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Early Detection of Renal Damage Using Kidney Injury Molecule -1 (KIM-1) In Dogs Associated with Dehydration

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BACKGROUND: Dehydration may be associated with a declined kidney function and serious signs of kidney injury. The kidney response differently to dehydration levels which are not yet known. **Aim**: detection of the effect of dehydration on kidney functions levels and kidney injury biomarkers in dehydrated dogs and compare it to normal. **Material and methods**: A total number of 12 dogs were enrolled in this study (6 dehydrated and 6 controls). Inclusion criteria was presence of history of fluid loss for 48-hour, dehydration stage based on clinical assessment and the assessment of urine specific gravity. Blood samples were withdrawn from each animal on Ethylenediamine tetraacetic acid tube for hematology and plain tube to estimate total protein, albumin, BUN and creatinine. Ultrasonography was conducted on non-sedated dogs using a micro convex 5–8 MHz transducer. Urine samples were acquired through catheterization to determine urine specific gravity, then urine sediment was derived by centrifugation and commercially available Enzyme-linked immunoassay test was utilized to assess the levels of kidney injury molecule-1. **Results**: Hematological parameters showed non-significant elevation in packed cell volume in association with significant elevation in mean corpuscular volume were recorded in dehydrated dogs. Non-significant elevation in blood urea nitrogen, creatinine, and total protein were recorded in dehydrated dogs compared with control dogs. Urinalysis showed significant elevation in urine specific gravity and KIM-1 (P value ≤0.05) in urine samples of dehydrated dogs compared to control dogs. **Conclusion**, dehydration state might be impact the reading of urinary KIM-1 even in the absence of apparent azotemia.

Keywords: Dehydration, KIM-1, Dogs, Kidney injury.

Introduction

Water is an essential element in life, plays a pivotal role in maintaining physiological equilibrium in living organisms [1]. Dehydration a condition characterized by an inadequate amount of water in the body, poses a serious threat to various physiological functions, particularly those of the kidneys [2]. The canine kidney is a vital organ responsible for maintaining water balance, is particularly susceptible to the effects of dehydration [3]. Dehydration can result from various factors, including inadequate water intake, excessive fluid loss through vomiting or diarrhoea, or environmental conditions such as heat stress [4].

In recent years, the focus on understanding the mechanisms underlying dehydration-induced renal damage has grown, prompting investigations into specific biomarkers [5]. One such biomarker that has gained popularity is Kidney Injury Molecule-1 (KIM-1), a transmembrane protein expressed in renal tubular cells [6]. KIM-1, originally identified as a marker of tubular injury in humans, has been recognized as a

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promising indicator of renal damage in various species, including dogs [7]. The physiological significance of KIM-1 in the kidney is becoming more clear, KIM-1 confers on epithelial cells the ability to distinguish and phagocytose dead cells that are present in the post-ischaemic kidney and contribute to the obstruction of the tubule lumen that characterizes acute kidney injury (AKI) [8]. In a healthy kidney, KIM-1 is absent, though, after hypoxia, a huge number of epithelial tissues of renal proximal convoluted tubules can be identified by urine test. This elevated Kim-1 expression is an effective indicator for renal ischemia and hypoxic injury [7]. KIM-1 undergoes significant upregulation and incorporation into the apical membrane of the proximal tubule, and it remains present within the epithelial cell until the cell has fully recovered, establishing it as an excellent biomarker for identifying kidney injury [8]. Kim-1 was produced and secreted at an early stage of AKI when the pathological alterations were only mild; moreover Kim-1 concentration was directly proportional to the magnitude of renal injury, suggesting that the increased concentration was an indication of the prognosis of AKI [7].

The approval of KIM-1 by the US Food and Drug Administration (FDA) as an indicator of drug-induced kidney injury (DIKI) in rodent models [9] stems from its notable sensitivity and specificity as an early biomarker for proximal tubular injury. In the realm of human medicine, KIM-1, characterized as a transmembrane glycoprotein, is recommended as a responsive biomarker for acute kidney injury (AKI) due to its upregulation during instances of sudden injury [6].

Dehydration can lead to a cascade of events, including hypovolemia, reduced glomerular filtration rate, and alterations in renal blood flow, ultimately impacting the integrity of renal tubular cells [10]. KIM-1, being a sensitive biomarker, has been implicated in the early detection of renal injury [11], making it a potential candidate for assessing dehydration-induced kidney damage.

