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Renal injury associated with anabolic steroid use: a case series.

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Abstract

Introduction: The use of anabolic androgenic steroids (AASs) has been linked to a spectrum of kidney injuries ranging from acute kidney failure to end-stage chronic kidney disease. In these cases, not only the direct nephrotoxic effects of AAEs but also the impacts exacerbated by risky practices such as high-protein diets, multivitamin supplementation, and intentional dehydration are documented.

Clinical cases: Three patients consulted for emergencies at the Federico Lleras Acosta Hospital in Ibagué, Colombia. All patients received high doses of anabolic steroids associated with high-protein diets and creatine supplementation. All the participants were young men with good muscles, but none had a body mass index (BMI) ≥ 30 kg/m². The clinical events occurred in February, June, and August 2024. All patients had elevated creatinine levels, and the first clinical case presented with proteinuria in the nephrotic range. Microscopic urinalysis did not reveal glomerular hematuria, and the urine dipstick test was positive for protein and hemoglobin; immunoserology and viral serology were negative, and ultrasonography revealed chronicity.

Conclusions: AASs have direct toxic effects on the glomerulus and tubular system or can induce injury indirectly through adaptive mechanisms, hydroelectrolytes, and endocrine disorders. Evidence suggests that AAEs can cause direct kidney damage, including focal segmental glomerulosclerosis, acute interstitial nephritis, and rhabdomyolysis. In addition, high protein intake can increase the risk of kidney injury.

Keywords:

Anabolic androgenic steroids, rhabdomyolysis, acute kidney injury, high-protein diet.

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Anabolismally androgenic steroids (AASs) are mainly used to improve physical performance, and their use has grown exponentially, becoming a global abuse problem; most of the users are middle-aged men [1]. The chronic use of AAS has significant adverse effects, including alterations in carbohydrate and lipid metabolism, which increases the risk of cardiometabolic diseases [2].

Androgenic anabolic steroids include growth hormone, testosterone, and their hormonal derivatives. They are commonly used by high-performance athletes and amateur bodybuilders, in addition to high-fat-soluble and water-soluble vitamins and creatine supplementation. These are used to improve muscle strength, athletic performance, and endurance. Herlitz et al. [3] reported on 10 bodybuilders who developed kidney failure and focal segmental glomerulosclerosis (FSGS) while consuming anabolic steroids and protein and creatine supplements with a daily protein intake ranging from 300 to 550 g/day. High protein intake has been a cause for concern owing to increased glomerular filtration rates and is experimentally associated with glomerular hyperfiltration and FSGS.

Recent evidence suggests that anabolic steroids are directly toxic to the glomeruli and that segmental sclerosis is the result of the loss of podocytes mediated by apoptosis through an androgen receptor of podocytes [4,5]. It can also cause nephrocalcinosis, acute interstitial nephritis, and rhabdomyolysis [6 -7].

Case series

Three patients were consulted for emergencies at the Federico Lleras Acosta Hospital in Ibagué, Colombia. All patients received high doses of anabolic steroids associated with high-protein diets and creatine supplementation. All the participants were young men with good muscles, but none had a body mass index (BMI) ≥ 30 kg/m². The clinical events occurred in February, June, and August 2024.

Clinical case 1

Clinical history

A 38-year-old male patient with no known pathological history refers to a habit of intense physical training, consisting of two hours a day of lifting, six days a week, supplemented with jogging 2 to 3 times a week. High-protein food is a dietary habit. Concerning the medications that he has used for the last 5 years, he regularly consumes and injects anabolic steroids: intramuscular testosterone (4 mg/week) and oral trenbolone enanthate (80 mg every 15 days).

The patient was admitted with the following nephritic syndrome: hypertension, oliguria, and azotemia. The temporality of this case is presented in Figure 1. In the last 6 months, he had edema in the lower limbs and occasional foamy urine. Seven days before admission, she presented with intensified edema and asthenia, which made it impossible for her to perform physical activity, headache, and abdominal pain in her flanks. Upon admission, the body mass index was 28 kg/m2, and the patient presented with arterial hypertension of 150/90 mmHg and edema in the lower limbs with pitting +++. Cardiopulmonary and neurological examinations were regular. The patient presented with acne on the anterior and posterior chest.

