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Acute tubulointerstitial nephritis as a cause of acute renal failure in a patient with class III diabetic nephropathy: case report.

José Lucas Daza [1], María Paola Joven Sierra [2], Camila Alejandra Parra Márquez [2], Verónica Piedad Remache Otañez [3], Vanessa Villavicencio Cerón [4], Ronald Alfredo Cevallos Macías [5], Otto Javier Intriago Montesdeoca [6], Miguel Sacoto Mazzini [6], Marcela Patricia Perero Falcones [6].

1. Nephrology Service, Colombian Association of Nephrology and Arterial Hypertension, Bogotá, Colombia.
2. Faculty of Medicine, University of Tolima, Ibagué, Colombia.
3. Renal Pathology Service, VproPath, Pathological Anatomy Laboratory, Quito, Ecuador.
4. Nephrology Service, Hospital of the Ecuadorian Institute of Social Security Portoviejo, Ecuador.
5. Postgraduate Department of Critical Medicine and Intensive Care, Pontificia Universidad Católica del Ecuador, Portoviejo, Manabí.
6. Intensive Care Unit, Portoviejo General Hospital, Ecuador.

Received: March 23, 2025.
Accepted: June 1, 2025.
Published: June 6, 2025.
Edited by Dr. Franklin Mora Bravo.

How to cite:
Daza J, Joven M, Parra C, Remache V, Villavicencio V, Cevallos R, et al. Acute interstitial tubule nephritis as a cause of acute renal failure in a patient with class III diabetic nephropathy: case report. REV SEN 2025; 13(2):138-145.

DOI: http://doi.org/10.56867/120
Sociedad Ecuatoriana de Nefrología, Diálisis y Trasplantes.

ISSN-L: 2953-6448

Abstract

Introduction: Acute tubulointerstitial nephritis (TIN) with eosinophilia and acute tubular injury is a rare but clinically significant cause of acute kidney injury (AKI), especially in patients with chronic comorbidities.

Clinical case: A 44-year-old woman with type 2 diabetes, a history of hypertension, and a previous episode of diabetic ketoacidosis, was admitted with severe metabolic decompensation secondary to infectious gastroenteritis and dehydration. During her course in the intensive care unit, she developed KDIGO stage III AKI, requiring renal replacement therapy. A kidney biopsy confirmed the diagnosis of acute TIN with tubular injury, attributed to multiple factors: severe hypovolemia, recent exposure to potentially nephrotoxic drugs (allopurinol, antibiotics, and nonsteroidal anti-inflammatory drugs), and gastrointestinal sepsis.

Treatment: Treatment included hemodynamic support, insulin therapy, culture-adjusted antibiotics, and intermittent hemodialysis. Outcome: The patient showed metabolic improvement and partial recovery of renal function after three dialysis sessions. Her creatinine level remained at 1.6 mg/dL without requiring hemodialysis.

Conclusions: This case presents acute renal deterioration in the context of previously undiagnosed chronic kidney failure. ITN was suspected due to the presence of eosinophiluria, nonnephrotic proteinuria, absence of evident glomerular findings, and exposure to potentially nephrotoxic drugs.

Keywords:

Acute tubulointerstitial nephritis, Acute kidney injury, Diabetic nephropathy, Case report.

* Corresponding author
Email: José Lucas Daza < drlucasdaza@gmail.com >
Address: Colombian Association of Nephrology and Hypertension. Cl. 94 #No. 15-32 Office 309, Bogotá, Colombia. Tel [57] 315 6494495.

Renal failure (ARF) is a complex clinical entity characterized by a rapid decrease in the glomerular filtration rate (GFR), accompanied by nitrogen retention and hydroelectrolytic and acid–base imbalances [1-3]. Among its various causes, tubulointerstitial nephritis (TIN) represents an uncommon but clinically significant form, primarily when it is associated with eosinophilia and acute tubular damage [4].

