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CASE PRESENTATION

Regarding a case of acute renal failure induced by nonsteroidal anti-inflammatory drugs

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ABSTRACT

Introduction: acute kidney injury induced by nonsteroidal anti-inflammatory drugs is a frequent and potentially reversible complication when detected in a timely manner.
Objective: to describe the clinical presentation and evolution of a patient with acute kidney injury secondary to the use of anti-inflammatory drugs.
Case presentation: a 20-year-old male patient, originally from pichincha and residing in ambato, with a history of knee surgery and consumption of multiple analgesics and anti-inflammatory drugs, including Celecoxib and Ketorolac, is presented. He was admitted with abdominal pain, recurrent vomiting, and oliguria. Laboratory tests revealed elevated creatinine levels (3,25 mg/dl), increased urea, and electrolyte disturbances. Abdominal ultrasound showed findings suggestive of acute kidney injury. The medications were discontinued, and conservative management with hydration and fluid-electrolyte control was initiated. Clinical evolution was favorable, with progressive normalization of creatinine levels and recovery of renal function in the following weeks.
Conclusions: this case highlights the importance of early diagnosis and prompt discontinuation of anti-inflammatory drugs in patients with signs of kidney injury. It underscores the need for responsible prescribing and appropriate monitoring in at-risk patients, particularly those with preexisting comorbidities. Conservative management can reverse renal dysfunction and prevent progression to irreversible renal failure.

Keywords: Anti-Inflammatory Agents, Non-Steroidal; Drug-Related Side Effects and Adverse Reactions; Renal Insufficiency; Acute Kidney Injury.

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INTRODUCTION

Acute kidney injury (AKI) encompasses a set of potentially reversible kidney injury mechanisms, the degree of functional recovery of which may be complete, partial, or nonexistent, depending on the magnitude and duration of parenchymal damage. Currently, it constitutes a significant public health problem, associated with the high prevalence of risk factors and comorbidities in the general population, including hypertension, diabetes mellitus, and obesity. This is compounded by the widespread and, in many cases, indiscriminate access to analgesic and nonsteroidal anti-inflammatory drugs (NSAIDs), whose mechanism of action is based on the inhibition of cyclooxygenases 1 and 2 (COX-1 and COX-2), with the consequent decrease in prostaglandin synthesis, which confers a potential nephrotoxic effect.[1,2,3]

NSAIDs interfere with the arachidonic acid cascade by blocking the COX-1 and COX-2 pathways, leading to a significant reduction in prostaglandin production. From a renal perspective, prostacyclin, prostaglandin E2 (PGE2), and prostaglandin D2 (PGD2) are particularly relevant.[4,5] The decrease in these mediators is associated with vasoconstriction and renal ischemia, favoring the development of acute nephrotoxicity; additionally, PGE2 inhibition is related to sodium and water retention in the intravascular compartment.[6,7,8,9]

These mechanisms are explained by the physiological functions of cyclooxygenases in the kidney. COX-1 participates in hemodynamic regulation, promoting vasodilation of the afferent arteriole and maintaining glomerular filtration rate, while COX-2 is predominantly involved in sodium and water excretion. PGE2 induces natriuresis by activating tubular receptors located in the loop of Henle and collecting ducts, inhibiting the transport of sodium and chloride from the tubular lumen into the cells. It also exerts an antagonistic effect on antidiuretic hormone and generates negative feedback on the renin-angiotensin-aldosterone system (RAAS) and the sympathetic nervous system (SNS), contributing to the autoregulation and preservation of renal blood flow.[1]

Advanced age, chronic or abusive NSAID use, drug interactions, and the presence of comorbidities significantly increase the risk of NSAID-induced nephrotoxicity. Among the most relevant risk factors is systemic hypertension, a condition that exacerbates activation of the RAAS and the SNS, altering renal feedback mechanisms and predisposing to the development of AKI.[1,6,8]

