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Effect of Interrupted Water Restriction on E-Cadherin Expression in Renal Tissue of Male Mice

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ABSTRACT:

Background: It has been shown that chronic or repeated water deprivation induces renal functional and structural changes including tubular epithelial cell damage and interstitial changes. Reduced E-cadherin expression correlates with epithelial dysfunction and has emerged as a critical marker of renal epithelial health and structural maintenance.

Methods: A total of 60 adult male Swiss albino mice (25-34 g) were used in the present experiment and were randomly divided into three groups (n=20 mice/group). Group (A) was a control. Groups (B) and (C) were exposed to intermittent water restriction during the 45 days of the experiment. Mice were euthanized and their kidneys were dissected for histopathological and immunohistochemical analysis.

Results: The results showed that E-cadherin proteins expression was significantly reduced ($p < 0.001$) in renal tissue of water restriction animals than well hydrated animals which had appeared as dark brown coloration of DAB staining with anti-Ecadherin IHC. These results were assessed by application of Aprio scope image software program for detecting strong positive results of the IHC E-cadherin protein expression in renal tissue of male mice.

Conclusion: Intermittent water restriction reduces the levels of E-cadherin in renal tissue, indicating impairment of the epithelial integrity of the nephron. These data suggest that dehydration might be involved in early renal structural damage and in the progression of kidney disease.

1. INTRODUCTION

Water is necessary for normal physiological function and for the maintenance of cellular homeostasis. Water balance, or hydration of the organism , is an important factor for the performance of many functions of the body[1]. Resting adult men need about 2.5 L of water a day, and about 3.2 L when doing light exercise. People who are very active in warm climates need about 6 L per day [2]. Mice: 15 ml water/100 g body weight/day. For example, a 25 gram adult mouse needs 25 grams x 15 ml/100 grams = 3.75 ml/day [3]. The kidneys are a key regulator of body water balance, with the ability to adjust water reabsorption and urine concentration according to hydration status . The daily output of urine is 1.5 L, typically 1% of the original volume of the filtrate [4]. There is increasing evidence that inadequate fluid intake and recurrent episodes of dehydration may contribute to renal dysfunction and the etiology and progression of chronic kidney disease (CKD) [5]. Experimental and epidemiological data have shown that long-term or repeat water restriction leads to structural and functional changes in renal tissue, including tubular injury, interstitial changes and renal functional impairment.

The kidneys are retroperitoneal organs, and are located laterally to the vertebral column between T12 and L3, but their position is influenced by body position, respiration and anatomy [6, 7]. The liver displaces the right kidney downward relative to the left. The renal hilum is located on the medial surface and is the entry and exit for blood vessels, nerves, lymphatics and the renal pelvis [8]. Most of nephron components including renal corpuscles, proximal and distal convoluted tubules are in the renal cortex outer region of the kidney [9]. The nephron is the functional and structural unit of the kidney [10]. Each kidney contains about two million nephrons [8]. The renal corpuscle consists of a glomerular capillary tuft surrounded by Bowman's capsule supported by mesangial cells, glomerular basement membrane and podocytes [10].

The proximal convoluted tubule is composed of simple cuboidal epithelium with a brush border of microvilli. It serves to reabsorb around 60-65% of filtered water and sodium chloride [11]. The loop of Henle is the basis for the countercurrent multiplier system that establishes the medullary osmotic gradient [12]. It consists of descending and ascending limbs with different thin and thick segments [13]. The distal convoluted tubule is composed of cells with abundant mitochondria but lacking a defined brush border and is involved in the fine-tuning of urine by sodium and chloride reabsorption, potassium secretion and further urine dilution [14, 11].

The nephron functions normally only if the integrity of the renal tubular epithelium, maintained by specialized intercellular junctions [15] . One of these junctions is the adherens junctions, which are important for the maintenance of epithelial architecture and tissue stability [16]. Cadherins are a family of calcium dependent transmembrane glycoproteins which mediate cell-cell adhesion. E-cadherin is one of the most important members of this family .E-cadherin is a key component of adherens junctions in epithelial tissues and is a highly conserved adhesion molecule. E-cadherin maintains cohesion in epithelium, polarity and structural organization via calcium dependent interactions between adjacent cells [17]. In the kidney E-cadherin is mainly expressed in distal tubular segments and collecting ducts, where it is involved in maintenance of tubular integrity and normal epithelial function . The extracellular region of E-cadherin extends from the cell surface to interact with cadherins on neighboring cells and contains an intracellular region with binding sites for the interaction with catenins and other regulatory proteins [18]. Changes in E-cadherin expression are associated with epithelial damage, loss of tissue architecture, and reduced renal function. Down-regulation of E-cadherin is regarded as a hallmark of epithelial-mesenchymal transition (EMT), a process characterized by loss of epithelial characteristics and gain of mesenchymal properties. EMT has been associated with renal fibrosis and progression of chronic kidney disease[19]. During EMT, cleavage of E-cadherin results in instability of adherens junction and the release of β-catenin, a transcriptional activator for cell proliferation [20].