Diagnostic workshop

Creatinine upon admission was 6 mg/dl (Table 1). The urine dipstick test was positive for protein and hemoglobin. Immunoserology and viral serology were negative. Renal ultrasound revealed pathology with findings of chronicity. A renal biopsy was performed, which was reported on the ninth day of the hospital stay, in which the presence of focal and segmental sclerosis was reported in the glomeruli, without deposits of IgA, IgG, IgM, C3, or light chains. The incidence of interstitial fibrosis and tubular atrophy (IF/TA) is greater than 60% (Figure 2).

Evolution

At 24 hours, renal replacement therapy was started due to oliguria and azotemia, with a creatinine level of 6 mg/dl. On Day 14 of the hospital stay, the patient was discharged in good general condition, with an indication for hemodialysis in the renal unit corresponding to the health provider entity, in addition to suspending the use of steroids, intense training, and protein supplements. Three months after the patient started hemodialysis, the patient continued to be dependent on renal replacement therapy, with no evidence of significant improvement in renal function.

Table 1. Clinical characteristics of the patients, duration of weight lifting activity, estimation of plasma creatinine and GFR by CKD-EPI, body mass index, proteinuria in 24 hours and serum albumin.

Case	Age	Sex	BMI	Creatinine	TFGe	Training activity	Proteinuria	Albumin
1	38	Man	28 k/m²	6 mg/dl	9 ml/min/1.73m²	12 hours/week	7.4 g/día	3.4 g/dl
2	37	Man	29 k/m²	5.8 mg/dl	11.6 ml/min/1.73m²	8 hours/week	6.5 g/día	3.6 g/dl
3	32	Man	28 k/m²	11 mg/dl	5.8 ml/min/1.73m²	10 horas/week.	1.2 g/día	3.8 g/dl

EGFR: Estimated glomerular filtration rate.

Figure 1. Timeline in case 1.

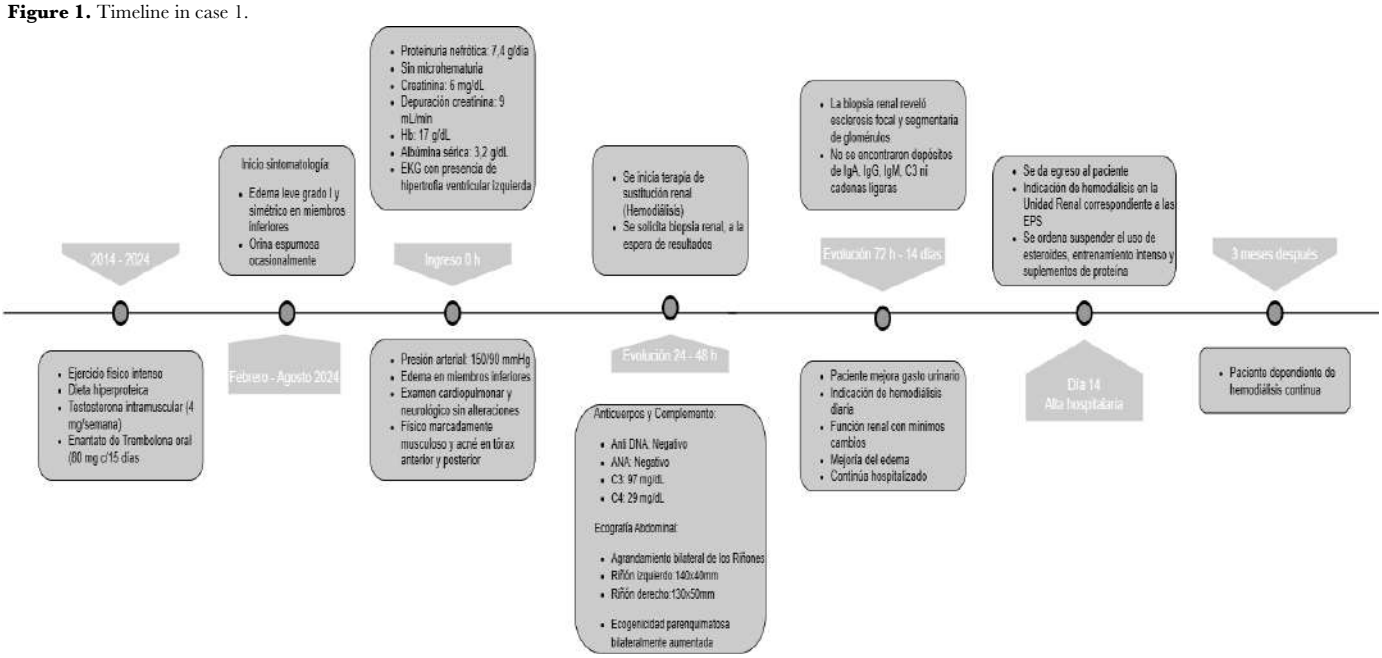
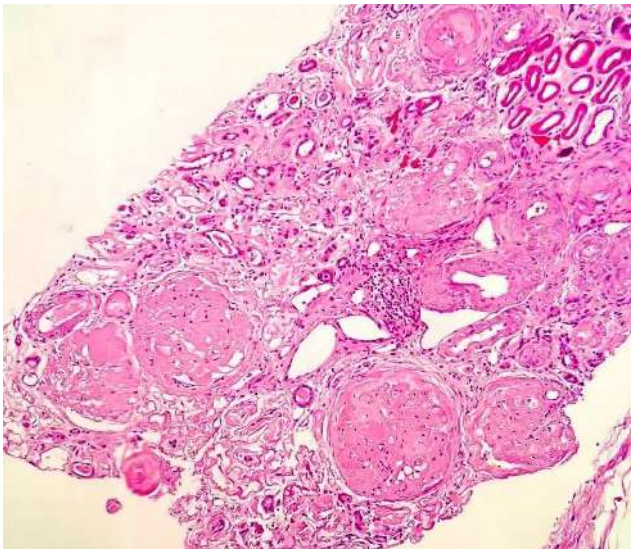


