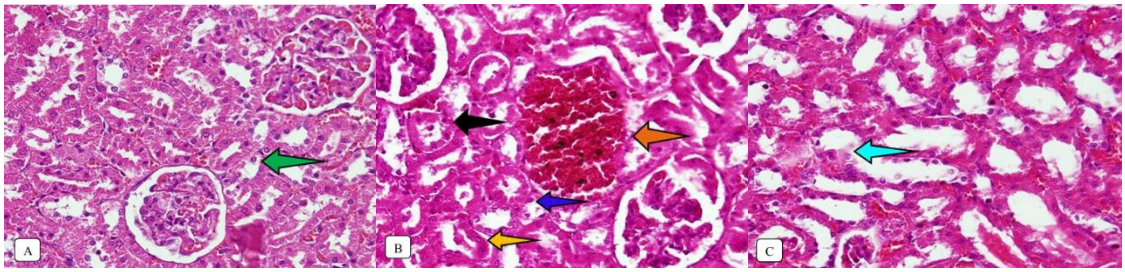

Figure 1. Microscopic image of renal tubule cells with 400x magnification and HE staining. A) Group I, normal tubule cells (green arrow). Group II, identifying damage in the form of pyknosis (black arrow), hemorrhage (orange arrow), karyolysis (yellow arrow), and karyorrhexis (blue arrow). C) Group III, less tubular cell damage compared to the group II, hydrophilic degeneration (turquoise arrow).

In Figure 1, Group I shows the histopathological picture of the negative control group that was not given AlCl3, showing normal tubular cells, and the table shows that the negative control group had less tubular cell damage than the other groups. Group II shows histopathological findings in the AlCl3-induced positive control group, including tubular cell damage (pyknosis, cell degeneration, karyolysis, karyorrhexis), and significant tissue hemorrhage. the positive control group appears to have greater tubular cell damage than the other groups. Group III shows the histopathological findings in AlCl3-induced kidney cells after treatment with HWJMSCs. In this group, there was more tubular cell damage than in group I but less than in Group II. This indicates a reduction in renal tubular cell damage in the treatment group.

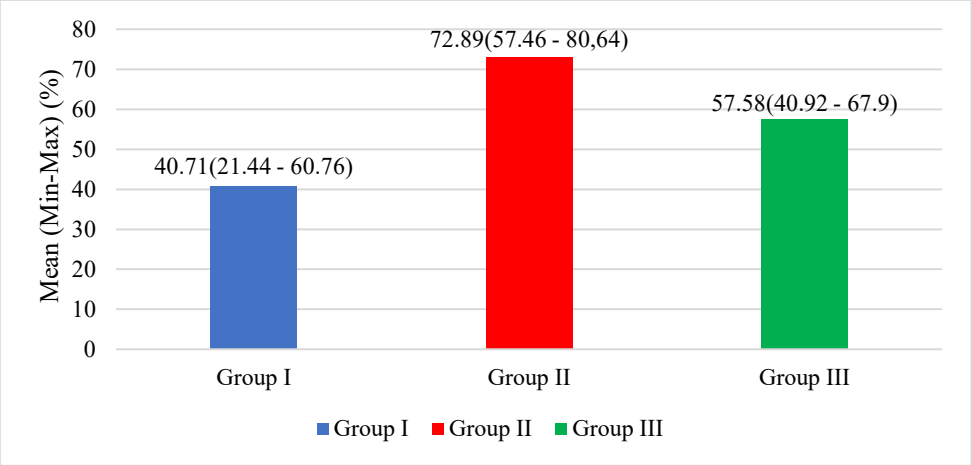

Figure 2. Effect of HWJMSCs on the Average Damage to Renal Tubules on rat model induced AlCl3.

The results of the calculation of tubule cell damage in each group are presented in Figure 2. The average renal tubule cell damage in each group was 40.71% in the negative control (Group I), 72.89% in the positive control (Group II), and 57.58% in the treatment group (Group III). The analyzed data showed that the percentage of renal tubule cell damage was highest in the positive control group compared to the negative control and treatment groups.

Table 1 The Difference in the Average Damage to Renal Tubules in Each Group Using the Post Hoc Test

Research Groups		*P* Value
Group I	Group II	0,001*
	Group III	0,061
Group II	Group I	0,001*
	Group III	0,009
Group III	Group I	0,061
	Group II	0,099

Note: * significant *p* value

The data normality test showed the data were normally distributed ($p > 0.05$). Then, a one-way ANOVA was performed, and the results showed $p = 0.001$ ($p < 0.05$), indicating a significant difference in the average number of tubule cells across groups. Furthermore, because the homogeneity test proved positive, a Post Hoc Test could be performed using the Bonferroni test to determine the difference in the average number of tubule cell damage in each group. Table 1 shows a significant difference in mean damage to renal tubular cells between group I and group II, as indicated by the p-value of 0.001 ($p<0.005$). Meanwhile, the p-values between group III and group I and Group II were $p = 0.061$ and $p = 0.099$, respectively, indicating ($p > 0.005$).

DISCUSSION

Negative Control Group Renal Tubule Cells

Natural cell death has been observed for many years and has long been considered a passive phenomenon and viewed as the inevitable endpoint of biological systems. Cells can remain dormant, supporting the relationship between organ structure and function, proliferate, sometimes becoming hypertrophic, or die. The regulation of homeostatic balance between cell proliferation and cell death is important for the development and maintenance of multicellular organisms.[18] Therefore, this is in line with the results of this study, where in the negative control, there was some damage to the renal tubule cells in mice that AlCl3 did not induce because this was physiological cell death.