E-cadherin is necessary for correct spatial patterning of the tubular epithelial cells, which is critical for efficient solute transport, selective permeability and nephron function. Classical cadherins, mainly E- and N-cadherins, are Ca2+-dependent ionic transmembrane cell adhesion molecules, primarily localized at adherens junctions. [21]. Disruption of E-cadherin mediated adhesion may lead to loss of epithelial integrity and altered tissue architecture [22]. E-cadherin downregulation is involved in pathologic modifications of renal tissue, cell-cell adhesion and epithelium integrity. Alterations in E-cadherin expression have been associated with epithelial damage, tubular dysfunction and histological abnormalities, suggesting its importance as a marker of renal epithelial health and maintenance of structure [23]. Thus, E-cadherin has emerged as a useful biomarker for assessing renal epithelial integrity and morphological preservation. E-cadherin maintains the epithelial layers and prevents cellular dissociation via the calcium-dependent intercellular bonds, which results in the preservation of the organized morphology necessary for normal physiological function [24].

The negative impact of dehydration and water deprivation on renal functions has been increasingly recognized, but little is known regarding the effects of these factors on E-cadherin expression in renal tissue. Therefore, the present study was designed to evaluate the effect of intermittent water restriction on the immunohistochemical expression of E-cadherin in the kidneys of male mice and its possible effect on renal epithelial integrity.

Aim of this study:

is to evaluate the effects of interrupted water restriction on the kidney tissue of male mice by Evaluation of the immunohistochemical expression of E-cadherin .

2.MATERIAL AND METHOD

2.1 Mice and housing

Sixty adult male Swiss albino mice with body weights ranging from 25 to 34 g were used in this experiment. Mice were housed in plastic cages at the Animal Care Facility, College of Medicine, Al-Nahrain University, following standard laboratory housing guidelines. The cages were constructed from non-toxic materials, designed with rounded corners, and could be easily cleaned. Each cage contained five mice. All animals were fed a standard pellet diet freely. To restrict water intake, water bottles were removed for all groups except the control group, which had free access to tap water. The animals were housed in a regulated setting with a 14-hour light and 10-hour dark cycle at ambient temperature in a clean, well-ventilated space.

2.2Animal Grouping and Experimental Water Restriction Protocol in Mice

The experimental animals were randomly divided into three groups. Groups A and B each contained 20 mice, and Group C was considered the control group and contained 20 mice to provide a reliable baseline for comparison. The groups were classified as follows:

1. Group A (Control Group): This group was maintained under same environmental conditions, with free access that the normal daily water consumption of mice (15 mL/100 g body weight) [3] to standard pellet diet and tap water for 45 days..

2. Group B: Each mouse was given 1.5 mL of water per day by oral gavage, divided into two doses of 0.75 mL each, for 45 days, with intermittent water restriction .Food was provided ad libitum in the form of a standard pellet diet.

3. Group C: Each mouse was given 1.5 mL of water per day by oral gavage, divided into three doses of 0.5 mL each, for 45 days, with intermittent water restriction. Food was provided ad libitum in the form of a standard pellet diet.

2.3 Experimental protocol

2.3.1 Standard Procedure for the Preparation of Paraffin-Embedded Tissue Blocks:

The kidney tissue specimens undergo processing for paraffin embedding using a series of conventional histological techniques. The processes included tissue fixation, dehydration, clearing, paraffin infiltration, embedding, sectioning, deparaffinization, staining, and final mounting. Each process was done in sequence order to ensure the generation of high-quality paraffin sections suitable for microscopic analysis [25] .