Owing to the presence of some similarities between canine and human renal physiology [12]. The canine model presents a unique opportunity to study dehydration-induced renal damage. Assessing KIM-1 levels in the urine of canine patients successfully identified tubular injury during the initial stages of non-azotemic infections [13].

Scanty papers discussed the relation between dehydration in canine patients and expression of urinary KIM-1, therefore, this study seeks to elucidate the specific impact of dehydration on KIM-1 in dogs.

Material and Methods

Ethical approval

This study was performed in accordance with the ethical standards of the institution Animal Care and Use Committee, faculty of Veterinary Medicine Cairo University, Egypt. This study was granted ethical Vet CU 25122023858

Animals and sampling

This study enrolled twelve (n=12) mongrel dogs, (8 males and 4 females), six (n=6) dehydrated dogs and six (n=6) control dogs. Dog's age range was between 2-4 years, weight between 15-20 Kg,

Inclusion criteria was presence of history of fluid loss for 48-hour caused by vomiting and diarrhea, on physical examination, patient showed rapid and weak pulses, dryness of mucous membranes and moderate loss of skin turgor, dehydration status was assessed based on physical and clinical examination to be moderate (7%), and the assessment of urine specific gravity.

Blood samples were withdrawn from each animal on Ethylenediamine tetraacetic acid (EDTA) tube for hematology and plain tube was used for serum separation to estimate total protein, albumin, Blood urea nitrogen (BUN) and creatinine using specified test kits (Spectrum-diagnostic, Egypt).

Urine samples were acquired through the catheterization technique illustrated by Taylor (2016) [14]. The refractometer was employed to determine the specific gravity of the urine. The urine sediment was derived by subjecting the samples to a 10-minute centrifugation at 2500 g, subsequently; 0.25 ml of each canine urine sediment was frozen at -80°C.

Following the collection of all samples, commercially available Enzyme-linked immunoassay (ELISA) test from BT LAB (E0147Ca, China) were utilized to assess the levels of KIM-1, the test was conducted in accordance with the manufacturer›s guidelines.

Ultra-sonographic examination

Ultrasonographic assessments were conducted on non-sedated dogs using the Esaote MyLabOne equipped with a micro convex 5–8 MHz

transducer. Dogs were positioned in right lateral recumbency for left kidney examination and left lateral recumbency for right kidney examination. Scanning below the transverse process of the first to third lumbar vertebrae was performed on both the left and right kidneys to identify any alterations in tissue structure.

Statistical analysis

Data are presented as mean ± SE, data set of control and dehydrated dogs were analyzed using Student T-test, P value ≤ 0.05 considered significant.

Results and Discussion

Hematological parameters are depicted in Table 1. There were no recorded changes in hemoglobin, MCHC and WBCs in dehydrated patients compared to control group. Non-significant elevation in PCV in association with significant elevation in MCV was recorded in dehydrated dogs compared to control dogs. For serum biochemistry, there was no recorded change in albumin. Non-significant elevation in BUN, creatinine, and total protein were recorded in dehydrated dogs compared with control dogs.

Non-significant elevation in packed cell volume (PCV) in association with significant elevation in mean corpuscular volume (MCV) were recorded in dehydrated dogs, the finding of this work were consistent with other reports [15] where there was no significant difference in PCV between euhydrated dogs and severely dehydrated one. Meanwhile, significantly higher MCV values was observed between this groups and these findings were attributed to dehydration ratio.

Non-significant elevation in BUN, creatinine and total protein were recorded in dehydrated dogs compared with control dogs (Table 1). For clinical detection of decreased Glomerular Filtration Rate (GFR), significant loss of nephrons and drops in overall kidney function are necessary, with assessments of up to 50% or more decline in function occurring before azotemia develops [16]. The traditional clinical kidney parameters serum blood urea nitrogen (BUN) and serum creatinine (Cr) were relatively insensitive in our study, which indicates the lower sensitivity and deprived predictive value of these conventional biomarkers at the early stage of kidney injury. The traditional diagnostic indicator serum creatinine was only significantly increased in case of severe kidney injury [7]. Recently, serum creatinine, the gold standard for testing AKI, has a number of drawbacks that impact the condition's early detection and prognosis.

Examination of urine sample showed significant elevation in urine specific gravity and KIM-1 in dehydrated dogs compared to control dogs as depicted in Table 2.

TABLE 1. **Hematobiochemical alterations in control and dehydrated dogs**.