This entity can be triggered by infections, systemic diseases, or, more frequently, exposure to drugs, which are responsible for up to 70% of cases according to some series [5]. Histologically, drug-induced TIN is characterized by an interstitial inflammatory infiltrate composed of lymphocytes, plasma cells, and, occasionally, eosinophils, in addition to varying degrees of edema and tubular damage [6-7]. Clinically, it can be insidious or manifest as rapidly progressing AKI, especially in the presence of aggravating factors such as hypovolemia, exposure to nephrotoxic agents, renal hypoperfusion, or sepsis [6].

This article describes the case of a middle-aged female patient with a long history of type 2 diabetes mellitus and previous episodes of diabetic ketoacidosis who developed KDIGO stage III AKI in the context of ATIN with urinary eosinophilia and acute tubular injury. Among the factors implicated were severe diarrhea, dehydration, and possible exposure to drugs such as allopurinol, a xanthine oxidase inhibitor used to reduce uric acid production [8]. No single triggering cause could be identified. The clinical evolution required the initiation of renal replacement therapy, with partial recovery after hemodynamic and metabolic stabilization.

Clinical case

Medical records

A 44-year-old female patient with a 22-year history of insulin-requiring type 2 diabetes mellitus. She suffered from systemic arterial hypertension, had a 30% intellectual disability, and had a history of recurrent urinary tract infections. Her medical history included a previous episode of diabetic ketoacidosis with admission to the intensive care unit (ICU), orotracheal intubation, and tracheostomy. Her surgical history included a cesarean section, cholecystectomy, and nasal surgery for recurrent skin cancer. She was allergic to penicillin and was routinely treated with NPH insulin and losartan.

The patient was admitted to the emergency department with a three-day history of vomiting, diarrhea, and abdominal pain, which did not improve with outpatient treatment. He had previously received ibuprofen and cephalexin for symptomatic management.

Diagnostic workshop

Hyperkalemia, metabolic acidosis, hypophosphatemia, mild pancreatitis, and concomitant infection were documented.

Treatment

He required admission to the ICU, where he received intensive intravenous hydration, continuous insulin therapy, broad-spectrum antibiotic therapy, and hemodialysis support for persistent oliguria and refractory hyperkalemia.

During her stay in the ICU, potentially nephrotoxic drugs were used, including allopurinol and piperacillin/tazobactam as the initial empirical antibiotics, despite a history of penicillin allergy, which was later replaced with meropenem. This drug exposure, combined with severe hypovolemia due to gastrointestinal loss and sepsis, is considered to contribute to the deterioration of renal function.

Evolution

After partial recovery from diuresis and improvement in acid–base parameters, renal replacement therapy was discontinued. However, given the persistence of renal dysfunction, a percutaneous renal biopsy was performed 8 days after admission. A histopathological study (Figure 2) revealed class III diabetic nephropathy with the presence of acute tubulointerstitial nephritis with eosinophilic infiltration and acute tubular injury, findings compatible with inflammatory and ischemic damage superimposed on chronic kidney disease. The chronicity index was 6 points, with global glomerular sclerosis (25–30%), mild tubular atrophy (20–25%), moderate interstitial fibrosis (26–30%), and severe hyaline arteriopathy.

These findings support the diagnosis of acute kidney injury superimposed on chronic kidney disease in the context of diabetic ketoacidosis, renal hypoperfusion, exposure to nephrotoxic drugs, and a systemic inflammatory response. The patient was discharged with persistently impaired renal function and scheduled for follow-up by a nephrologist to evaluate for progression to end-stage chronic kidney disease and possible indications for permanent renal replacement therapy. The timeline of this case is presented below (Timeline 1). The patient has been under continuous outpatient evaluation for 3 months and maintains a serum creatinine of 1.6 mg/dL without requiring renal replacement therapy.

Figure 1. Timeline in case 1.