Figure 2. Kidney biopsy in case 1.

A



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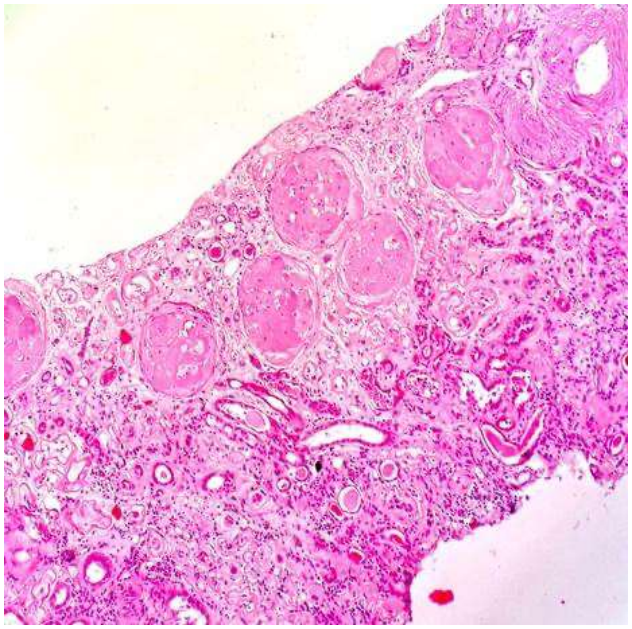
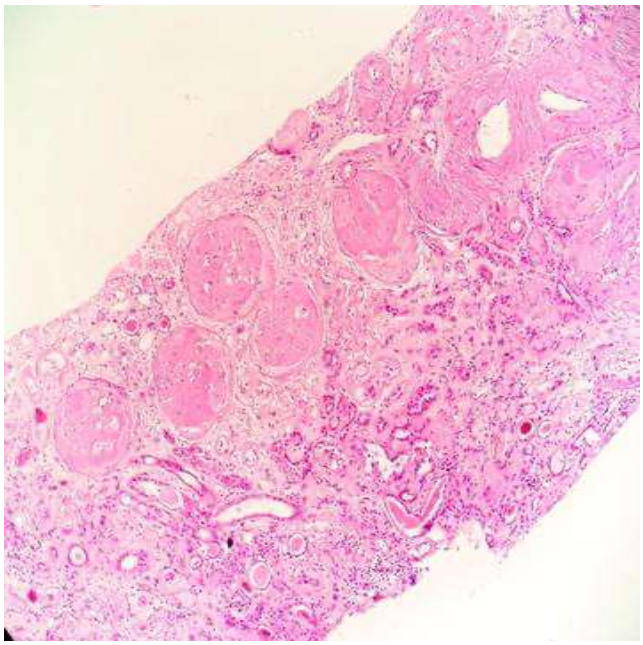




Hematoxilin & Eosin staining of the renal parenchyma with glomeruli showing global sclerosis, accompanied by fibrosis and tubular atrophy.

