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The Effect of Analgesic Drugs (Voltaren, Profen and Panadol) on Kidney Function

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ABSTRACT

Overuse of analgesics, particularly readily available over-the-counter (OTC) medications like acetaminophen, aspirin, and other non-steroid anti-inflammatory drugs (NSAIDs), contributes to a worldwide problem of analgesic misuse in both developed and developing nations. NSAIDs are a necessary choice for managing pain because they target the cyclooxygenase enzyme (COX) pathway, which is central to both inflammation and pain signaling. They work by targeting two cyclooxygenase enzymes COX-1 and COX-2, which reduces the production of prostaglandins (PGs). The clinical utility of these agents is limited by their potential to induce adverse effects, notably acute kidney injury (AKI), which may contribute to the development of chronic kidney disease (CKD) in severe or prolonged cases. Renal function is susceptible to alteration by NSAIDs via inhibition of COX-1 (regulating renal hemodynamics and glomerular filtration rate) and/or COX-2 (mediating sodium and water excretion).

This study aimed to investigate the effect of analgesic medications, including Voltaren (diclofenac), Profen (ibuprofen), and Panadol (paracetamol/acetaminophen), on kidney function, specifically assessing creatinine, urea, and uric acid concentration. Sixty participants were enrolled in the study and divided into four groups of 15 individuals each: 15 individuals taking Voltaren, 15 individuals taking Profen, 15 individuals taking Panadol, and 15 individuals not using any analgesic medications, who served as the control group.

There was a significant increase in concentration of each of urea, uric acid, creatinine after four months of having the drugs that mention above compared with control group.

Keyword: Analgesic, diclofenac, non-steroid anti-inflammatory drugs.

INTRODUCTION

In 2020, the international association for the study of pain (IASP) describes pain as an unpleasant sensory and emotional experience associated with or similar to that resulting from potential or actual damage (Raja *et al*., 2020).

Pain perception results from a complex interplay of sensory neuron activation, electrochemical signal transmission along neural pathways, and central processing in higher brain regions, this experience is subject to modulation by stimulus characteristics (intensity, duration), emotional state, and contextual factors (Alorfi, 2023b). Analgesics, commonly referred to as painkillers, are pharmaceuticals employed to alleviate pain, these agents are broadly classified into two categories: opioid (narcotic) and non-opioid (non-narcotic), opioid analgesics, derived from opium alkaloids, are indicated for the management of pain ranging from mild to severe, these agents exert their analgesic effects by interacting with opioid receptors within the central nervous system, non-opioid analgesics encompass acetaminophen (paracetamol) and non-steroidal anti-inflammatory drugs (NSAIDs), these agents are readily available as over-the-counter (OTC) medications, their mechanism of action involves modulation of both peripheral nerve receptors and the central nervous system (Hanifah *et al*., 2020). NSAIDs constitute a pharmacotherapeutic class approved by the U.S. Food and drug administration (FDA) for their antipyretic, anti-inflammatory, and analgesic properties (Shekelle *et al*., 2016).The mechanism of action of NSAIDs, as determined by John Vane in 1960 through in vitro experimentation, is the inhibition of the cyclooxygenase (COX) enzyme, which plays a pivotal role in prostaglandin biosynthesis (Sohail *et al*., 2023).The metabolism of prostaglandin H2 yields five primary prostaglandins, among which are thromboxane A2(TXA2), a potent inducer of platelet aggregation and subsequent thrombus formation within platelets, and prostacyclin (PGI2), a vasodilator that exerts inhibitory effects on platelet aggregation within the endothelium, two distinct cyclooxygenase (COX) isoenzymes, designated COX-1 and COX-2, are commonly identified, COX-1 exhibits constitutive expression and plays a physiological role in gastric mucosal protection against gastric acid and in the biosynthesis of TXA2 by platelets, in contrast, COX-2 is predominantly inducible by inflammatory mediators in a broad spectrum of tissues and is associated with inflammatory processes; however, constitutive expression of COX-2 has been observed, contributing to physiological functions such as renal physiology, reproductive function, bone resorption, and neurotransmission (Kasturi *et al*., 2019). Voltaren active ingredient is diclofenac, which is an FDA-approved drug for managing acute and chronic pain associated with inflammatory musculoskeletal conditions, including osteoarthritis, rheumatoid arthritis, and ankylosing spondylitis (Tampucci *et al*., 2019). Diclofenac, an NSAID, is derived from phenylacetic acids (Alfaro, 2023). Diclofenac sodium yields analgesic, anti-inflammatory, and antipyretic effects (Zhang *et al*., 2024). It has been accessible to consumers as an over-the-counter (OTC) drug since its approval in 1988 (Elnashar *et al*., 2024).