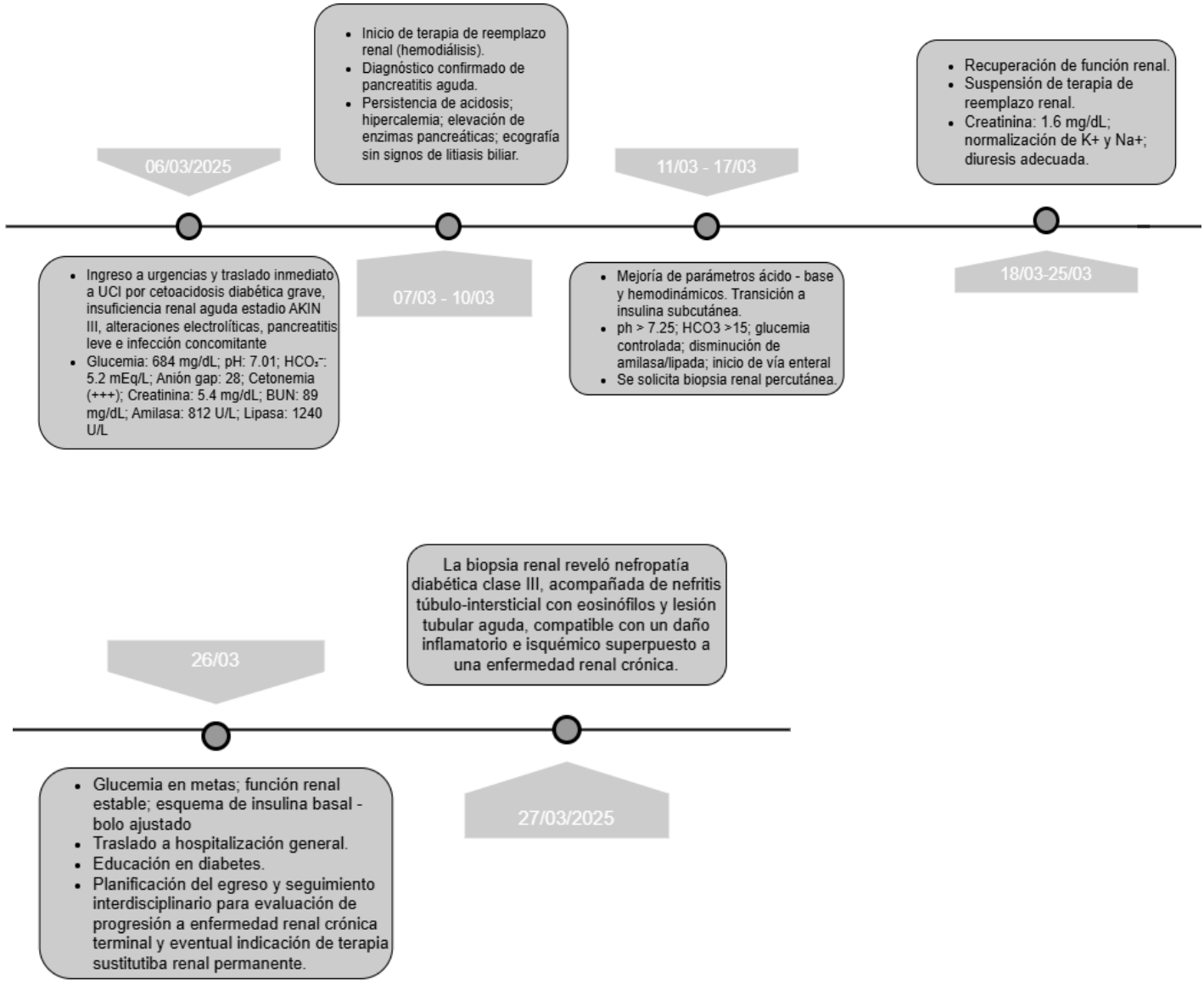
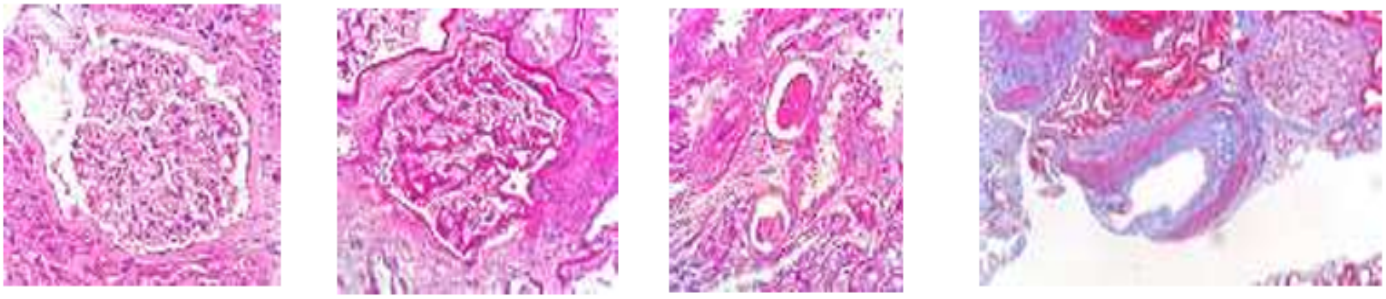