Parameter	Control	Dehydrated dogs	P value
RBCs (10^6/µl)	6.143±0.3538	5.130± 0.3396	0.0980
Hb (g/dl)	13.30± 0.6557	14.63± 0.6060	0.2022
PCV (%)	39.77± 2.063	44.75±2.287	0.1812
MCV (fl)	69.74± 1.508	84.40± 4.992	0.0228
MCHC (g/dl)	33.80± 2.166	32.75± 0.60	0.6127
WBCs (10^3/µl)	7.233± 1.185	8.225± 0.770	0.4947
BUN (mg/dL)	9.333± 0.4216	14.71± 2.251	0.0781
Creatinine (mg/dL)	0.825± 0.0769	1.09± 0.123	0.1238
TP (g/dl)	6.488± 0.4351	7.245± 0.6410	0.3660
Albumin (g/dl)	3.574± 0.2007	3.429± 0.1171	0.5954

P value ≤0.05 is considered significant

TABLE 2. urine specific gravity and KIM-1 expression in dehydrated and control dogs.

Parameter	Control	Dehydrated dogs	P value
SP gravity	1.027±0.002	1.045± 0.001	0.0004
KIM-1 (ng/dl)	0.3167± 0.20	6.700± 0.549	0.0001

P value ≤0.05 is considered significant.

Urinalysis showed significant elevation in urine specific gravity and KIM-1 in urine samples of dehydrated dogs compared to normal control dogs. In order to improve the ability to predict the occurrence of AKI more and more efforts were made to discover novel urinary biomarkers prior to serum creatinine [17]. Euhydration is the state of having ideal body water content, and it is directly maintained by the kidneys. In human medicine; Dehydration is the process of losing body water; while hypohydration is the state of inadequate body water brought on by acute or chronic dehydration [18]. There is no universal gold standard assessment for dehydration so dehydrated state can be reliably identified by monitoring changes in two or more markers over time [19]. The kidneys get a significant amount of their cardiac output through the renal artery, which divides into numerous smaller arteries and arterioles before being filtered by the nephron, the kidney's functional unit [20]. The state of dehydration causes a cascade of hormonal responses to motivate water conservation including the activation of the renin-angiotensin- aldosterone system and the release of vasopressin [21]. One of the important consequences of increased plasma renin is the production of angiotensin II, Activating the angiotensin II results in many physiological actions that are important during dehydration [20]. Dehydration may be associated with a compromised kidney function and potential signs of kidney injury [22]. A case of dehydration resulting in lowering extracellular volume [23]. And It has been hypothesized reduced renal blood flow and circulatory blood volume have been proposed as potential causes of kidney attenuation, ischemic kidney stress, and even "temporary" kidney injury [24]. Acute kidney injury (AKI) is defined as a rapid reduction in kidney function (e.g., GFR), with increasing concentrations of nitrogenous waste products in the plasma (e.g., creatinine, urea) [25]. Prerenal cause of AKI is the most prevalent type of AKI, resulting from kidney hypoperfusion brought on by decreases in actual or effective arterial blood volume [26]. This hypoperfusion triggers the production of vasopressin, raises RSNA, and activates the renin-angiotensin-aldosterone pathway.

Urine specific gravity was high in the examined patients; increased urine specific gravity is due to an increased urine concentration because of dehydration. There is a decrease in renal blood flow and GFR owing to dehydration, and the renal vasculature develops an ischemia environment [20]. These results suggest that dehydration develops a state of kidney injury as a consequence of induced kidney stress.

In the present study, KIM-1 was elevated in urine sample without corresponding increase in serum creatinine. In the study performed by Lippi, et al., [27] they observed a fast elevation in urine KIM-1 2-hours following kidney injury and this elevation was sustained for up to 48 hours while serum creatinine did not start to rise except after 12 -24 hr post injury. Traditionally changes in kidney function are quantified via measures of GFR and urine output to identify the severity of AKI. Nowadays there are recent biomarkers that could identify kidney damage at an earlier stage. KIM-1 is a transmembrane glycoprotein that is not expressed in healthy group however expressed in urine in case of dehydration. During AKI, KIM-1 is mainly upregulated in proximal tubule cells, which is reflected by increases in urinary KIM-1 [28]. Thus, increases in urinary KIM-1 likely indicate proximal tubule injury, although the etiology is nonspecific. Therefore, KIM-1 might be a specific predictor for early AKI. The potential diagnostic and prognostic value of this biomarker needs to be validated further in the future. It has been hypothesized that In this case, the elevations in AKI biomarker are transient and of a lower magnitude than those observed in clinical situations. However, an increase in AKI biomarker in this non-clinical situation likely reflects an increased potential to develop AKI because some underlying pathophysiological processes taking place [29]. This explanation is compatible with the theory that small and transient increases in AKI biomarkers are indicative of acute kidney stress [30]. Accordingly, a higher risk of developing AKI is indicated by elevations in AKI biomarkers, and the degree of this risk is correlated with the elevations in AKI biomarkers.

