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Vapor-Induced Acute Kidney Injury Presenting as a Pulmonary–Renal Syndrome in an Adolescent

Cagla Cagli Piskin

drcaglacagli@gmail.com

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi https://orcid.org/0000-0002-0912-3045

Bahriye Atmis

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi

Zahide Orhan Ok

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi

Kıvılcım Eren Ates

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi

Gulfiliz Gonlusen

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi

Aysun K. Bayazit

Cukurova University Faculty of Medicine: Cukurova Universitesi Tip Fakultesi

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Abstract

Electronic cigarette use is increasingly common among adolescents and is associated with a widening range of toxic effects, although renal complications remain poorly characterized and are rarely confirmed histologically in this age group. We describe a previously healthy 14-year-old boy who developed oliguria, dark- colored urine, severe hypertension, seizures, and pulmonary infiltrates two days after electronic cigarette use. Serum creatinine rose to 10.6 mg/dL, requiring hemodialysis. The presentation raised concern for a pulmonary–renal syndrome, prompting empiric corticosteroids and plasma exchange. However, serological testing was negative, urinary β2-microglobulin was markedly elevated, and kidney biopsy showed acute tubular injury without glomerular or immune-mediated disease. Immunosuppression was withdrawn, and kidney function recovered completely, remaining normal at 18 months. This case shows that vaping-associated acute tubular injury can mimic a pulmonary–renal syndrome and highlights the value of kidney biopsy in adolescents with unexplained severe acute kidney injury.

Summary – What is new

Electronic cigarette use can cause biopsy-proven, dialysis-requiring acute tubular injury that mimics a pulmonary–renal syndrome; kidney biopsy averted unnecessary immunosuppression and supported a favorable prognosis with complete recovery.

Introduction

Electronic cigarette use is increasingly prevalent among adolescents and is recognized as a cause of multisystem toxicity. Whereas e-cigarette or vaping product use–associated lung injury is well characterized, kidney involvement is poorly defined, and biopsy-confirmed reports in children are exceptional [1–5]. We describe an adolescent with dialysis-requiring acute kidney injury after electronic cigarette exposure whose presentation mimicked a pulmonary–renal syndrome but proved to be biopsy-confirmed acute tubular injury.

Case report

A previously healthy 14-year-old boy presented with oliguria, dark-colored urine, severe hypertension, and three episodes of generalized tonic–clonic seizures. His history included two recent, unexplained episodes—transient somnolence with hallucination-like symptoms, and suspected hypertension—both with unremarkable evaluations, and a mild, self-limited upper respiratory infection. He took no medications and had no personal or family history of kidney, autoimmune, or hypertensive disease, and no known exposure to nephrotoxins. However, on further questioning, he disclosed regular use of electronic cigarettes in the period preceding symptom onset. On admission, he weighed 87 kg (+ 3.47 SDS) and measured 177 cm in height (+ 2.48 SDS). He was agitated and intermittently somnolent but remained oriented, with severe hypertension (160/100 mmHg), tachypnea, and bilateral pulmonary

crackles. There was no edema, rash, arthritis, or organomegaly. Brain computed tomography and diffusion-weighted magnetic resonance imaging were unremarkable despite recurrent seizures. Given the presence of severe hypertension, the neurological manifestations were attributed to hypertensive encephalopathy. Thoracic computed tomography showed right lower-lobe consolidation with ground-glass opacities and a centrally located cavitary lesion within an area of right upper-lobe consolidation (Fig. 1a), and kidney ultrasonography revealed increased cortical echogenicity without obstruction. A broad respiratory viral panel, including SARS-CoV-2, was negative.

Serum creatinine increased from 1.18 mg/dL at admission to a peak of 10.6 mg/dL on hospital day 5. (Fig. 1b). Urinalysis repeatedly demonstrated hematuria and proteinuria, with markedly dark-colored urine and a urine protein-to-creatinine ratio of 2.1 mg/mg. Complement levels were normal, serum albumin remained within the normal range (45 g/L), and antinuclear, anti–double-stranded DNA, and antineutrophil cytoplasmic antibodies were negative. Urinary β2-microglobulin was markedly elevated (6.45 mg/L; normal range 0–0.2 mg/L), indicating significant tubular injury. Oliguria progressed to anuria with fluid overload and transient left ventricular systolic dysfunction.

Given rapidly progressive kidney failure, anuria, hypertension, pulmonary infiltrates, and active urinary findings, a pulmonary–renal syndrome was considered. The child was admitted to intensive care and intermittent hemodialysis was started. Pending the work-up, pulse methylprednisolone and plasma exchange were given for possible rapidly progressive glomerulonephritis or vasculitis.