Acute renal failure (ARF) is characterized by a decrease in the glomerular filtration rate, with retention of nitrogenous waste products, especially urea, and elevated serum creatinine, which typically increases at least 1,5 times compared to baseline values. Clinically, it may present with oliguria or anuria. Associated metabolic abnormalities include metabolic acidosis, which in advanced stages may be accompanied by altered mental status and seizures. In certain cases, hypoperfusion and acute renal ischemia can progress to acute tubular necrosis, manifesting as hypertension and pitting edema.[5,10,11,12]

Other presentations include acute interstitial nephritis and nephrotic syndrome. Although the pathophysiological mechanisms are not fully understood, the involvement of delayed hypersensitivity reactions, the diversion of arachidonic acid metabolism toward leukotriene production, and T-cell activation is postulated, which can lead to minimal change disease, characterized by significant proteinuria. Hydroelectrolytic disturbances are also common, notably hypernatremia, hyponatremia, and hyperkalemia.[13,14,15]

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The use of diagnostic criteria today has been established through scales or scores that allow quantification and staging of the degree of acute kidney injury, including RIFLE, AKIN, and KDIGO, each with its own particularities.[3,10,14] For its part, in imaging studies, ultrasound allows evaluation of increased echogenicity in acute parenchymal kidney injury, which is related to the size and contour of the kidney, vascular characteristics, and perfusion pattern; Doppler ultrasound can identify the state of the blood vessels; the use of renal biopsy is useful in the diagnosis of glomerulonephritis; magnetic resonance imaging and angiographic studies corroborate the presence of renal inflammation and tubular dysfunction.[3,11]

Medical treatment is primarily conservative in the context of acute renal failure; however, there are occasions when renal replacement therapy (dialysis) is required. It begins with the pharmacological withdrawal of NSAIDs, correction of the diet with low protein and sodium content, adequate caloric intake, optimal fluid balance, and immediate resolution of disorders such as hyperkalemia, hyponatremia, acute edema, and metabolic acidosis.[7,11]

CASE REPORT

A 20-year-old male patient, originally from the province of Pichincha, residing in the city of Ambato, single marital status, blood type O+, who goes to the Ambato General Teaching Hospital, comes to the emergency room due to repeated vomiting, abdominal pain located in the epigastrium and lumbar pain.

Regarding his personal medical history, he reports having had an anterior cruciate ligament rupture (ICD-10:M236) and a meniscus tear (ICD-10:M232). Referring to his surgical history, the patient reports having undergone arthroscopic anterior cruciate ligament and meniscus repair, for which he is currently taking cefuroxime, celecoxib, ketorolac, tramadol, and paracetamol, which were prescribed during surgery and at the time of discharge.

Physical examination

Weight: 66 kg; Height: 1,88 m; Blood Pressure: 119/80 mmHg; Heart Rate: 96 bpm; Urine Output: <1000 ml; Head: Normocephalic, no lesions; Eyes: Pink conjunctivae, antitecric sclerae, isocoric pupils, normoreactive to light and accommodation; Nose: Nasal pyramid without lesions, nasal passages patent, no secretions; Mouth: Dry mucous membranes, normal teeth, oropharynx not congested and not erythematous; Ears: Normal structure and implantation, external auditory canal patent, no secretions; Neck: Symmetrical, no lymphadenopathy, thyroid not palpable; Skin: Normal, dry; Heart: Heart sounds of normal pitch and intensity; Lungs: Vesicular breath sounds preserved, no abnormal sounds; Thorax: Preserved expansion, no masses or lymphadenopathy are palpable; Abdomen: Soft, depressible abdomen that follows the respiratory rhythm, normal bowel sounds on auscultation, normal tympany on percussion, abdomen painful on deep palpation at the level of the epigastrium, right and left hypochondrium; Extremities: Edema at the level of the right knee joint and the respective leg, pitting edema ++/+++; Lumbar region: Positive fist percussion.