Clinical case 2

A 37-year-old male patient with no known pathological history referred to a habit of intense physical exercise consisting of 8 hours a week. The patient maintained a high-protein diet (Figure 3). The medications consumed during the last seven years included chronic anabolic steroid consumption: oral trenbolone (40 mg per month), supplementation with vitamins A, D and E, and creatine consumption of approximately 60 g/day. The patient was admitted for burning pain in the epigastrium 8/10, radiating to the dorsal level. Upon admission, the patient presented a blood pressure of 120/60 mmHg.

Diagnostic workshop

The patient presented with nephrotic proteinuria without the presence of microhematuria, a creatinine clearance of 9 mL/min, a serum calcium level of 13 mg/dl, a PTH level of 120 pg/ml, and an increase in the level of pancreatic enzymes to 7 times the baseline value. The urine dipstick test was positive for protein and hemoglobin. Immunoserology and viral serology tests were negative. Ultrasonography reported findings of chronicity and multiple calcifications in the renal parenchyma, and nonobstructive bilateral urolithiasis was reported, for which simple urotomography without contrast was requested, where intense bilateral nephrocalcinosis was observed (Figure 4).

Evolution

In the 24 hours after admission, the intake of vitamin D was stopped because the symptoms were pancreatitis of pharmacological origin, accompanied by hypercalcemia induced by increased intake of the polyvitamin. Vigorous venous hydration was started with 0.9% saline infusion and treatment with furosemide and corticosteroids.

During that time, until 15 days of recovery, the patient showed a progressive improvement in urinary output, and renal function began to have minimal changes; moreover, the levels of pancreatic enzymes, serum calcium and PTH returned to normal levels (8.5 mg/dl and 22 pg/ml, respectively). On Day 15, the patient with a creatinine level of 1.3 mg/dl was discharged, and a suspension of steroids, intense training and protein supplements was recommended.

Table 2. Types of anabolic steroids used by patients, creatine supplements, protein and parathyroid hormone and plasma calcium laboratory findings.

Case	Vitamins	Type of anabolic steroid	Creatine	Protein	Calcium and PTH	Vitamin D
1	Does not refer	4 mg of intramuscular testosterone weekly 80 mg of oral Trenbolone Enanthate every 15 days	40 g/día	40 g/día	Calcio: 8.7 mg/dL, PTH: 60 pg/mL	Does not refer
2	Vitamine A: 100000 UI Vitamine D: 10.000 UI Vitamine E: 20 UI	40 mg Trenbolone Enanthate monthly.	60 g/día	60 g/día	Calcium: 13 mg/dl, PTH: 120 pg/mL	25(OH)D: 80 ng/ml.
3	Does not refer	Estanozolol, 50 mg/día.	60 g/día	It does not refer.	Calcium: 9 mg/dL, PTH: 41 pg/mL	It does not refer.

PTH: Parathyroid hormone.

Figure 3. Timeline in case 2.