Positive Control Group Renal Tubule Cells

This study found greater damage to renal tubule cells compared to other groups, with an average damage percentage of 72.89%. This is in line with previous studies.

A study was conducted by injecting AlCl3 intraperitoneally at a dose of 5mg/kgBW for two weeks. The histopathological findings showed mild necrosis of the kidney, with increased Bowman's space, glomerular capillary degeneration, macrophage infiltration, and thickening of the outer renal membrane.[6]

A study was conducted by injecting AlCl3 at a dose of 30mg/kgBW intraperitoneally every five days for fifteen days. The changes in kidney histology included widening of the renal cortex and Bowman's capsule, atrophy and destruction of the glomerulus, tubular obstruction, and degeneration of tubular cells with pyknotic nuclei.[4]

Renal function tests in a study showed that at the onset of Aluminium exposure, renal function in rats was damaged, primarily in the renal tubules. This phenomenon can be explained by the fact that chemicals are concentrated in the renal tubules after water is reabsorbed, allowing certain substances that are non-toxic in plasma to reach toxic concentrations in the renal tubules and precipitate, which can cause damage. TUNEL staining showed that apoptotic cells were significantly increased in kidney cells from the Al group, mainly in the urinary tubular system. This was further confirmed by histological results, which showed that Aluminium exposure mainly damaged the renal tubule system.[18,19]

AlCl3 toxicity can cause renal tubule cell degeneration by increasing ROS production. Reactive oxygen species oxidize micromolecules, thereby disrupting the redox status of cells. Aluminium also induces LPO in tissues, decreases reduced GSH levels, and reduces the activity of antioxidant enzymes such as CAT, GSH PX, and GST. Reactive oxygen species induced by nephrotoxic substances can also cause cell injury and apoptosis, leading to caspase-3 activation and ultimately impairing tissue function and morphology.[19]

Free radicals produced by Al can also affect cell integrity, induce LPO in intracellular membranes, and form cross-links with membrane macromolecules. Lipid peroxidation causes permanent damage to biological membranes (plasma, mitochondrial, and nuclear membranes), macromolecules (DNA, RNA, proteins, enzymes), and micromolecules (amino acids) and other smaller molecules. This lipid peroxidation will cause OS.[4,20]

In a study by Liu et al. (2022), it was shown that OS can produce MDA, which can cross-link to protein molecules, thereby inducing cell apoptosis. In this condition, the accumulation of ROS in cells can disrupt mitochondrial permeability and the release of apoptosis factors, such as cytochrome C, which can trigger caspase activation and ultimately lead to cell apoptosis.[22]

Treatment Group Renal Tubule Cells

Based on the analysis of data from the negative control and treatment groups, there was no significant difference, indicating that the amount of damage in the two groups was nearly identical. Therefore, it can be concluded that the treatment group experienced improvement in damaged renal tubule cells. This is in line with research stating that, in their study, a dose of 2×10^6 was administered. After 2 weeks, histological changes in the kidneys were manifested by a significant reduction in renal vacuolar degeneration, inflammatory cell infiltration, and renal interstitial fibrosis after UC-MSC treatment.[22]

In a study, HWJMSCs has been shown to release bioactive molecules such as cytokines/chemokines and growth factors, including granulocyte colony-stimulating factor, leukemia inhibitory factor, macrophage colony-stimulating factor, prostaglandin E2 (PGE2), interleukin-10 (IL-10), TGF-β, vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and insulin-like growth factor-1 (IGF-1). Mesenchymal stem cells do not directly replace damaged epithelial cells; instead, they modulate the kidney's regenerative response, which, in turn, has been shown to accelerate recovery. This indicates that the cells replacing the kidney epithelium are known to originate from

within the kidney itself. The surviving kidney cells differentiate and replicate to restore the damaged epithelium.[14]

Further analysis of the Post Hoc test revealed no significant difference between the positive control group and the treatment group. MSC-UC therapy has been proven to improve kidney cell repair. However, MSC-based therapy is limited by the low survival rate of MSCs themselves. Several factors, such as anoikis, ischemia, inflammation, and ROS production, reduce the effectiveness of MSC-based therapy. Researchers say that pre-conditioning MSCs can protect them from the harmful environment at the site of damage and improve their function.[23]

Previous studies have also shown that MSC treatment is ineffective. The data obtained showed that MSCs did not contribute significantly to epithelial renewal after renal cell injury, leading researchers to conclude that the significant impact on epithelial renewal in renal cell injury may be due to paracrine or endocrine effects unrelated to MSC transdifferentiation.[24]

A requirement for the effectiveness of MSC therapy is its ability to migrate to damaged tissue. Research has shown that MSCs can be localized at the site of injury, but only a small portion is delivered to that location. Migration or proliferation may be influenced to a greater or lesser extent by local tissue injury, so that the amount delivered to the site of injury studied is quite low.[25]

This research was conducted in accordance with the established steps and procedures, but it cannot be denied that there are still limitations that need to be considered. The main Limitation is the decline in the health of the experimental mice, which cannot be avoided due to environmental factors. The second Limitation is that the MSC dose used in the study was insufficient to optimally treat renal tubular cells.

CONCLUSION

Human Wharton's jelly mesenchymal stem cells can repair damage to rat renal tubule cells caused by aluminium chloride induction, so that the damage to the tubule cells in the treatment group is close to that in the negative control group. However, there is no significant difference compared with the positive control group, indicating that human Wharton's jelly mesenchymal stem cells are not yet optimal for repairing damage to renal tubular cells induced by aluminium chloride.

Declaration by Authors

Ethical Approval: Approved

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