Figure 2. Renal biopsy in the case: Class III diabetic nephropathy, tubulo-interstitial nephritis with eosinophils + acute tubular injury.



Renal parenchyma consisting of cortex, medulla, corticomedullary junction; which had 15 glomeruli, 4 with global sclerosis, no segmental sclerosis, 2 with hypoperfusion, all present thickening of the glomerular basement membranes, without filling defects, with expansion of the mesangial matrix and focal formation of Kimmelstiel-Wilson nodules (PAS + JONES +), no endocapillary hypercellularity, no extracapillary lesions. The tubules show tubular atrophy that compromises between 20-25%. The epithelium is desquamated and some tubules have dilated lumens. The interstitium shows foci of fibrosis in 25-30% of its surface and patches of lymphocytic inflammatory infiltrate (unrelated to areas of fibrosis, they are accompanied by eosinophils). Medium-caliber vessels show intimal fibrosis/sclerosis that compromises more than 50% of their thickness. Arterioles are hyalinized. Pathological techniques used: HE, PAS, TRICHROME, JONES.

Discussion

Acute kidney injury (AKI) is a complex nephrological emergency, the etiology of which is often multifactorial, especially in patients with comorbidities such as long-standing diabetes mellitus, hypovolemia, and exposure to potentially nephrotoxic drugs. In this context, tubulointerstitial nephritis (TIN) with urinary eosinophilia and acute tubular injury represents a relevant, although frequently underdiagnosed, cause of acute kidney injury [9].

Therefore, early diagnosis of NTI is crucial in patients with multiple cumulative risk factors, such as those mentioned above, to prevent progression to chronic kidney failure or, in severe cases, the need for renal replacement therapy. This progression is usually mediated by inflammatory and ischemic processes that convergently affect the renal parenchyma, severely affecting the interstitium and renal tubules [10].

In the present case, the patient developed KDIGO stage III AKI requiring renal replacement therapy in the context of a gastrointestinal infection, severe dehydration, metabolic acidosis, and recent exposure to multiple nephrotoxic medications, including cephalexin, ibuprofen, and probably allopurinol. The combination of these factors represents a "nephrotoxic storm" that can precipitate acute kidney injury, both tubular and interstitial. Drug-induced TIN has been widely documented as a potential cause of AKI, with beta-lactam antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), and xanthine oxidase inhibitors being some of the most frequently implicated agents [9, 10].

Drug-induced TIN accounts for up to 27% of hospital-acquired intrinsic AKI but is underdiagnosed in the absence of biopsy [11]. In this case, histological confirmation of eosinophilic inflammatory infiltration in the renal interstitium, combined with evidence of acute tubular necrosis and chronic changes such as glomerular sclerosis and interstitial fibrosis, allowed for the diagnosis of TIN superimposed on class III diabetic nephropathy, which frames a syndrome of "acute on chronic kidney injury" [12].

An early diagnosis would have allowed the early discontinuation of potentially implicated agents—cephalexin, ibuprofen, and allopurinol—as well as the mitigation of progression factors, such as the use of diuretics in the context of hypovolemia. These interventions could prevent the perpetuation of tubular dysfunction and the progression to irreversible renal damage [6].