Profen is a drug in which active ingredient is ibuprofen is and widely available over the counter pain reliever that is also effective at reducing inflammation and fever, it is classified as an NSAID and is derived from propionic acid (Zoubek *et al*., 2020). Ibuprofen is prescribed commonly in conditions like osteoarthritis, rheumatoid arthritis, juvenile idiopathic arthritis, and acutely painful musculoskeletal conditions (Ershad *et al*., 2024).

Panadol which is also an analgesic drug, and the active ingredients is paracetamol (Waris *et al*., 2017). It is known as paracetamol in many countries and it is common, widely available analgesic and antipyretic drug that has been in use for decades (Jaeschke and Ramachandran, 2024). As a safer analgesic, it is commonly used alone or in combination with other medications (Alhfidh and Othman, 2022). Paracetamol in adults is used for the management of various types of acute painful conditions that include headache, musculoskeletal pain, period pain, osteoarthritic pain, back pain, dental pain also for the management of postoperative pain (Ayoub, 2021). The medication works by inhibiting the production of prostaglandins in the central nervous system, by inhibiting their production in the central nervous system (CNS), the medication helps alleviate

pain, it's a good option for managing mild to moderate pain, in other words, it's a pain reliever, not an anti-inflammatory treatment (Alorfi, 2023a). NSAIDs are associated with adverse events affecting different organ systems they mediated inhibition of renal prostaglandin biosynthesis is a significant contributor to nephrotoxicity, the maintenance of glomerular filtration rate (GFR), a critical determinant of renal function, is dependent upon prostaglandin activity, especially under conditions of hypovolemia (Dhanvijay *et al*., 2013). Also play important role in the modulation of several renal functions (Abiola *et al*., 2019). Also, paracetamol toxicity doesn't just damage the liver; it also affects the kidneys and brain, two studies have demonstrated paracetamol's toxic effects on renal tissue (Shemiss, 2024).

The pharmacological mechanism of action is predicated on the inhibition of cyclooxygenase (COX) enzymes, which directly impacts prostaglandin biosynthesis, although hypertension, sodium and water retention, edema, and hyperkalemia are infrequent sequelae, these potential complications warrant consideration in susceptible patient populations, as they may predispose to the development of acute kidney injury (AKI) (Zokari, 2022). Additionally, these pharmacological interventions can elicit renal ischemia via the inhibition of renal prostaglandin biosynthesis, a process essential for the maintenance of afferent arteriolar vasodilation and thus, adequate renal perfusion, this inhibitory action engenders an elevation in vascular resistance within these arterioles, resulting in a consequential reduction in intraglomerular hydrostatic pressure, falling below physiological set points, this diminution of intraglomerular pressure directly impacts glomerular filtration rate (GFR), potentially predisposing the affected individual to the development of acute kidney injury (AKI) (Sivaraj and Umarani, 2018).

MATERIALS AND METHODS

Study area

This research was carried out in the Department of Biology/ Faculty of Science /University of Mosul, Iraq.

Experimental design

This study investigated 60 male subjects aged 30 to 60 years, with body weights ranging from 65 to 85 kg. The subjects were stratified into four cohorts of 15 individuals each, as follows:

1. Group 1 served as the control group and received no analgesic treatment.
2. Group 2 included patients who received daily Voltaren 25 mg.
3. Group 3 included patients who received daily Profen 1000 mg dose.
4. Group 4 included patients who received daily Panadol 1000 mg.