As serological testing remained unrevealing and complement levels were normal, a kidney biopsy was performed to establish the diagnosis and guide management. The biopsy demonstrated preserved glomeruli without proliferative or crescentic lesions. The interstitium showed only minimal focal edema with sparse mononuclear cell infiltration, insufficient to support acute tubulointerstitial nephritis. In contrast, the tubular compartment was prominently affected, with epithelial simplification, degenerative epithelial changes, intratubular erythrocyte casts, and necrotic cellular debris, consistent with acute tubular injury. Immunofluorescence studies were negative for IgG, IgA, IgM, C3, C4, C1q, and fibrinogen. The findings established acute tubular injury and excluded immune-complex glomerulonephritis and pauci-immune vasculitis (Fig. 1c, 1d).

The biopsy findings excluded immune-mediated kidney disease, allowing plasma exchange to be discontinued and corticosteroids to be tapered and withdrawn. Urine output recovered, hemodialysis was discontinued, and creatinine declined steadily. At 3 months, creatinine was 0.74 mg/dL with normal urinalysis and a urine protein-to-creatinine ratio of 0.08 mg/mg. At 18 months the child remained asymptomatic, with creatinine 0.63 mg/dL, normal urinalysis, and normal blood pressure off all antihypertensive therapy.

Discussion

This case illustrates three lessons about vaping-associated kidney injury in adolescents. First, electronic cigarette exposure can precipitate severe, dialysis-requiring acute tubular injury in a young person with

no prior kidney disease. Although vaping-related lung injury is well described, kidney involvement is poorly characterized; most reports involve adults, and histopathological confirmation is uncommon [5]. Kidney biopsy established isolated acute tubular injury following recent electronic cigarette exposure, with no evidence of glomerular or immune-mediated kidney disease. Second, the presentation closely mimicked a pulmonary–renal syndrome. Severe hypertension, dark-colored urine, hematuria, proteinuria, pulmonary infiltrates, neurological manifestations, and rapidly worsening kidney function initially suggested rapidly progressive glomerulonephritis or vasculitis, prompting pulse corticosteroids, plasma exchange, and hemodialysis while the evaluation proceeded. Serology was persistently negative, and the kidney biopsy ultimately redirected both diagnosis and treatment—excluding an immune-mediated process and sparing the patient prolonged, unnecessary immunosuppression. This emphasizes the diagnostic value of kidney biopsy when severe acute kidney injury mimics immune-mediated disease despite unrevealing serological findings.

The presentation closely resembled that of a previously reported 31-year-old man from Mexico who developed generalized tonic–clonic seizures, pulmonary injury, hematuria, and severe acute kidney injury following electronic cigarette use, with unremarkable neuroimaging and complete kidney recovery [6]. Unlike that patient, our patient required hemodialysis and underwent kidney biopsy, providing histopathological confirmation of acute tubular injury. Taken together, these cases suggest that severe vaping-associated toxicity can present with combined neurological, pulmonary, and kidney involvement, although the underlying renal pathology may remain unrecognized without biopsy [6]. Although the pathophysiological mechanisms remain incompletely understood, electronic cigarette aerosols contain numerous potentially nephrotoxic substances, including nicotine, tetrahydrocannabinol additives, heavy metals, aldehydes, flavoring compounds, and ultrafine particles [5]. Proposed pathways include direct tubular toxicity, oxidative stress, endothelial dysfunction, inflammatory injury, and ischemia. In our patient, the markedly elevated urinary β2-microglobulin and the tubular-predominant biopsy support a primary tubular process. Although intratubular erythrocyte casts may suggest glomerular bleeding, the preserved glomeruli and negative immunofluorescence findings indicate severe tubular injury as their source. Furthermore, the absence of glomerular lesions and the patient's complete recovery of kidney function are consistent with a predominantly tubular process. A limitation of this report is the absence of toxicological analysis of the vaping product and its constituents. Therefore, the specific agent responsible for the observed kidney injury could not be identified. Nevertheless, the onset of symptoms shortly after electronic cigarette exposure, together with the exclusion of alternative causes of acute tubular injury and the presence of concurrent pulmonary abnormalities, supports a vaping-associated etiology. Third, our case provides reassuring outcome data. Despite dialysis-requiring acute kidney injury and a peak creatinine level exceeding 10 mg/dL, the child achieved complete clinical and biochemical recovery, and kidney function remained normal at 18 months of follow-up. This suggests that vaping-associated acute tubular injury, despite its severity, may be fully reversible when recognized and managed promptly. In conclusion, electronic cigarette exposure should be considered in adolescents with otherwise unexplained severe acute kidney injury, particularly when pulmonary and neurological features coexist. Vaping-associated acute tubular injury may mimic a pulmonary–renal syndrome, and

kidney biopsy is invaluable for securing the diagnosis and avoiding unnecessary immunosuppression. Greater awareness may reduce diagnostic delay and prevent unnecessary immunosuppression in this emerging entity.