An immunopathological approach revealed that allopurinol can act as a hapten by binding to endogenous proteins, forming antigenic complexes that activate CD4+ T lymphocytes and mast cells. This activation triggers an inflammatory cascade mediated by the release of IL-5, which promotes the recruitment of eosinophils to the renal interstitium [4]. The presence of eosinophilic infiltrate in the renal biopsy is highly suggestive of ITN of immunoallergic origin, with that induced by allopurinol being a key differential diagnosis in the presence of eosinophiluria and acute renal failure in patients with recent exposure to said drug. Furthermore, its hepatic metabolism can generate free radicals that induce direct tubular damage, which is exacerbated in patients with previous kidney disease [13, 14].

Acute tubular injury results from necrosis or apoptosis of tubular epithelial cells, especially in segments with high oxygen demand, such as the proximal convoluted tubule and the thick ascending limb of the loop of Henle. In this case, severe and sustained hypovolemia, secondary to diarrhea, vomiting, and inappropriate use of diuretics, resulted in a critical decrease in renal blood flow, with preferential ischemia in the outer renal medulla [15].

Ischemia activates Toll-like receptors on tubular cells, inducing the secretion of proinflammatory cytokines (IL-6, TNF-α), intrarenal vasoconstriction, and disruption of the tubular cytoskeleton. This leads to loss of cell polarity and the formation of obstructive casts, perpetuating the reduction in glomerular filtration and favoring the collapse of the tubulointerstitial architecture [16].

Additionally, the nephrotoxic drugs involved act as cofactors in tubular damage:

- Ibuprofen inhibits the synthesis of prostaglandins, reducing renal perfusion [17].
- Cephalexin may induce type IV hypersensitivity reactions, with interstitial mononuclear and eosinophilic infiltrates (18).
- Allopurinol: associated with immunoallergic responses with tubular necrosis, eosinophilia, and, in severe cases, drug-induced hypersensitivity syndrome (DRESS) [4].

Recent exposure to allopurinol, in the context of hypovolemia, sepsis, and hyperglycemia, created an inflammatory and immunologically activated environment, favoring the development of TIN with acute tubulointerstitial damage. Additionally, extrarenal losses due to severe diarrhea and the sustained hypovolemic state likely contributed to prolonged renal hypoperfusion and acute tubular injury. This condition triggers a local inflammatory response, which is intensified in the presence of hyperglycemia, acidosis, and bacterial endotoxins, all of which are present in this clinical case. The use of diuretics during the oliguric phase, without an adequate response, could also have worsened renal injury owing to increased volume and sodium depletion [19].

Elevated pancreatic enzymes without abdominal pain or classic signs of pancreatitis increase the possibility of a systemic inflammatory response or a functional reaction secondary to transient hypercalcemia or sepsis. While the exact mechanism remains unclear, a connection between renal dysfunction and subclinical pancreatic abnormalities has been documented in critical situations, such as diabetic ketoacidosis [20, 21]. Future studies on the impact of hypertension and ultrafiltration on the recovery of renal function should be conducted [22].

Conclusions

This case presents acute renal deterioration in the context of previously undiagnosed chronic kidney disease. ITN was suspected due to the presence of eosinophiluria, nonnephrotic proteinuria, absence of evident glomerular findings, and exposure to potentially nephrotoxic drugs. Timely identifying these factors allows for establishing targeted therapies, preventing progression to chronic kidney disease, and reducing associated mortality.

Abbreviations

IL: Interleukin.
AKI: Acute kidney injury.
NTI: Tubulointerstitial nephritis.

Additional information

No supplementary materials have been declared.

Acknowledgments

Not applicable.