Additional tests

Upon hospital admission (March 26, 2023), the complete blood count showed generally normal parameters. The white blood cell count was normal (7,64 × 10^3/mm^3), with slight relative neutrophilia (74,7 %) and mild lymphopenia (17,8 %), without evidence of anemia or thrombocytopenia. Hemoglobin (15 g/dL), hematocrit (44 %), and red blood cell indices were within normal ranges, ruling out active systemic infections or associated hematological abnormalities.

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Regarding the initial blood biochemistry, impaired renal function was evident. In the first determinations on March 26, 2023, serum creatinine was elevated (1,96 mg/dL), with progressively increasing values in subsequent tests on the same day (2,12 mg/dL), despite normal blood urea nitrogen and urea levels, suggesting an acute deterioration of glomerular filtration rate in the early stages. Amylase remained within normal parameters, ruling out associated pancreatic pathology.

Hours later, a new biochemical test (March 26, 2023) revealed a significant worsening of renal function, with a serum creatinine of 3,25 mg/dL, accompanied by hyperphosphatemia (5,6 mg/dL). Serum electrolytes (sodium, potassium, and chloride) remained within normal ranges, without any serious hydroelectrolytic disturbances at that time, which is consistent with acute kidney injury of functional origin induced by NSAIDs, in an advanced stage according to KDIGO criteria.

In subsequent follow-up visits, conducted on March 30, 2023, a partial improvement in renal function was observed, with a decrease in creatinine to 1,90 mg/dL and urea levels within normal limits. Serum electrolytes remained stable, and plasma albumin was normal (4,02 g/dL), indicating adequate nutritional status and the absence of nephrotic syndrome.

During the follow-up on April 4, 2023, creatinine continued to decrease to 1,07 mg/dL, with normalization of urea. However, mild hyperkalemia (5,4 mEq/L) was observed, without documented clinical repercussions, and slightly elevated sodium levels were also noted, findings that suggest a renal recovery process still in the tubular adjustment phase.

Finally, in the late follow-up on May 15, 2023, renal function parameters had completely normalized, with creatinine at 0,86 mg/dL and urea at 28,9 mg/dL. Serum electrolytes, including calcium, remained within physiological ranges, confirming the reversibility of the acute kidney injury after discontinuation of NSAIDs and the implementation of conservative management.

The imaging studies included a standard abdominal ultrasound, the details of which can be seen in Figure 1.

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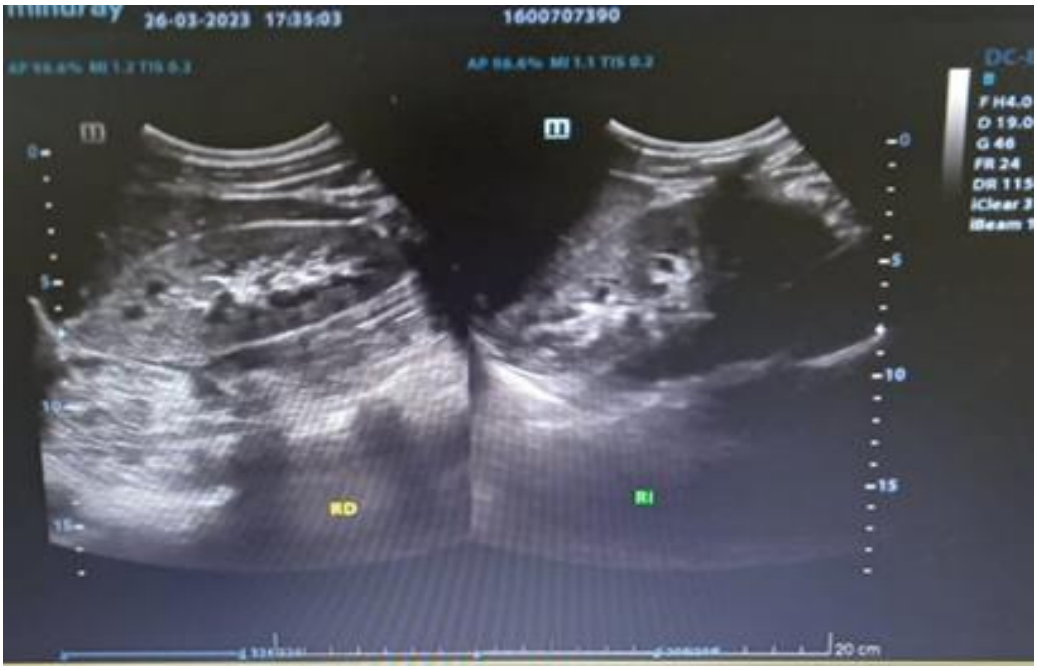

Fig. 1 Standard abdominal ultrasound.