Biological samples were collected from the patients and their clinical status was monitored over a period of four months. Patients with chronic diseases such as hypertension, diabetes, and cardiovascular diseases were excluded from the study since

Sample collection

Approximately 3 ml of blood was drawn from each participant and collected in serum separator tubes (gel tubes). The tubes were allowed to clot at room temperature for 20 minutes. The samples were then centrifuged at 3000 rpm for 15 minutes to obtain serum, which was then used for the required analysis.

Statistical analysis

To analyze the results of the tests and determine the mean and standard deviation, one-way analysis of variance (ANOVA) was performed to determine the mean and standard deviation in a completely randomized design (CRD). The study aimed to identify differences among the groups of analgesic users receiving treatment at different intervals, compared to the control group. To determine these intergroup differences, Duncan's multiple range test was used for all studied variables. Differences were considered statistically significant at a probability level of $P<0.05$. Statistical analysis was performed using the statistical analysis system. Software to calculate the mean and standard deviation (Hilton, 2014).

RESULTS AND DISCUSSION

There are 60 patients, and values are presented as mean standard deviation. At the probability level ($P \leq 0.05$), shapes and various letters indicate a significant difference.

Fig. (1) demonstrate significant increase in urea concentration in the group of patients receiving Voltaren at a dose of 25 mg/kg body weight compared to the other groups ($P \leq 0.05$). The mean ± standard deviation for the Voltaren group was (65.42 ± 0.38)mg/100 ml. The mean ± standard deviation for the Profen group 1000 mg/kg body weight was (52.39 ± 0.54)mg/100 ml. The mean ± standard deviation for the Panadol group 1000 mg/kg body weight was (40.28 ± 0.07) mg/100 ml, and the control group had a mean ± standard deviation of (30.15 ± 0.02)mg/100 ml.

Fig. (2) demonstrate significant increase in uric acid concentration in the group of patients receiving Voltaren at a dose of 25 mg/kg body weight compared to the other groups ($P \leq 0.05$). The mean ± standard deviation for the Voltaren group was (11.45 ± 0.65)mg/100 ml. The mean ± standard deviation for the Profen group 1000 mg/kg body weight was(9.66 ± 0.27)mg/100 ml. The mean ± standard deviation for the Panadol group 1000 mg/kg body weight was (7.20 ± 0.06)mg/100 ml, and the control group had a mean ± standard deviation of (6.88 ± 0.03)mg/100 ml.

Fig. (3) demonstrate significant increase in creatinine concentration in the group of patients receiving Voltaren at a dose of 25 mg/kg body weight compared to the other groups ($P \leq 0.05$). The mean ± standard deviation for the Voltaren group was (119.77 ± 0.61)mg/100 ml. The mean ± standard deviation for the Profen group 1000 mg/kg body weight was (101.66 ± 0.59)mg/100 ml. The mean ± standard deviation for the Panadol group 1000 mg/kg body weight was (80.24 ± 0.10)mg/100 ml, and the control group had a mean ± standard deviation of (65.04 ± 0.37)mg/100 ml.

The medications under investigation (diclofenac, ibuprofen, and paracetamol) were used to manage localized pain symptoms, namely back pain, neck pain, and headache, which are non-systemic and non-inflammatory conditions in otherwise healthy individuals, scientific literature does not support any direct pathophysiological connection between these localized pain conditions and renal impairment, therefore these conditions are not independently capable of altering renal markers such as serum creatinine, blood urea nitrogen (BUN), or uric acid levels, the underlying conditions are localized, not systemic, the medical conditions that prompted medication use in this study are musculoskeletal or neurologically localized in nature (Hayashi *et al*., 2021).