Ultrasound examination showed non-significant changes between the dehydrated group and the control one.

Ultrasonography of kidney is considered a noninvasive diagnostic modality that is not dependent on kidney function and it has minimal side effects, it also allows depiction of renal internal architect (31). In the present study there were no alterations detected via ultrasound examination, Nelson and Couto (2019) (31), reported that the presence of normal ultrasound picture of kidney does not rule out the possibility of renal disorder.

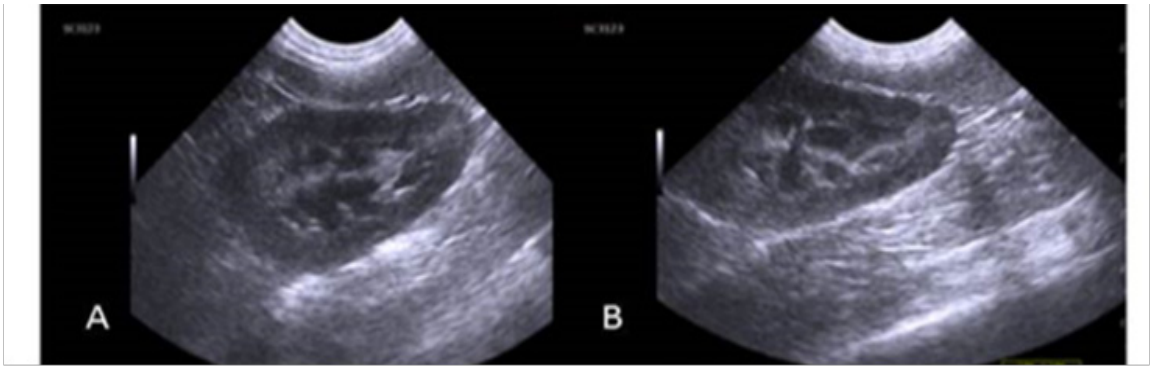

Fig. 1. A: Sagittal view in 2 years old male healthy dog: showed left kidney renal cortex has normal echogenicity when compared with the adjacent spleen and there is a clear demarcation between cortex and medulla. B: Sagittal view in 2 years old male dehydrated dog: showed left kidney renal cortex has normal echogenicity when compared with the adjacent spleen and there is a clear demarcation between cortex and medulla.

Conclusion

In conclusion, dehydration state might be impact the reading of urinary KIM-1 leading to its elevation, this elevation might be detected in earlier course of condition and before triggering elevation in traditional renal biomarkers (BUN and Creatinine).The increase in AKI biomarker in this non-clinical situation likely reflects the increased potentiality to develop AKI .

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Conflicts of interest

There are no conflicts to declare

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References

1. Hatcher, J. *Fluid Therapy—The Essentials*. *World Small Animal Veterinary Association Congress Proceedings* (2018) available at: https://www.vin.com/doc/?id=8896578.

2. Bartges, J. W. Chronic Kidney Disease in Dogs and Cats. *Veterinary Clinics: Small Animal Practice*, **34**(4), 867–885 (2004).

3. Lippi, I., Perondi, F., Gori, E., Pierini, A., Bernicchi, L. and Marchetti, V. Serum Bicarbonate Deficiency in Dogs with Acute and Chronic Kidney Disease. *Veterinary Sciences*, **10**(5), 363 (2023).

4. Taylor, A.J. and Kuhl, E.A. *EMS Canine Evaluation and Treatment of Dehydration*, (2023).

5. De Loor, J., Daminet, S., Smets, P., Maddens, B. and Meyer, E. Urinary biomarkers for acute kidney injury in dogs. *Journal of Veterinary Internal Medicine*, **27**(5), 998-1010 (2013).

6. Han, W.K., Bailly, V., Abichandani, R., Thadhani, R. and Bonventre, J.V. Kidney Injury Molecule-1 (KIM-1): a novel biomarker for human renal proximal tubule injury. *Kidney International*, **62**(1), 237-244 (2002).