2.3.2 Immunohistochemical Reaction:

2.3.2.1 Immunohistochemical Detection of E-Cadherin in Renal Tissue:

Polyclonal antibody against E-cadherin (IgM) specific for mouse E-cadherin protein, a junctional complex protein, was used. The immunohistochemical staining procedure was performed according to the instructions provided by the manufacturing Sigma-Aldrich is based in Germany, while the staining kit was provided by Abcam, located in the United Kingdom.

2.3.2.2 The components of the Immunohistochemical detection kit:

• 5 mL of hydrogen peroxide blocking solution

• 5 mL of protein blocking reagent

• 5 mL of antibody amplifier

• 5 mL of goat anti-rabbit horseradish peroxidase (HRP) polymer conjugate

• 1 mL of DAB chromogen

• 5 mL of DAB substrate solution

2.4 Microscopic Examination and Imaging :

Kidney tissue sections stained with (H&E) , and immunohistochemically stained with DAB to illustrate E-cadherin expression were microscopically assessed using a Leica DM750 light microscope (Germany). Representative microscopic fields were digitally captured using an MC500 microscope camera (5-megapixel resolution), and the obtained pictures were saved in JPEG format. All microscopic analyses and image capture processes were performed at the Anatomy Laboratories, Department of Anatomy, College of Medicine, Al-Nahrain University.

2.5 IHC Reaction Assessment Using the Aperio ImageScope Program

The Aperio ImageScope V12 Positive Pixel Count method was used to quantitatively measure the intensity of particular staining in tissue sections, facilitating the assessment of protein markers including E-cadherin . This image processing method has been extensively used in prior histopathological and immunohistochemical studies [26] [27]. The program categorized staining strength into specific color classifications: strong positive staining was denoted by brown, positive staining by orange, weak positive staining by yellow, negative staining by blue, and white regions signified the absence of tissue in the examined segment.

2.6 statistical analysis of the data:

The current study's data were analyzed using version 22 of the Statistical Package for Social Sciences (SPSS) software. A one-way analysis of variance (ANOVA) was used to assess statistical significance and to compare the mean ± standard deviation and error between the control group and the water restriction groups. This test was used to assess the significance of mean differences across the examined groups. The positive pixel count of immunohistochemical reactivity for E-cadherin protein expression in renal epithelial cells was assessed across the experimental groups. p-value below 0.05 was considered a statistically significant.

3. RESULT

They showed a significant decrease ($p < 0.001$) in E-cadherin protein expression in the renal tissue of water-restricted animals than those of control group (A) that had been appear as dark brown coloration of DAB staining with anti-cadherin IHC at magnification of (40X) Figure (4-1) , (4-3) & table (4-1) . These results were evaluated by applying Aprio scope image software program for detecting strong positive results of the IHC E-cadherin protein expression in renal tissue of male mice (Fig 4-2).

