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A Case of Acute Tubulointerstitial Nephritis Secondary to the Use of "Pouffa"

Jean Claude Manirakiza[1*], Nassima Hussein Abdelaziz[1], Olivier Emerusabe[1], S. El Khayat[1], N. Bennani Guebessi[2], N. Mtioui[1], M. Zamd[1], G. Medkouri[1], M. Benghanem[1]

[1]Department of Nephrology, Hemodialysis and Transplantation, Ibn Rochd University Hospital, Casablanca, Morocco
[2]Department of Pathology, Ibn Rochd University Hospital, Casablanca, Morocco

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***Corresponding author:** Jean Claude Manirakiza
Department of Nephrology, Hemodialysis and Transplantation, Ibn Rochd University Hospital, Casablanca, Morocco

Abstract | **Case Report**

Acute tubulointerstitial nephritis (ATIN) is defined by the presence of histological lesions affecting the renal interstitium and tubules, most often secondary to a local inflammatory reaction associated with leukocyte infiltration. The causes are numerous. We report a case of ATIN secondary to abuse of "pouffa" (a cocaine-derived drug). The patient was an 18-year-old man admitted for severe renal failure with a serum creatinine level of 348 mg/L, associated with proteinuria of 1.46 g/24 h, active urinary sediment with microscopic hematuria, and sterile leukocyturia. A renal biopsy showed edematous interstitial tissue with inflammatory infiltrates and tubulitis lesions consistent with ATIN. The patient required emergency hemodialysis sessions and was subsequently treated with corticosteroid therapy, leading to complete recovery of renal function after two weeks. The effects of pouffa on the kidneys are devastating. Early diagnosis and prompt management are the key to full recovery of renal function.

Keywords: Acute tubulointerstitial nephritis, pouffa, renal biopsy, corticosteroids.

INTRODUCTION

Acute tubulointerstitial nephritis (ATIN) accounts for 25–27% of biopsies performed for organic acute kidney injury (AKI) [1, 2]. Histologically, it is characterized by interstitial inflammatory infiltrates leading to edema, with tubulitis lesions [3, 4]. The nephrological clinical presentation is often nonspecific, with acute kidney injury, without edema, hypertension, or macroscopic hematuria. The disease may progress to fibrosis, resulting in chronic tubulointerstitial nephritis (CTIN).

The etiologies are multiple, including drug-induced causes (direct toxicity or immuno-allergic mechanisms, 80%), infectious, autoimmune (lupus, etc.), and tubulointerstitial nephritis and uveitis syndrome. In some cases, no etiology is identified (idiopathic ATIN) [5]. However, the list of potential causes of ATIN continues to expand. We report a case of ATIN secondary to the use of "pouffa" (or crack), a drug derived from cocaine residues mixed with chemical products including ammonia.

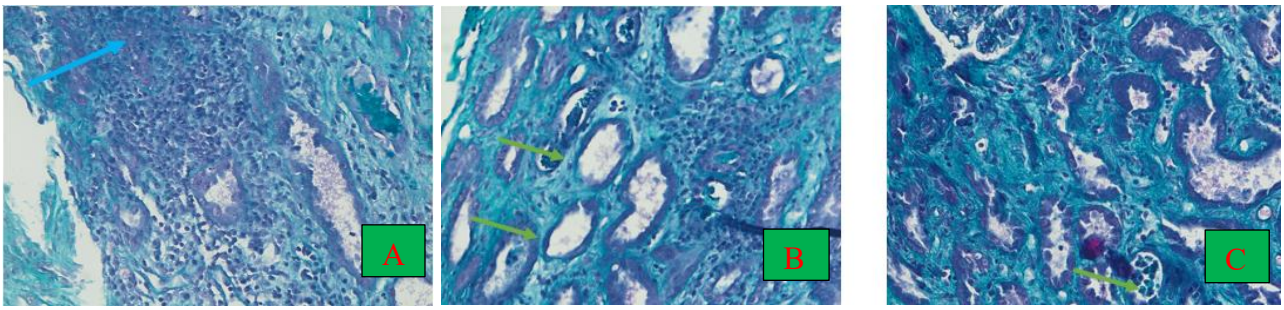
CASE PRESENTATION

The patient was an 18-year-old high school graduate admitted for severe renal failure with a creatinine level of 348 mg/L, associated on questioning with a history of pouffa use for three months, discontinued one week prior to admission. Clinical examination was normal, with preserved urine output. Urine dipstick testing showed 2+ protein and 1+ blood.

Renal impairment was characterized by uremia of 2.2 g/L, serum creatinine of 348 mg/L, 24-hour proteinuria of 1.46 g, and active urinary sediment with microscopic hematuria and aseptic leukocyturia. There was mild microcytic anemia with hemoglobin at 11.5 g/dL. Immunological tests showed normal complement fractions C3 and C4, and negative antinuclear and anti-DNA antibodies.