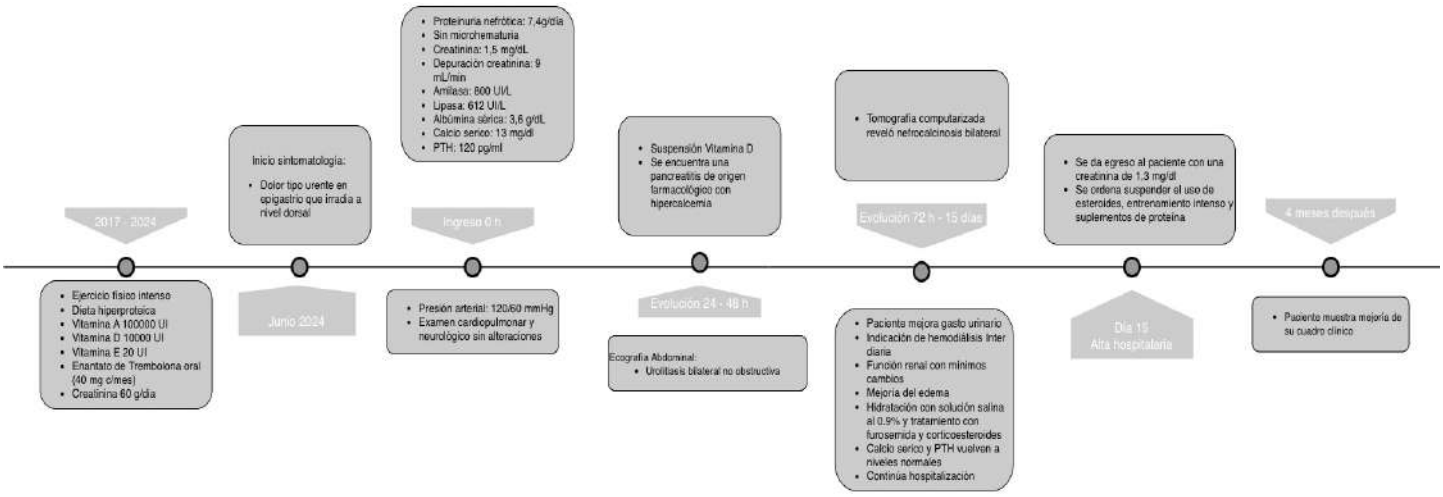


Figure 4. Simple urotomography without contrast.

A

B

Tomography of the abdomen and pelvis axial and coronal cut shows bilateral nephrocalcinosis.

Clinical case 3

A 32-year-old male patient with no known medical history and a 4-year habit of intense exercise for two hours a day reported that at the same time, he began chronic use of anabolic steroids (stanozolol, 50 mg/day) and consumed creatine 60 g/day. The patient presented with generalized myalgias, changes in the color of the urine and anuria. These changes began six months before admission and were exacerbated by the week of admission.

Upon admission, the vital signs were as follows: blood pressure 140/94 mmHg, heart rate 87 bpm, respiratory rate 17 bpm, height 1.80 m and weight 99 kg. Cardiopulmonary and neurological examinations were normal.

Diagnostic workshop

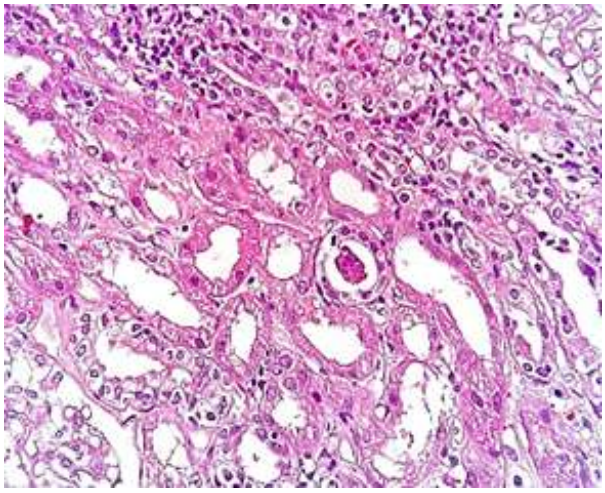
At the 24-hour follow-up, a renal ultrasound was performed, in which renal atrophy and a decreased sinus-parenchymal ratio were detected. On Day 12, a renal biopsy was performed, which revealed chronic interstitial nephritis, with interstitial edema with severe inflammatory infiltrate composed of lymphocytes, plasma cells and frequent eosinophils; interstitial fibrosis; tubular atrophy of 70%; acute tubular necrosis with regenerative foci; and immunofluorescence negative. Figure 5). The microscopic examination of urine did not reveal glomerular hematuria. The urine dipstick test was positive for protein. Immunoserology and viral serology were negative.

Evolution

At 72 hours, due to persistent anuria, renal replacement therapy is started. The use of steroids and the habit of intense exercise are discontinued. The patient is stabilized, and hospital discharge is indicated to maintain renal replacement therapy. After 4 months, the patient remains on renal replacement therapy (Figure 6).

Figure 5. Renal biopsy in case 3.