DISCUSSION

A review of the clinical case and the scientific literature suggests that the indiscriminate use of NSAIDs is associated with multiple complications affecting renal function. These drugs can interfere with renal autoregulation mechanisms, particularly through prostaglandin inhibition, which reduces renal blood flow. Consequently, serious syndromes can develop, characterized by electrolyte imbalances, hypercreatininemia, and decreased urine output. If these manifestations are not identified early, they increase the risk of persistent or irreversible kidney damage.

Previous studies have described similar findings, showing that NSAID-induced reduction in renal perfusion favors the appearance of hydroelectrolytic alterations, hypercreatininemia and oliguria, clinical manifestations that can evolve into persistent renal damage if not identified early.[16,17] These results reinforce the need for strict monitoring of the use of these drugs, even in patients without known renal history.

Azevedo et al.,[9] point out that age is one of the main aggravating factors for acute kidney injury. This age group is particularly vulnerable not only due to the physiological changes associated with aging, such as the decrease in glomerular filtration rate and renal functional reserve, but also due to the high prevalence of painful chronic diseases such as arthritis, osteoarthritis, and synovitis. In addition, the frequent use of palliative therapies leads to prolonged and repeated use of NSAIDs, significantly increasing the risk of nephrotoxicity.

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Lucas et al.,[1] emphasize that NSAID-mediated kidney injury should be assessed on an individual basis, considering each patient's clinical context. This assessment is especially relevant in hospitalized patients, who often present with comorbidities, dehydration, or concomitant exposure to other nephrotoxic drugs. In this regard, there is a need to identify safer therapeutic alternatives within this pharmacological group, using minimum effective doses and relying on classification tools such as the RIFLE, AKIN, and KDIGO scales, which allow for timely, standardized, and comparative monitoring of renal function.

Finally, Godoy Villalva and colleagues,[4] emphasize the importance of directing future research toward identifying risk factors and genetic predisposition in young patients without prior kidney disease. They also highlight the need to further study biomarkers other than traditional blood urea nitrogen (BUN) in this age group, which could facilitate the early detection of acute kidney injury. Identifying these markers would improve predictive capacity regarding the clinical course and reversibility of kidney damage associated with NSAID use, thus optimizing prevention and management strategies.

Although this group is generally considered low risk, several studies have documented cases of severe AKI in young adults exposed to NSAIDs, particularly in contexts of dehydration, intense exercise, or unidentified individual susceptibility.[8] The exploration of genetic factors and the identification of early biomarkers other than traditional ones, such as NGAL or KIM-1, are emerging as promising lines of research to improve the early detection and prediction of the reversibility of kidney damage. These strategies could optimize the prevention and clinical management of NSAID-induced nephrotoxicity, with direct implications for clinical practice and patient safety.[18,19,20]

CONCLUSION

Acute kidney injury associated with nonsteroidal anti-inflammatory drug (NSAID) use is a clinical condition that, in its early stages, should be managed conservatively, with treatment tailored to the patient's age and the dosage ingested. In advanced stages, it may require renal replacement therapies such as peritoneal dialysis, hemodialysis, or transplantation. Early management promotes recovery of renal function and prevents further damage. Hypercreatininemia and decreased urine output are the main indicators of suspicion, with or without constitutional symptoms. In conclusion, this is a potentially reversible or irreversible condition, with cardiovascular, electrolyte, and biochemical manifestations that must be assessed on an individual basis, guiding physicians toward prudent and judicious management of this drug class.

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