Renal ultrasound revealed kidneys of normal size with good corticomedullary differentiation and no dilation of the collecting system. Renal biopsy showed that among the 11 glomeruli sampled, there was no cellular proliferation or deposits. The edematous interstitial tissue contained inflammatory infiltrates

composed of lymphocytes, plasma cells, and neutrophils, consistent with acute tubulointerstitial nephritis associated with tubular necrosis. Immunofluorescence revealed fine, weak, and non-significant mesangial C3 deposits.



A: Renal biopsy showing interstitial edema and a polymorphous inflammatory infiltrate with tubulitis lesions (blue arrow). B and C: Renal biopsies showing tubular necrosis lesions (green arrows)

Therapeutic Management

From a therapeutic standpoint, the patient underwent six sessions of hemodialysis and received three boluses of Solumedrol, followed by oral corticosteroid therapy (1mg/kg/day). In addition, given the context of substance abuse, a specialized addiction medicine consultation was requested in order to support the patient in discontinuing pouffa use and to prevent the risk of relapse. The clinical course was marked by an improvement in renal function, with a follow-up serum creatinine level of 9.6 mg/L after one month.

DISCUSSION

In our case, acute kidney injury with preserved urine output and leukocyturia suggested acute tubulointerstitial nephritis (ATIN), tubular necrosis in the recovery phase, and a superimposed glomerular involvement in view of the glomerular syndrome (proteinuria >1 g/24 h with active urinary sediment). Renal biopsy confirmed the diagnosis of acute tubulointerstitial nephritis associated with lesions of acute tubular necrosis.

Tubulointerstitial nephritis was first described by Councilman in 1898 [6]. While examining autopsies of patients who had died from bacterial septicemia with renal failure, he noted severe interstitial lesions in the absence of renal bacterial invasion [7]. Since then, numerous studies have improved the understanding of the basic mechanisms of this condition, its epidemiology, etiologies, clinical presentation, and outcome.

Typically, ATIN presents as acute kidney injury with preserved urine output, sometimes with polyuria, and may be associated with systemic extrarenal manifestations including fever, arthralgia, skin rash, and hypereosinophilia, suggestive of a hypersensitivity reaction [8, 9]. Our patient had severe renal failure with preserved urine output but did not exhibit any symptoms suggestive of hypersensitivity. However, according to the literature, not all patients present with the full spectrum of symptoms, and only 5–10% display the classic triad (fever, rash, hypereosinophilia) [8,9].

Although suspicion of ATIN may be based on clinical findings and urinary laboratory abnormalities, renal biopsy is generally required to establish a definitive diagnosis, as clinical and biological features are not specific [8,10]. As in our patient, histology reveals interstitial inflammatory infiltrates with edema associated with tubulitis lesions. Glomeruli and vessels are usually normal unless another superimposed pathological process is present [10].

According to the literature, drug-induced hypersensitivity (NSAIDs, antibiotics, etc.) is the leading cause of ATIN [11]. In our patient, there was no history of exposure to medications known to cause ATIN. The occurrence of severe organic acute kidney injury in the context of pouffa abuse led us to suspect its role in the development of renal injury. According to published data, cocaine exerts harmful effects on the kidneys through a multifactorial mechanism involving alterations in renal hemodynamics due to vasoconstriction, abnormalities in mesangial matrix secretion and degradation associated with oxidative stress, and renal atherogenesis [12]. Crack abuse (a cocaine residue) has previously been associated with the occurrence of ATIN [13]. In our case, the implicated substance was pouffa.

From a therapeutic perspective, corticosteroid therapy was justified in our patient due to the severity of renal failure and the absence of significant spontaneous improvement, as well as to halt the interstitial inflammatory process and prevent progression to fibrosis, which allowed recovery of renal function. According to the literature, discontinuation of the offending drug or toxic agent is the cornerstone of ATIN management and enables recovery of renal function in most patients. If no improvement is observed within 3–7 days, corticosteroids are suggested at a dose of 1 mg/kg/day (not exceeding 60 mg/day) for two weeks, followed by gradual tapering over up to three months.

However, the use of corticosteroids remains controversial due to the lack of prospective randomized studies [5, 14, 15]. Renal prognosis is often favorable, but progression to chronicity may occur in cases of delayed diagnosis and treatment [11].

CONCLUSION

Pouffa consumption among young people is becoming increasingly concerning. Its effects are more potent but shorter-lasting than those of cocaine, which encourages users to increase the frequency of intake. Several toxic effects have been described, particularly at the renal level. To our knowledge, we report the first case of acute tubulointerstitial nephritis secondary to pouffa use in Morocco.

Conflict of Interest

The authors declare no conflicts of interest.

Acknowledgments

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