A



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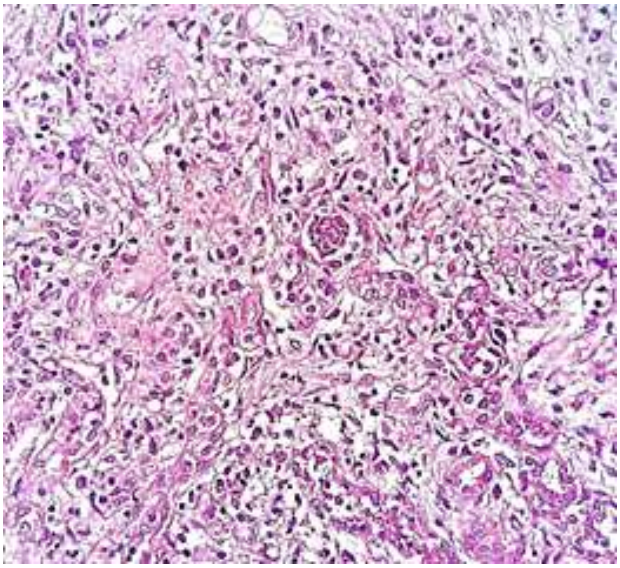

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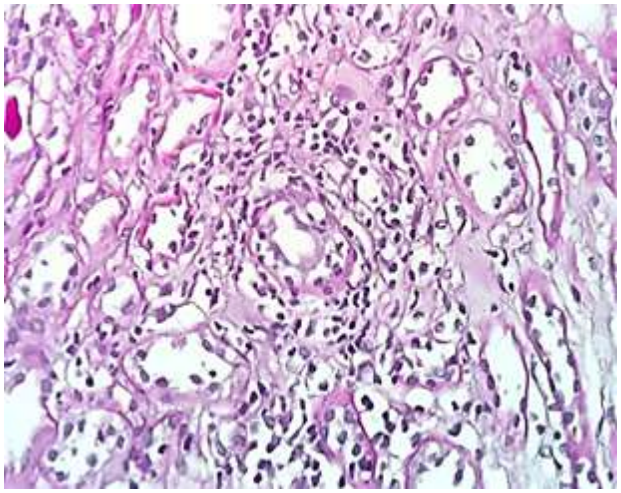
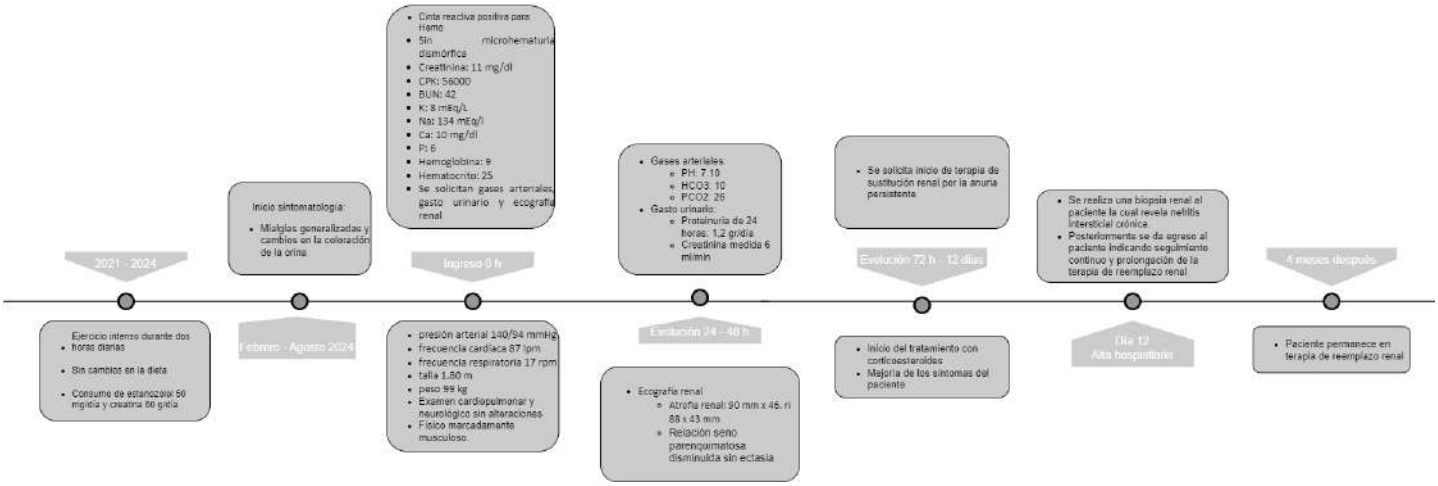


(A) y (B) Hematoxylin and eosin staining, tubule dilation, flattening of the epithelium, presence of cellular debris in lumens. (C.) PAS staining, with inflammatory infiltrate in the interstitial and tubulitis, foci of loss of the brush edge.

