

# CASE REPORT: “ACUTE TUBULOINTERSTITIAL NEPHRITIS RELATED TO COCAINE USE”

Dr. Joary Vargas Santana<sup>1</sup>, Dr. Giovanna Arteaga Muller<sup>1</sup>, Dr. Mara C. Olivo Gutiérrez<sup>1</sup>, Dr. Carina S. Zapata Beltrán<sup>1</sup>, Dr. Daniela Campos Elvir<sup>1</sup>, Dr. Nayeli N. López Villa<sup>1</sup>, Dr. Ma. Virgilia Soto Abraham<sup>2</sup>.

1. Hospital Universitario “Dr. José Eleuterio González”, Departamento de Nefrología, Monterrey, México.

2. Instituto Nacional de Cardiología Ignacio Chávez, Estado de México, México.



## Introduction

Acute interstitial nephritis (AIN) represents a common cause of acute kidney injury and accounts for 15 to 27% of kidney biopsies performed due to this condition. Cocaine-associated AIN is an extremely rare clinicopathological entity.

## Methods

Case report that was presented at Hospital Universitario UANL, Monterrey, N.L

## Results

47-year-old male patient, with a history of occasional alcoholism, inactive for 1 month, smoking with a smoking rate of 25 packs/year, inactive for 1 month, drug addiction daily cocaine for 14 years with increase of consumption during the last month, denies chronic degenerative diseases. It began 1 week prior to admission with a decrease in urinary volume, edema of the lower limbs, asthenia, adynamia, nausea, vomiting of gastro-alimentary contents, for which reason the patient attended for evaluation. Within his laboratory findings with hb 14.6 g/dl, albumin 3.1 g/dl, Cr 22.3, BUN 166, Na 120, K 7.2, Ca 7.8, P 8.8, GFR: 2.1 ml/min by CKD-EPI, EGO with pH 6.5, density 1,012, proteins 131, erythrocytes more than 50 per field, leukocytes from 51 to 80 per field, esterase negative, bacteria scarce. Urinary sediment was obtained with a predominance of dysmorphic erythrocytes and acanthocytes. Proteins in 24-hour urine with 0.03 g in a volume of 150 ml, ultrasound with kidneys of normal size and morphology, without signs of hypertensive retinopathy. Viral panel negative, C3 and C4 within normal range, ANAS negative, ANCAS negative, Anti-GBM negative. Renal replacement therapy was initiated through hemodialysis. A renal biopsy was performed that showed active tubulo-interstitial nephritis with eosinophils, multifocal acute tubular lesion with moderate regenerative changes of the epithelium and with intratubular microcalcifications, moderate nodular hyaline arteriolopathy, negative immunofluorescence, without criteria for chronicity.

## Conclusions

Cocaine-Associated Kidney Injury may have variable underlying causes, timely recognition of this entity due to its non-specific clinical presentation is crucial because these patients can develop serious deterioration in renal function. Kidney damage due to cocaine has been more frequently associated with rhabdomyolysis, vasculitis, malignant hypertension, and thrombotic microangiopathy, which were ruled out in our patient. However, there are few cases that suggest a link between acute interstitial nephritis and exposure to cocaine. At the time of suspension and steroid treatment at a dose of 1 mg/kg/day, renal replacement therapy was withdrawn and complete recovery of renal function occurred.

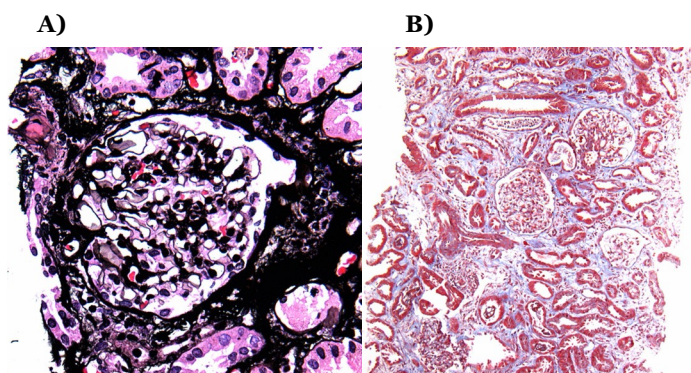


Fig. 1, A) H/E Stain: Glomerulus with open capillary loops, homogeneous basement membrane, without spicules, no mesangial proliferative changes, segments of endocapillary hypercellularity, or active extracapillary proliferative lesions are observed. B) Trichrome Stain: It is observed in the tubulo-interstitial compartment with generalized edema, loss of the tubular “back to back” pattern, with an inflammatory infiltrate made up of lymphocytes, plasma cells, neutrophils and eosinophils.

## BIBLIOGRAPHY

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