The observed statistically significant increase in urea, uric acid, and creatinine concentrations in the group of patients receiving Voltaren at a dose of 25 mg/kg body weight after four months may be attributed to the drug's known side effect of reducing renal prostaglandin synthesis (Sivaraj and Umarani, 2018). Where Prostaglandins play a crucial role in maintaining the glomerular filtration rate (GFR), which is of paramount importance as prostaglandins regulate various renal functions. Specifically, PGE2 is considered a tubular prostaglandin affecting the renal tubules, while prostacyclin (PGI2) is a vascular prostaglandin playing a vital role in regulating renal vascular tone (Abiola *et al*., 2019). The mechanism may involve the action of diclofenac sodium (DS), the active ingredient in Voltaren, inhibiting cyclooxygenase (COX) enzymes, thereby affecting prostaglandin synthesis, this compound participates in various physiological processes within the kidney, such as glomerular filtration, tubular transport, and renin secretion, while associated with hypertension, sodium and water retention and hyperkalemia (elevated potassium levels) are also potential adverse effects, which can be of particular concern for at-risk patients who may develop acute kidney injury (AKI) (Zokari, 2022). This explains the elevated concentrations of urea, uric acid, and creatinine observed in the patients.

Regarding ibuprofen, administered at a dose of 1000 mg/kg body weight, its effect was less pronounced than that of Voltaren, however, with cumulative use over four months, renal dysfunction developed, likely due to reduced prostaglandin synthesis, resulting in a deterioration of renal function as evidenced by elevated urea, uric acid, and creatinine concentrations, this is because the inhibition of renal prostaglandins, which are responsible for dilating the afferent

arterioles supplying the kidneys, leads to increased renal vascular resistance, this, in turn, reduces intraglomerular pressure below physiological levels, consequently decreasing the glomerular filtration rate (GFR) and potentially leading to acute kidney injury (AKI) (Sivaraj and Umarani, 2018). This can occasionally result in complications such as urinary retention and/or renal insufficiency, with the potential for the development of acute tubular necrosis (ATN) (Ershad *et al*., 2024).

Regarding the effect of Panadol (paracetamol/acetaminophen) on renal function, it was less pronounced than that of the two aforementioned drugs (Voltaren and Profen), however prolonged use of high doses, especially in individuals without pre-existing renal issues, increases susceptibility to acute kidney problems, continuous use of high doses can lead to acute damage to the proximal renal tubules, potentially causing acute kidney injury (AKI) and, in some cases, contributing to renal interstitial fibrosis and papillary necrosis (Park, 2020).

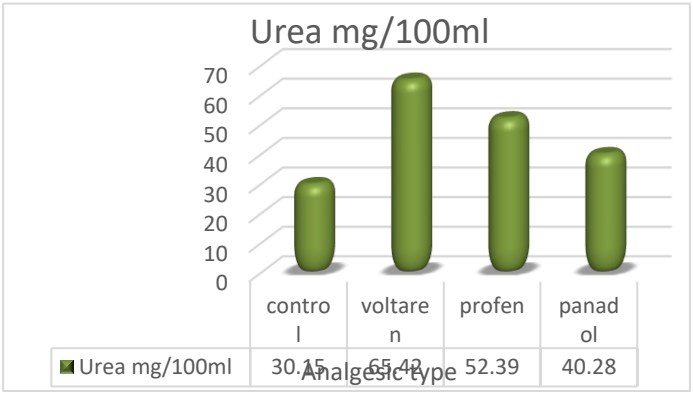

Fig. 1: The effect of analgesic drugs on urea concentration.

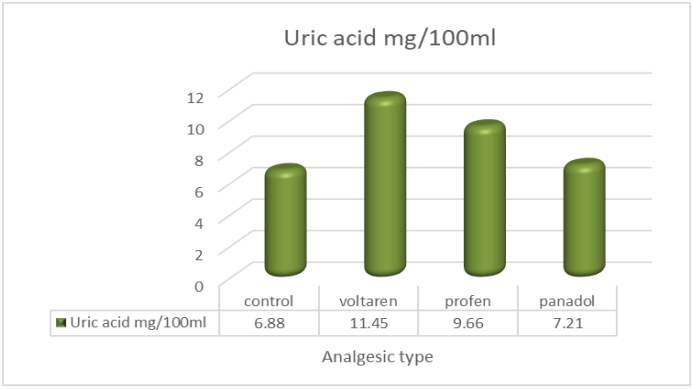

Fig. 2: The effect of analgesic drugs on uric acid concentration.