Figure 6. Timeline in Case 3.



Discussion

The use of AAS has been associated with various types of kidney damage, including acute injury, chronic kidney disease, and glomerular toxicity, such as focal and segmental sclerosis, and direct and indirect nephrotoxic effects via the activation of the inflammatory cascade.

Focal segmental glomerulosclerosis (FSGS) secondary to chronic abuse of AAS occurs as a consequence of nephrotoxicity on the filtration barrier (FSGS secondary to toxicity) and by adaptive responses to elevated glomerular capillary pressures and flow rates (post-adaptive FSGS).). Increased lean body mass is associated with increased renal plasma flow and vasodilation of the afferent arteriole, leading to an increased glomerular filtration rate (GFR). This hyperfiltration is also due to a decrease in the number of nephrons [8]. With AAS, activation of the NF-kB and TGF-B1 pathways, which play a role in kidney damage by inducing inflammation and renal fibrosis through increased proinflammatory cytokines and proapoptotic effects, has been observed [9]. The presence of proteins in Bowman's capsular space, a condition that can occur due to the nephrotoxicity of EAAs, further exacerbates damage to podocytes. The postadaptive form of FSGS tends to have a lower incidence of nephrotic syndrome [10], which becomes relevant in patient 1, where clinical and paraclinical findings of greater severity are evident (proteinuria in the

nephrotic range), which supports the idea of an additional nephrotoxic lesion due to AAS.

Nephrocalcinosis is proposed to be a consequence of the abuse of AAS and hypervitaminosis due to hydroelectrolytic alterations causing the accumulation of calcium in the renal system, as evidenced in the tomography of Patient 3. There is evidence that testosterone increases the serum level of 1.25 dihydroxycholecalciferol, and it has been proposed that the steroid receptor acts as a coactivator for vitamin D to bind to its receptor [11]. The patient used vitamin supplements A, D, and E, and hypercalcemia with elevated PTH levels was observed due to this supplementation. The daily use of 50,000 IU vitamin A is considered to cause adverse effects; it stimulates resorption by osteoclasts and/or PTH secretion, which also explains why the hormone is not suppressed due to high calcium levels. Similarly, vitamin D is associated with hypercalcemia at doses ranging from 10,000 IU daily and is known for its effect on the synthesis of 1.25 dihydroxycholecalciferol [12]. High calcium concentrations are known risk factors for pancreatitis; hence, pancreatic enzymes are elevated, resulting in organ damage.

Rhabdomyolysis in patients due to steroid abuse has multiple causes that together become an enhancer of muscle injury. The supratherapeutic doses of stanozolol cause injury to differentiated skeletal muscle cells and their apoptosis [13]. Coupled with vigorous exercise, it increases the risk of muscle injury and concomitant kidney damage [14], such as in patient 2.

Conclusions

AAS have direct toxic effects on the glomerulus and the tubular system or can induce injury indirectly through adaptive mechanisms, hydroelectrolytic disorders, and the endocrine system. AAS can cause direct kidney damage, including focal and segmental glomerulosclerosis, acute interstitial nephritis, and rhabdomyolysis. In addition, high protein intake can increase the risk of kidney damage.

Abbreviations

AAS: anabolic androgenic steroids.
FSGS: Focal and segmental glomerulosclerosis.
PTH: Parathyroid hormone.
GFR: Glomerular filtration rate.

Supplementary information

The supplementary materials have not been declared.

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Does not apply.

Authors' contributions

José Lucas Daza: Conceptualización, Administración del proyecto, Supervisión, validación, visualización, Escritura – revisión y edición.
María Sierra: Conceptualización, metodología, investigación.
Nicole Cifuentes: Conceptualización, metodología, investigación.
Camila Márquez: Conceptualización, metodología, investigación, Escritura – Borrador original.
Augusto Salazar: Metodología, investigación, Escritura – Borrador original.
Verónica Remache: Conceptualización, metodología, investigación.
Vanessa Villavicencio Cerón: Conceptualización, Escritura – revisión y edición.
All the authors read and approved the final version of the manuscript.

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The study was self-financed by the authors. The patient's insurance company assumed the costs of hospitalization and hemodialysis treatments.

Availability of data or materials

Does not apply.

Declarations

Ethics committee approval and consent to participate

It does not apply to clinical cases.

Consent for publication

The authors have written permission from the patients.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Author information

Not declared.

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