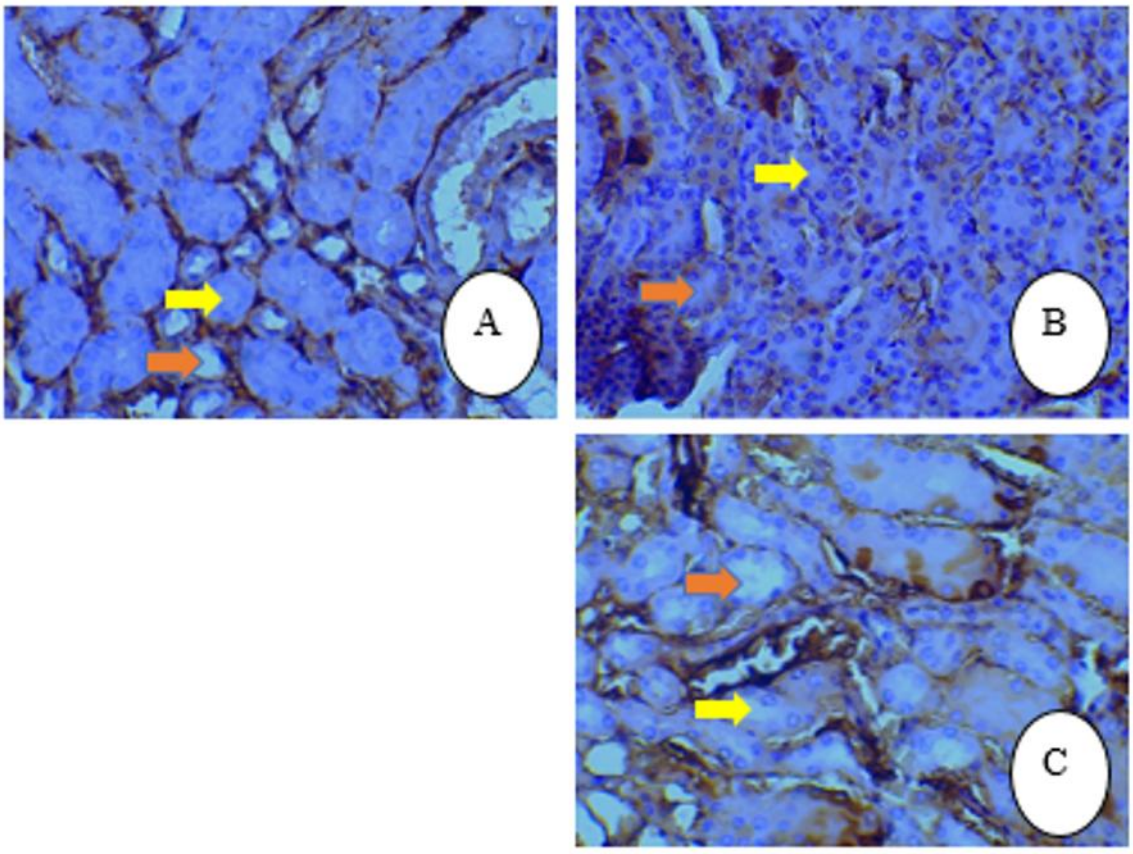

Figure (4-1) : reveal E cadherin proteins expression in renal tissue of animals with water restriction that had been appear as dark brown coloration staining with anti-cadherin IHC for group A (control), group (B) for low water intake twice daily, & group (C) for low water intake three time daily, PCT (yellow arrow), DCT (red arrow) at magnification of (40X).

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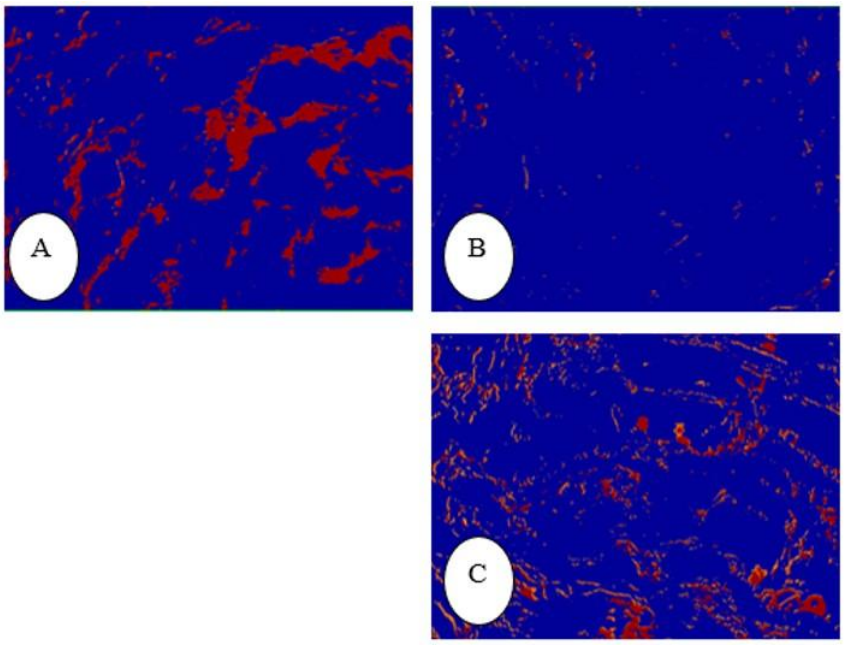

Figure (4-2) : reveals E cadherin proteins expression of renal tissue in animals with water restriction that were evaluated by applying Aprio scope image software program for detecting strong positive (appear red color) , positive (appear orange color), weak positive (appear yellow color), negative (appear blue color) for group A (control), group (B) for low water intake twice daily, & group (C) for low water intake three time daily, at magnification of (40X).