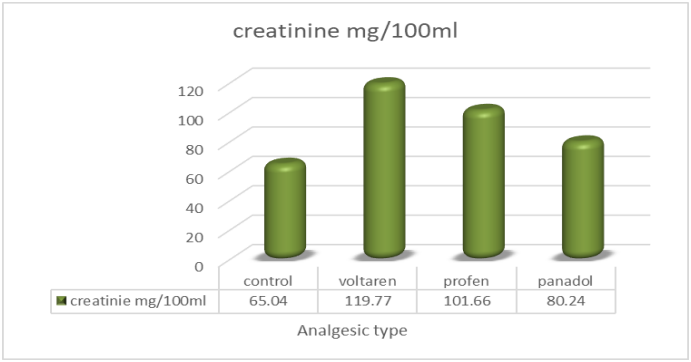

Fig. 3: The effect of analgesic drugs on creatinine concentration.

CONCLUSIONS

The observed renal effects are primarily attributable to the medications, not the underlying conditions, in the current study, participants were selected based on strict inclusion criteria that excluded individuals with chronic systemic diseases known to affect kidney function-such as diabetes, cardiovascular disease, or autoimmune disorders

We conclude that use of analgesic drugs over a period of four months led to a change in kidney function, as urea, uric acid, and creatinine all increased, and the effect of Voltaren was the strongest, followed by ibuprofen, and finally, Panadol.

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REFERENCES

Abiola, T.S.; Adebayo, O.C.; Babalola, O.O. (2019). Diclofenac-induced kidney damage in wistar rats: Involvement of antioxidant mechanism. *J. Biosci. Med*., **7**(12), 44-57. DOI:10.4236/jbm.2019.712005

Alfaro, R.A.; Davis, D.D. (2023). "StatPearls". Diclofenac. Treasure Island, FL.

Alhfidh, H.A.; Othman, N.S. (2022). Estimation of p-aminophenol via diazotization and coupling reaction with 4-chlororesorcinol-application on paracetamol. *Raf. J. Sci*., **31**(1), 57-68. DOI:10.33899/rjs.2022.172930

Alorfi, N.M. (2023a). Pharmacological methods of pain management. *Inter. J Gen. Med*.,16, 3247-3256. DOI:10.2147/IJGM.S419239

Alorfi, N.M. (2023b). Prescribing patterns and pharmacological knowledge of analgesics among community pharmacists in Jeddah, Saudi Arabia. *Bull. Pharm. Sci*., **46**(1), 647-657.

Ayoub, S. (2021). Paracetamol (acetaminophen): A familiar drug with an unexplained mechanism of action. *Temp.*, **8**(4), 51-371. DOI:10.1080/23328940.2021.1886392

Dhanvijay, P.; Misra, A.K.; Varma, S.K. (2013). Diclofenac induced acute renal failure in a decompensated elderly patient. *J. Pharm. Pharmac*., **4**(2), 155-7. DOI:10.4103/0976-500X.110916

Elnashar, A.A.; Kama, H.I.; Magdy, M.A.; Gamal, T.; Hamid, L.; Ragab, S.F.M.; Khodier, S.A.; Mohamed, A.; Kamel, M.M.; El-shahawy, A.A.; Dalia, A.; Yehia, Y. (2024). Review on diclofenac toxicities in different organs. *Ain Shams J. Fore. Med. Clin. Toxi*., **42**, 10-24. DOI:10.21608/ajfm.2024.333661

Ershad, A.M.A.; Chen, R.J.; Vearrier, D. (2024). "Ibuprofen Toxicity". StatPearls. Treasure Island. StatPearls Publishing.