Authors' contributions

José Lucas Daza: Conceptualization, Project management, Supervision, validation, visualization, Writing – review and editing.
María Paola Joven Sierra: Conceptualization, methodology, research.
Camila Alejandra Parra Márquez: Conceptualization, methodology, research.
Verónica Remache: Conceptualization, methodology, research, writing – Original draft.
Vanessa Villavicencio Cerón: Methodology, research, writing – Original draft.
Ronald Alfredo Cevallos Macías: Conceptualization, methodology, research.
Otto Javier Intriago Montesdeoca: Conceptualization, methodology, research.
Miguel Sacoto Mazzini: Conceptualization, methodology, research.
Marcela Patricia Perero Falcones: Conceptualization, methodology, research.
All the authors read and approved the final version of the manuscript.

Financing

The authors self-funded the study. The patient's insurance provider covered the costs of hospitalization and hemodialysis treatments.

Availability of data or materials

Not applicable.

Statements

Ethics committee approval and consent to participate

These findings do not apply to clinical cases.

Consent for publication

The authors have written permission from the patient for publication.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Authors' information

José Lucas Daza: Internist – Nephrologist. Colombian Association of Nephrology and Arterial Hypertension, Bogotá, Colombia.
Email: drlucasdaza@gmail.com
ORCID: https://orcid.org/0000-0002-6430-5415

María Paola Joven Sierra: Faculty of Medicine, University of Tolima, Ibagué, Colombia.
Email: mpjovens@ut.edu.co
ORCID: https://orcid.org/0009-0000-1565-4451

Camila Alejandra Parra Márquez: Faculty of Medicine, University of Tolima, Ibagué, Colombia.
Email: caparrama@ut.edu.co
ORCID: https://orcid.org/0009-0006-2412-662X

Verónica Piedad Remache Otañez: Specialist in Pathological Anatomy, Central University of Ecuador 2013--2015. Doctor, UCE 2011. Renal Pathology Service, VproPath Pathological Anatomy Laboratory, Quito, Ecuador.
Email: draverorem@hotmail.com
ORCID: https://orcid.org/0000-0003-0543-4927

Vanessa Villavicencio Cerón: Medical Surgeon from the Technical University of Manabí (Ecuador, 2007). Nephrology Specialist from the Pontifical Catholic University of Argentina (Buenos Aires, 2015). Nephrology Service Staff, IESS Hospital of Portoviejo. Diploma in Interventional Nephrology, Mexico 2023.
Email: elvavice@hotmail.com
ORCID: https://orcid.org/0000-0003-0543-4927

Ronald Alfredo Cevallos Macías: Medical Surgeon from the Technical University of Manabí (Ecuador, 2016). Master's degree in Quality Management and Healthcare Auditing from the Technical University of Loja (Ecuador, 2021). Postgraduate in

Critical Medicine and Intensive Care from the Pontifical Catholic University of Ecuador (Ecuador, 2025). Physician, Postgraduate in Critical Medicine from the Pontifical Catholic University of Ecuador.
Email: rocemac@hotmail.com
ORCID: https://orcid.org/0000-0002-5145-6740

Otto Javier Intriago Montesdeoca: Doctor of Medicine and Surgery from the Catholic University of Santiago de Guayaquil (Guayaquil, 2006). He is a specialist in intensive care from the University of Salvador (San Salvador, 2014). He is an attending physician in the Portoviejo General Hospital's intensive care unit. Ecuador.
Email: drottointriago@hotmail.com
ORCID: https://orcid.org/0009-0007-6830-3904

Miguel Sacoto Mazzini: Doctor of Medicine and Surgery from the Technical University of Manabí (Portoviejo, 2012). Specialist in critical care medicine from the Catholic University of Argentina, Santa María de los Buenos Aires (Buenos Aires, Argentina, 2009). Attending Physician in the Intensive Care Unit at Portoviejo General Hospital, Ecuador.
Email: miguelsama@gmail.com
ORCID: https://orcid.org/0009-0002-4303-8845

Marcela Patricia Perero Falcones: Medical Surgeon from the Technical University of Manabí. (Portoviejo, 2021).
Email: marceperero28@gmail.com
ORCID: https://orcid.org/0009-0003-8402-7752

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DOI: Digital Object Identifier. **PMID**: PubMed Identifier.

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