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Crescentic glomerulonephritis in a man with a history of methamphetamine abuse; a possible cause for concern

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Methamphetamine abuse typically causes acute kidney injury mainly due to rhabdomyolysis, malignant hypertension and volume depletion and acute tubular necrosis. However, methamphetamine-induced crescentic glomerulonephritis (CresGN) has not been reported so far.

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Photonephrology

A 48-year-old man was referred to our nephrology clinic due to hematuria, proteinuria, coupled with elevated serum creatinine level (6.8 mg/dL). He reported a recent history of methamphetamine abuse by smoke inhalation. When admitted, his blood pressure was 150/90 mm Hg, and the report of fundoscopic examination was normal. Further laboratory examinations revealed: white blood cells (WBC) count, $8700\times10^3/mm^3$, platelets, $186\times10^3/mm^3$, hemoglobin; 9.6 g/dL, urea 145 mg/dL, sodium, 137 mEq/L, potassium, 4.7 mEq/L, calcium, 9 mg/dL, and phosphorus, 5.5 mg/dL. Antinuclear antibody (ANA), anti-double-stranded DNA (anti-dsDNA), and anti-neutrophil cytoplasmic antibody (c-ANCA and p-ANCA) were all negative. The complement CH50 level was low; 15.4 U/ml (Normal range: 51-150 U/mL), but both C3 and C4 levels were in the normal range. The serological tests for viral hepatitis B, C, and human immunodeficiency virus (HIV) were all negative.

An ultrasound-guided renal biopsy was conducted. Light microscopy revealed cellular crescents in six glomeruli and fibrous crescent in one among a total of 12 sampled glomeruli (Figure 1). Immunofluorescence microscopy showed mild capillary wall staining for IgG (+) and C3 (+ +), but negative for C1q, IgM, IgA, kappa, and lambda. The patient received pulse therapy with 500 mg/day methylprednisolone for three days followed by oral prednisone and cyclophosphamide intravenous (IV) pulse. Three weeks later, his serum creatinine level decreased to 2.1 mg/dL, the CH50 level returned to its normal range and his general condition improved significantly.

Methamphetamine abuse typically causes acute kidney injury mainly due to rhabdomyolysis, malignant hypertension and volume depletion and acute tubular necrosis. However, methamphetamine-induced crescentic glomerulonephritis (CresGN) has not been reported so far. Unfortunately, establishing the causal relationship of CresGN to methamphetamine nephrotoxicity in his case was impossible. Although, it illustrates the necessity of considering this drug as a possible cause of

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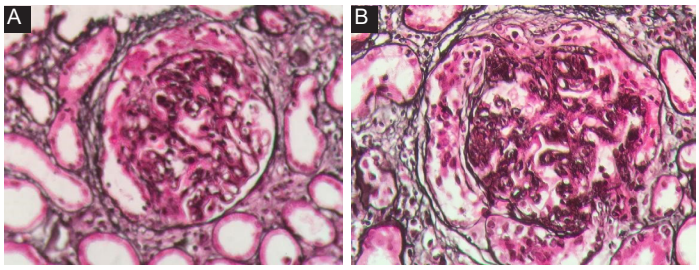

Figure 1. Two glomeruli show prominent cellular crescents formation compressing the native glomerular tufts. (A) The crescent partially covers the tuft. There is mild interstitial inflammatory cell infiltrate in the surrounding parenchyma (Jones' silver stain ×200). (B) The crescent is circumferentially covering the entire tuft. Mild periglomerular inflammatory infiltrate is present (Jones' silver stain ×400).

renal damage in such situations. Whilst it is difficult to prove that methamphetamine may be adulterated with other compounds such as cocaine and levamisole, that are known for their nephrotoxic pauci-immune CresGN causation (1-3).

Authors' contribution

Conceptualization: MA, Methodology: MM, ShA & HRJ, Validation: DJ, Formal Analysis: MM, Investigation: MA, Resources: DJ & SS, Data Curation: MA, Writing—Original Draft Preparation: MA & SS, Writing—Review and Editing: DJ & SS, Visualization: DJ & SS, Supervision: MA, Project Administration: MA, Funding Acquisition: -

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical issues

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