IHCcxCAD

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum
					Lower Bound	Upper Bound	
1.00	20	354.6765	128.91821	28.82699	294.3409	415.0121	119.94
2.00	20	56.5020	39.05106	8.73208	38.2255	74.7785	14.42
3.00	20	134.0110	56.61719	12.65999	107.5133	160.5087	44.87
Total	60	181.7298	151.99523	19.62250	142.4653	220.9944	14.42

(I) GROUP	(J) GROUP	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
1.00	2.00	298.17450*	26.67733	.000	233.9777	362.3713
	3.00	220.66550*	26.67733	.000	156.4687	284.8623
2.00	1.00	-298.17450*	26.67733	.000	-362.3713	-233.9777
	3.00	-77.50900*	26.67733	.014	-141.7058	-13.3122
3.00	1.00	-220.66550*	26.67733	.000	-284.8623	-156.4687
	2.00	77.50900*	26.67733	.014	13.3122	141.7058

*. The mean difference is significant at the 0.05 level.

Table (4-1) : reveals significance ($p < 0.001$) as diminish in E cadherin proteins expression in renal tissue of animals with water restriction groups (2,3) than those of control group (1) that had been appear in staining with anti-cadherin IHC

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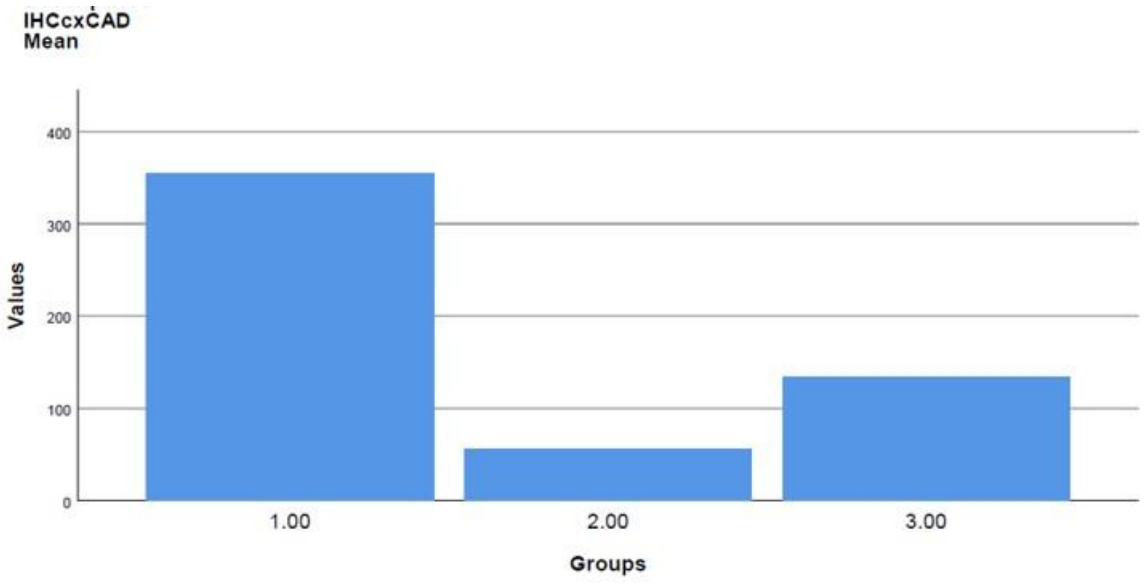

Figure (4-3) : Bar chart reveals significance ($p < 0.001$) as diminish in E cadherin proteins expression in renal tissue of animals with water restriction groups (2,3) than those of control group (1) that had been appear in staining with anti-cadherin IHC .