Hanifah, R.A.; Ningrum, F.H.; Kresnoadi, E.; Wicaksono, S.A. (2020). The effect of paracetamol and codeine analgesic combination on serum glutamic oxaloacetate transaminase levels in male wistar rats. *Dip. Med. J*., **9**(2). DOI:10.14710/dmj. v9i2.27080

Hayashi, K.; Miki, K.; Kajiyama, H.; Ikemoto, T.; Yukioka, M. (2021). Impact of non-steroidal anti-inflammatory drug administration for 12 months on renal function. *Front Pain Res*., **2**, DOI:10.3389/fpain.2021.644391

Hilton, P.R. (2014). "Statistics Explained". 3rd ed., by Routledge, Printed in the USA. PP.85-125.

Jaeschke, H.; Ramachandran, A. (2024). Acetaminophen hepatotoxicity: Paradigm for understanding mechanisms of drug-induced liver injury. *Annu. Rev. Pathol*., **19**, 453-78. DOI:10.1146/annurev-pathmechdis-051122-094016

Kasturi, J.; Palla, P.P.; Bakshi, V.; Boggula, N. (2019). Non-steroidal anti-inflammatory drugs: An overview. *J. Drug. Delivery. Ther*., **9**(1), 442-448. DOI:10.22270/jddt. v9i1-s.2287

Park, W.Y. (2020). Controversies in acetaminophen nephrotoxicity. *Kid. Res. Clin. Pract*., **39**(1), 4-6. DOI: 10.23876/j.krcp.20.027

Raja, S.N.; Carr, B.D.; Cohen, M.; Finnerup, N.B.; Flor, H.; Gibson, S.; Keefe, F.J.; Mogil, J.S.; Ringkamp, M.; Sluka, K.A.; Song, X-J.; Stevens, B.; Sullivan, M.D.; Tutelman, P.R.; Ushida, T.; Vader, K. (2020). The revised international association for the study of pain definition of pain: Concepts, challenges, and compromises. *Inter. Associ. Stud. Pain*. **161**(9), 1976-1982. DOI: 10.1097/j.pain.0000000000001939

Shekelle, P.G.; Newberry, S.J.; FitzGerald, J.D; Motala, A.; O'Hanlon, C.E.; Tariq, A.; Okunogbe, A.; Han, D.; Shanman, R. (2016). Management of gout: A systematic review in support of an American College of physicians clinical practice guideline. *Ann. Inter. Med*., **166**(1), 37-51. DOI:10.7326/M16-0461

Shemiss, L.E. (2024). Role of spirulina platensis on some physiological aspects in paracetamolinduced subacute toxicity in male rats. *Raf. J. Sci*., **33**(2), 70-82. DOI:10.33899/rjs.2024.183427

Sivaraj, R.; Umarani, S. (2018). Diclofenac-induced biochemical changes in nephrotoxicity among male Albino rats. *Inter. J. Basis. Clin. Pharm*., **7**(4), 640-643. DOI:10.18203/2319-2003.ijbcp20181162

Sohail, R.; Mathew, M.; Patel, K.K; Reddy, S.A.; Haider, Z.; Naria, M.; Habib, A.; Abdin, Z.U.; Chaudhry, W.R.; Akbar, A. (2023). Effects of non-steroidal anti-inflammatory drugs (NSAIDs) and gastroprotective NSAIDs on the gastrointestinal tract: A narrative review. *Cur*., **15**(4), 37080. DOI:10.7759/cureus.37080

Tampucci, S.; Carpi, S.; Digiacomo, M.; Polini, B.; Fogli, S.; Burgalassi, S.; Macchia, M.; Nieri, P.; Manera, C.; Monti, D. (2019). Diclofenac-derived hybrids for treatment of actinic keratosis and squamous cell carcinoma. *Mole*., **24**(9), 1793. DOI:10.3390/molecules24091793

Waris, N.; Kausar, R.; Waris, N.; Naz, F. and Raza, A. (2017). Effect of extra panadol on blood and behavior of rats. *J. Bioana. Biomed*., **9**(4), 206-211. DOI:0.4172/1948-593X.1000179