7. Zheng, J.S., Zhu, T.T. and Ruan, H.R. Screening of early diagnostic markers of gentamicin-induced acute kidney injury in canines. *Journal of Veterinary Research*, **63**(3), 405 (2019).

8. Bonventre, J.V. Kidney injury molecule-1 (KIM-1): a urinary biomarker and much more. *Nephrology Dialysis Transplantation*, **24**(11), 3265-3268 (2009).

9. Harjen, H.J., Nicolaysen, T.V., Negard, T., Lund, H., Sævik, B.K., Anfinsen, K.P., Moldal, E.R., Zimmer, K.E. and Rørtveit, R. Serial serum creatinine, SDMA and urinary acute kidney injury biomarker measurements in dogs envenomated by the European adder (Vipera berus). *BMC Veterinary Research*, **17**, 1-13 (2021).

10. Basile, D.P., Anderson, M.D. and Sutton, T.A. Pathophysiology of acute kidney injury. *Comprehensive Physiology*, **2**(2), 303 (2012).

11. Wajda, J., Dumnicka, P., Kolber, W., Sporek, M., Maziarz, B., Ceranowicz, P., Kuźniewski, M. and Kuśnierz-Cabala, B. The marker of tubular injury, kidney injury molecule-1 (KIM-1), in acute kidney injury complicating acute pancreatitis: a preliminary study. *Journal of Clinical Medicine,* **9**(5), 1463(2020).

12. Grossman, R.C. Experimental models of renal disease and the cardiovascular system. *The Open Cardiovascular Medicine Journal*, **4**, 257-264 (2010).

13. Dias, C.S., Paz, L.N., Solcà, M.S., Portela, R.W.D., Bittencourt, M.V. and Pinna, M.H. Kidney Injury Molecule-1 in the detection of early kidney injury in dogs with leptospirosis. *Comparative Immunology, Microbiology and Infectious Diseases*, **76**, 101637 (2021).

14. Taylor, S. M. *Small Animal Clinical Techniques. 2nd edn.,. Elsevier Health Sciences.* (2016).

15. Atata, J.A., Esievo, K.A.N., Adamu, S., Abdulsalam, H., Adam, M., Chiroma, M.A. and Avazi, D.O. Haematological and biochemical parameters of clinically dehydrated and euhydrated dogs. *Sokoto Journal of Veterinary Sciences,* **16**(4), 73-78 (2018).

16. Cianciolo, R., Hokamp, J. and Nabity, M. Advances in the evaluation of canine renal disease. *The Veterinary Journal*, **215**, 21-29(2016).

17. Shao, X., Tian, L., Xu, W., Zhang, Z., Wang, C., Qi, C., Ni, Z. and Mou, S. Diagnostic value of urinary kidney injury molecule 1 for acute kidney injury: a meta-analysis. *PloS one*, **9**(1), e84131 (2014).

18. McDermott, B.P., Anderson, S.A., Armstrong, L.E., Casa, D.J., Cheuvront, S.N., Cooper, L., Kenney, W.L., O'Connor, F.G. and Roberts, W.O. National athletic trainers' association position statement: fluid replacement for the physically active. *Journal of Athletic Training*, **52**(9), 877-895 (2017).

19. Sawka, M.N., Burke, L.M., Eichner, E.R., Maughan, R.J., Montain, S.J. and Stachenfeld, N.S. American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine and Science in Sports and Exercise*, **39**(2), 377-390 (2007).

20. Chapman, C.L., Johnson, B.D., Parker, M.D., Hostler, D., Pryor, R.R. and Schlader, Z. Kidney physiology and pathophysiology during heat stress and the modification by exercise, dehydration, heat acclimation and aging. *Temperature,* **8**(2), 108-159(2021).

21. Sparks, M.A., Crowley, S.D., Gurley, S.B., Mirotsou, M. and Coffman, T.M. Classical renin-angiotensin system in kidney physiology. *Comprehensive Physiology*, **4**(3), 1201-1228(2014).

22. Bongers, C.C., Alsady, M., Nijenhuis, T., Tulp, A.D., Eijsvogels, T.M., Deen, P.M. and Hopman, M.T. Impact of acute versus prolonged exercise and dehydration on kidney function and injury. *Physiological Reports,* **6**(11), e13734 (2018).

23. González-Alonso, J. Human thermoregulation and the cardiovascular system. *Experimental Physiology*, **97**(3), 340-346 (2012).

24. Lippi, G., Schena, F., Salvagno, G.L., Tarperi, C., Montagnana, M., Gelati, M., Banfi, G. and Guidi, G.C. Acute variation of estimated glomerular filtration rate following a half-marathon run. *International Journal of Sports Medicine*, 948-951(2008).