4. DISCUSSION

The present experiment reveals that e cadherin were affected by water restriction that were seen in tubular part of nephrons and this agree with other authors who found renal epithelial polarity is developed and maintained by the Ca2+-dependent cell adhesion molecules known as classical cadherins, such as E- and N-cadherin. Numerous cadherins are found in the kidney and are expressed differently in different nephron segments, according to these research. These investigations demonstrated that whereas E-cadherin is only very weakly present in the proximal tubule, it is abundant in the distal tubule, collecting duct, and most medullary segments. They came to the conclusion that E cadherin is expressed differently in the rat kidney's proximal and distal tubules, and they suggested that variations in cadherin expression and localization could explain why different nephron segments are more or less susceptible to renal pathology or nephrotoxic injury [28] .The present experiment reveal significance diminish in E cadherin proteins expression in renal tissue of animals with water restriction than those of control well hydrated animal group this agree with other researchers who found that A dehydrating environment may impact other organs, such as the skin, by injuring the epithelial cells, which disrupts the epithelial barrier function and leads to severe scarring. Their research indicated that subjecting keratinocytes to a dehydrated environment induces epithelial-to-mesenchymal transition (EMT), characterized by diminished caveolin-1 expression was associated with reduced levels of E-cadherin, which plays a role in the prevention and treatment of hypertrophic scars [29]. Fibrosis in the kidney results from the transformation of epithelial cells via the epithelial-mesenchymal transition (EMT) pathway, which facilitates fibrosis. Moreover, studies demonstrated that recurrent dehydration induces chronic kidney disease in both humans and animal models [30]. The current experiment reveals that dehydration constitutes a stressful condition, agree with further studY indicating that ischemic epithelial cells exhibit disruption of intercellular junctions and a deficiency in apical-basolateral protein polarity, which typically depends on the integrity of adherens junctions. They demonstrated a significant decrease in E-cadherin, the transmembrane protein associated with adherens junctions. The detected ischemia-induced deterioration of E-cadherin was not inhibited by any of the key proteolytic pathway inhibitors (i.e., proteasome, lysosome). Immunocytochemistry revealed the translocation of several proteins that usually comprise the adherens junction, including E-cadherin and β-catenin, from the lateral regions of the plasma membrane to intracellular sites. Moreover, immunoprecipitation using anti-E-cadherin and anti-β-catenin antibodies revealed that ATP depletion impaired normal E-cadherin-catenin interactions, resulting in the separation of α- and γ-catenin from E-cadherin and β-catenin complexes. The generation and maintenance of polarized epithelial cells depend on E-cadherin-mediated cell-cell adhesion and the proper functioning of adherens junctions; therefore, we suggest that the swift degradation of E-cadherin and the disintegration of adherens junctions are pivotal factors in the progression of the ischemic epithelial cell phenotype [31].The diminished in E-cadherin expression in mice with restricted water supply in present experiment were agree

with certain studies who recorded that Diabetic nephropathy (DN) is the primary cause of end-stage renal disease. They identify biomarkers for diabetic nephropathy in urine samples from groups of patients by doing comparative urinary analysis of E-cadherin. Immunological validation identified E-cadherin as only one marker capable of sufficiently differentiating between varying severities in DN. The predictive significance of E-cadherin has been established, highlighting a notable elevation in urine E-cadherin levels in cases of microalbuminuria and glomerular filtration rate (GFR) [32] .Diabetic nephropathy (DN) is a common complication of diabetes and the leading cause of chronic kidney damage. It is a progressive disorder marked by the steady decline of renal function over time. The role of Urine E cadherin as a biomarker for the early detection of dehydration in diabetic nephropathy. The urine E-cadherin was assessed in conditions of dehydration associated with diabetic nephropathy. E-cadherin is a novel biomarker for diabetic nephropathy, potentially used to evaluate the course of the condition. Consequently, assessing E-cadherin levels in diabetic patients may substantially influence the delay or prevention of end-stage renal disease. [33] .

LIMITATIONS

When interpreting the results, one must address some drawbacks of this research. First, the sample size was fairly small, and the study only used male mice. This might limit how well the results can be applied to larger groups that include females or other types of animals. Second, immunohistochemical analysis of E-cadherin expression was the central theme of the study without examining other molecular or functional markers of renal injury, including inflammatory mediators, oxidative stress, or renal function parameters. Third, the water restriction period and pattern were specific to this experimental design and may not be fully representative of all forms of dehydration occurring under physiological or clinical conditions. Furthermore, the study did not explore the reversibility of observed changes upon rehydration.

CONCLUSION

In conclusion, the results revealed a significant reduction in E-cadherin proteins expression in renal tissue of animals with water restriction than those of well hydrated animals that had been appear as dark brown coloration of DAB staining with anti-cadherin IHC. These results were evaluated by applying Aprio scope image software program for detecting strong positive results of the IHC E-cadherin protein expression in renal tissue of male mice.

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Ethical Approval

All experimental procedures involving animals were conducted in accordance with the institutional guidelines for the care and use of laboratory animals and complied with applicable ethical standards. The study protocol was reviewed and approved by the Research Ethics Committee of the College of Medicine, Al-Nahrain University, Baghdad, Iraq (Approval No. 20250902).

Author Contributions

Abdullah Miri Sahib contributed to the study conception and design, data collection, laboratory work, data analysis, interpretation of results, and manuscript drafting. Haider Abdulrasool Jaafar contributed to study supervision, methodology development, data interpretation, critical revision of the manuscript, and final approval of the submitted version. Both authors read and approved the final manuscript.

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