Zhang, Y.; Yang, D.; Shuai, B.; Ding, H.; Yang, J.; Wang, J.; Tang, L.; Yao, S.; Zhang, Y. (2024). Diclofenac sodium nanomedicine results in pain-relief and differential expression of the RNA transcriptome in the spinal cord of SNI rats. *Inter. J. Pharm*., **659**, 124276. DOI: 10.1016/j.ijpharm.2024.124276

Zokari, A.O.H. (2022). Adverse effects of diclofenac sodium on kidneys in adult volunteers. *Elect. J. Uni*. *Aden Basic Appl. Sci*., **3**(1), 37-40. DOI:10.47372/ejua-ba.2022.1.143

Zoubek, M.E.; Lucena, M.L.; Andrade, R.J.; Stephens, C. (2020). Systematic review: Ibuprofen-induced liver injury. *Alimen. Pharm*., **51**(6), 603-611. DOI:10.1111/apt.15645

تأثير بعض العقاقير المسكنة (الفولتارين والبروفين والبنادول) على وظائف الكلى

براء جمعه طه **منتهى محمود داؤود**

قسم علوم الحياة/ كلية العلوم/ جامعة الموصل

الملخص

يُساهم الاستخدام المفرط للمسكنات، وخاصةً الأدوية المتاحة بسهولة بدون وصفة طبية مثل الأسيتامينوفين (الباراسيتامول)، الأسبرين، ومضادات الالتهاب غير الستيرويدية الأخرى (NSAIDs)، في مشكلة عالمية لسوء استخدام المسكنات في كل من الدول المتقدمة والنامية. تُعد مضادات الالتهاب غير الستيرويدية خيارًا ضروريًا للسيطرة على الألم لأنها تستهدف مسار إنزيم الأكسدة الحلقية (COX)، الذي يُعدّ محورًا لكل من الالتهاب وإشارات الألم. تعمل هذه الأدوية عن طريق استهداف إنزيمي الأكسدة الحلقية COX-1 وCOX-2، مما يُقلل من إنتاج البروستاكلانديات (PGs) ومع ذلك، فإن الفائدة السريرية لهذه العوامل محدودة بسبب قدرتها على إحداث آثار جانبية، وعلى رأسها إصابة الكلى الحادة (AKI)، التي قد تُساهم في تطور مرض الكلى المزمن(CKD) في الحالات الشديدة أو المطولة. تتأثر وظائف الكلى سلبًا بمضادات الالتهاب غير الستيرويدية عن طريق تثبيط COX-1 (الذي يُنظم ديناميكا الدم الكلوية ومعدل الترشيح الكبيبي) و/أو COX-2الذي يُوسط إفراز الصوديوم والماء.

هدفت هذه الدراسة إلى بحث تأثير أدوية المسكنات، بما في ذلك فولتارين (ديكلوفيناك)، وبروفين (إيبوبروفين)، وبنادول (باراسيتامول/أسيتامينوفين)، على وظائف الكلى، مع التركيز بشكل خاص على تقييم تركيز الكرياتينين، واليوريا، وحمض اليوريك. شملت الدراسة ستين مشاركًا تم تقسيمهم إلى أربع مجموعات، كل مجموعة تضم 15 فردًا: 15 فردًا يتناولون فولتارين، و15 فردًا يتناولون بروفين، و15 فردًا يتناولون بنادول، و15 فردًا لا يستخدمون أي أدوية مسكنة، وشكلت هذه المجموعة الأخيرة المجموعة السيطرة. واظهرت نتائج الدراسة زيادة كبيرة في تركيز كل من اليوريا، وحمض اليوريك، والكرياتينين بعد أربعة أشهر من استخدام الأدوية المذكورة أعلاه مقارنة بالمجموعة الضابطة.

الكلمات الدالة: المسكنات، ديكلوفيناك الصوديوم، مضادات الالتهاب غير الستيرويدية.