25. Bellomo, R., Kellum, J.A. and Ronco, C. Acute kidney injury. *The Lancet*, **380**(9843), 756-766(2012).

26. Okusa, M.D., Portilla, D. Pathophysiology of acute kidney injury. *Brenner and Rector's The Kidney. Philadelphia*, *PA: Elsevier*; p. 906–939 (2019).

27. Lippi, I., Perondi, F., Meucci, V., Bruno, B., Gazzano, V. and Guidi, G. Clinical utility of urine kidney injury molecule-1 (KIM-1) and gamma-glutamyl transferase (GGT) in the diagnosis of canine acute kidney injury. *Veterinary Research Communications*, **42**, 95-100 (2018).

28. Ichimura, T., Hung, C.C., Yang, S.A., Stevens, J.L. and Bonventre, J.V. Kidney injury molecule-1: a tissue and urinary biomarker for nephrotoxicant-induced renal injury. *American Journal of Physiology-Renal Physiology*, **286**(3), F552-F563 (2004).

29. Schlader, Z.J., Hostler, D., Parker, M.D., Pryor, R.R., Lohr, J.W., Johnson, B.D. and Chapman, C.L. The potential for renal injury elicited by physical work in the heat. *Nutrients*, **11**(9), 2087 (2019).

30. Haroutounian, A., Amorim, F.T., Astorino, T.A., Khodiguian, N., Curtiss, K.M., Matthews, A.R., Estrada, M.J., Fennel, Z., McKenna, Z., Nava, R. and Sheard, A.C. Change in exercise performance and markers of acute kidney injury following heat acclimation with permissive dehydration. *Nutrients*, **13**(3), 841 (2021).

31. Nelson, R.W. and Couto, C.G. Small Animal Internal Medicine-E-Book: *Small Animal Internal Medicine-E-Book*. Elsevier Health Sciences (2019).

الكشف المبكر عن تلف الكلى الناجم عن الجفاف باستخدام جزيء إصابة الكلى (KIM-1) في الكلاب

نها محمود رياض[1] , ايمان شوقي رمضان[1] , محمد عوني الخياط[1] , نهي يوسف سالم[1] و ابراهيم عبد الستار صالح [1]

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الخلفية: قد يترافق الجفاف مع انخفاض وظائف الكلى وعلامات خطيرة لإصابة الكلى. استجابة الكلى بشكل مختلف لمستويات الجفاف التي لم تعرف بعد. **الهدف**: الكشف عن تأثير الجفاف على مستويات وظائف الكلى والمؤشرات الحيوية لإصابة الكلى في الكلاب المصابة بالجفاف ومقارنتها بالطبيعي. **المواد والطرق**: تم تسجيل 12 كلباً في هذه الدراسة (6 كلاب مجففة و6 كلاب مراقبة). وكانت معايير الاشتمال وجود تاريخ من فقدان السوائل لمدة 48 ساعة، ومرحلة الجفاف على أساس التقييم السريري وتقييم الثقل النوعي للبول. تم سحب عينات الدم من كل حيوان على أنبوب EDTA لأمراض الدم وأنبوب عادي لتقدير البروتين الكلي والألبومين وBUN والكرياتينين. تم الحصول على عينات البول من خلال القسطرة لتحديد الجاذبية النوعية للبول، ثم تم استخلاص رواسب البول عن طريق الطرد المركزي وتم استخدام اختبار ELISA المتوفر تجاريًا لتقييم مستويات KIM-1. **النتائج**: أظهرت مؤشرات الدم ارتفاعا غير ملحوظ في PCV بالتزامن مع ارتفاع كبير في MCV تم تسجيلها في الكلاب المجففة. تم تسجيل ارتفاع غير ملحوظ في نسبة BUN والكرياتينين والبروتين الكلي في الكلاب المجففة مقارنة بالكلاب الضابطة. أظهر تحليل البول ارتفاعًا ملحوظًا في الثقل النوعي للبول و KIM-1 (قيمة P ≥0.05) في عينات بول الكلاب المجففة مقارنة بكلاب المراقبة الطبيعية. **في الختام**، قد يكون لحالة الجفاف تأثير على قراءة البولية KIM-1 حتى في حالة عدم وجود آزوتيمية واضحة .

الكلمات الدالة : الجفاف ، الكلاب ، اصابات الكلي ، KIM-1 .