

## *Lonomia obliqua* (Caterpillar)–Related Kidney Failure: A Rare Histopathology Register

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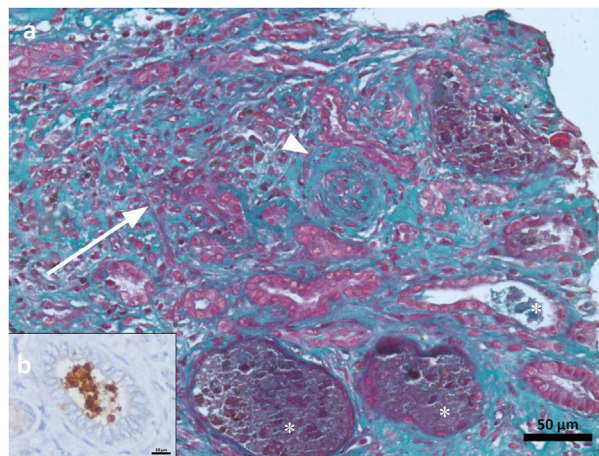


A 50-year-old man with no significant medical history presented to the emergency department 1 week after envenomation by multiple caterpillars (*Lonomia obliqua*) (Fig 1). A week prior, immediately following envenomation, he developed a papular, pruritic eruption, followed several hours later by fatigue, abdominal pain, and bruising. He subsequently developed anuria. On presentation, he had melena and epistaxis. The laboratory values showed serum creatinine of 11.0 mg/dL and blood urea nitrogen of 181 mg/dL, and hemodialysis was initiated. Antilonomic serum was administered shortly thereafter. He remained dialysis-dependent 1 month after the lonomic accident; accordingly, a kidney biopsy was performed. This revealed glomeruli with marked ischemic changes and global glomerulosclerosis, intense fibrosis, severe acute tubular necrosis, acute interstitial nephritis, hyperplastic arteriolosclerosis, hemosiderin deposition on Perls' staining, and myoglobin deposition on immunohistochemistry (Figs 2 and 3). Abdominal computed tomography showed kidneys with regular dimensions and contours. One year later, he remains dialysis-dependent.

Beyond urticarial dermatitis, pain, and swelling after contact with the caterpillar bristles, blood dyscrasia and hemorrhagic manifestations may appear, leading to mucosal or potentially fatal intracavitary bleeding.<sup>1</sup> Acute kidney injury (AKI) is present in 12% of cases.<sup>2</sup> The venom components interact with the coagulation



**Figure 1.** Group of *Lonomia obliqua* found in the same region where the patient had the accident. Photo taken by the authors.



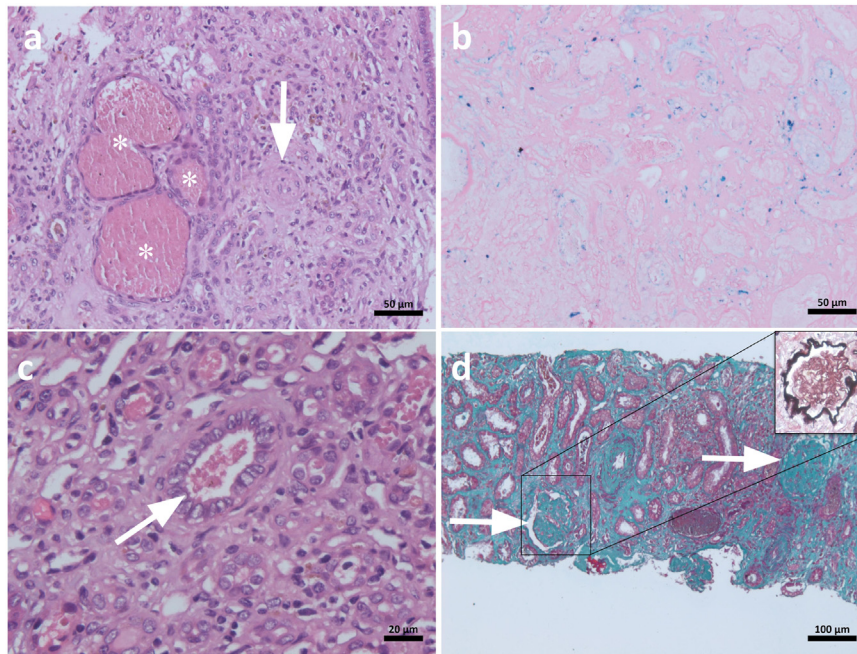
**Figure 2.** Kidney biopsy performed because of kidney failure induced by a lonomic accident reveals (A) multicompartmental involvement with intense fibrosis (in green on Masson's trichrome staining), acute tubular necrosis (white asterisk), inflammatory infiltrate (white arrow), and hyperplastic arteriolosclerosis (white arrowhead). (B) Immunohistochemistry shows intraluminal myoglobin deposits in the kidney tubule.

cascade, fibrinolysis, and platelet hypoaggregation, leading to a consumption coagulopathy. Because of the risk of bleeding, conducting an early kidney biopsy in human patients is difficult; this case is a unique and highly illustrative kidney biopsy related to lonomic accident-induced kidney failure.<sup>3</sup> In an animal model, lonomic accident-induced AKI is caused by hypotension, fibrin deposition, kidney hypoperfusion, and tubular necrosis, leading to loss of kidney function. Intense inflammation, tissue injury, heme-induced oxidative stress, coagulation, and complement system activation contribute to the kidney damage.<sup>4</sup>

Caterpillars are key constituents of many terrestrial ecosystems. With deforestation, they are invading urban areas. Data from the Brazilian Ministry of Health show 4,211 cases of lonomic accidents in the last 5 years,<sup>5</sup> but the number of cases is probably underreported.

### ARTICLE INFORMATION

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**Figure 3.** Kidney biopsy performed because of kidney failure induced by a lonomia accident reveals (A) intense mononuclear inflammatory infiltrate on hematoxylin-eosin (HE) staining, severe acute tubular necrosis (white asterisk), hyperplastic arteriosclerosis (white arrow) and hemosiderin deposition (see golden dots). (B) Hemosiderin deposition (see blue dots) on Perls' staining. (C) Myoglobin deposition on HE staining (white arrow). (D) Intense fibrosis and 2 sclerosed glomeruli on Masson's trichrome staining (white arrow). Inset figure: Glomeruli in progression to global sclerosis due to ischemic origin. Note the wrinkled appearance of the glomerular basement membrane and Bowman capsule (silver staining).

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