Declarations

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Competing interests The authors have no relevant financial or non-financial interests to disclose.

Consent to participate / publish Written informed consent for participation and for publication of the clinical details and images was obtained from the patient's parent/legal guardian.

Author contributions: All authors contributed to the conception and design of the study. CCP, BA, and AKB drafted the manuscript. AKB supervised the study. All authors contributed to the interpretation of the data, critically revised the manuscript, and approved the final version

Data availability: The data supporting the findings of this case report are available from the corresponding author on reasonable request.

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Figures

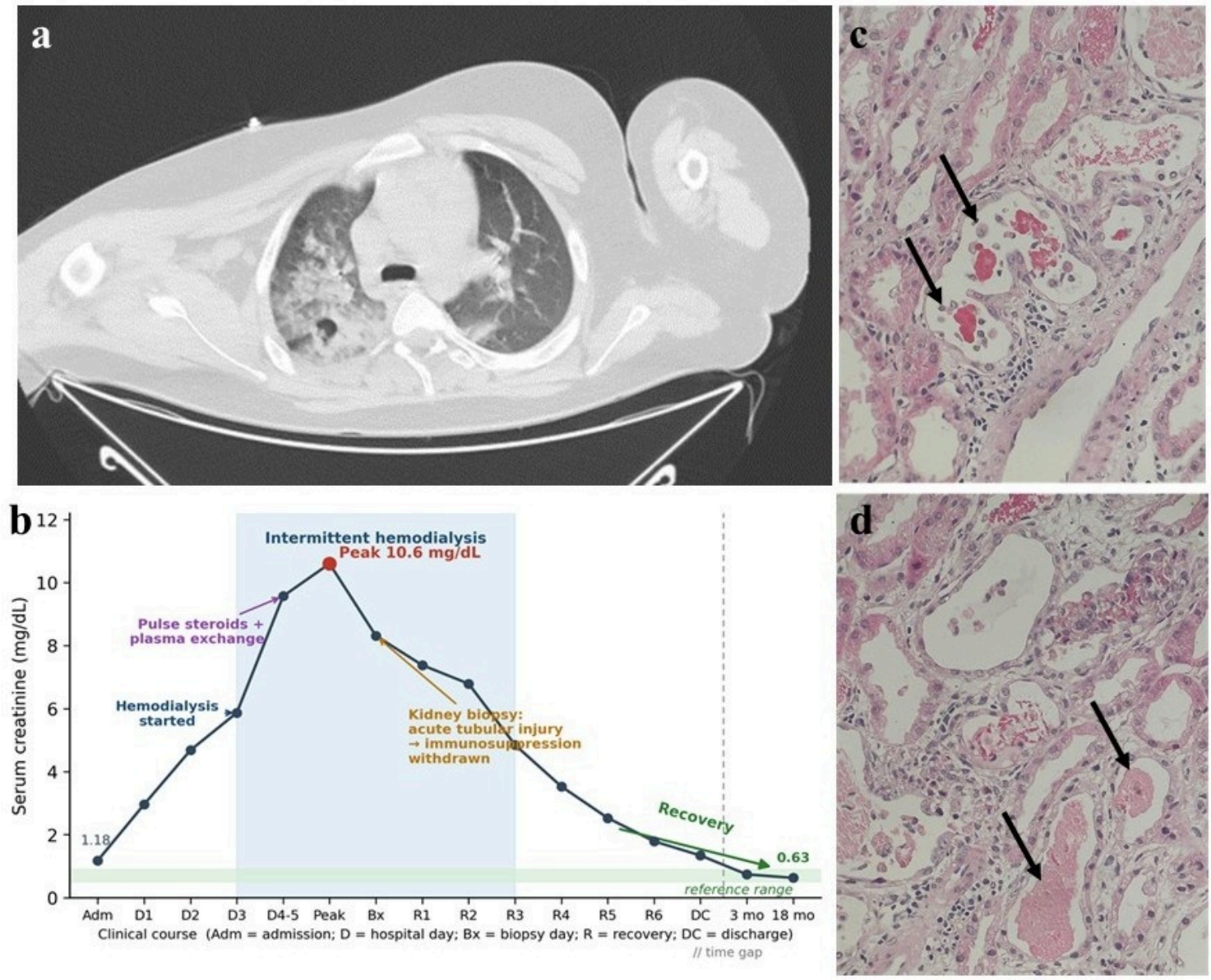


Figure 1

Clinical, pathological, and radiological findings. **a** Chest computed tomography demonstrates a cavitary consolidation in the central region of the right upper lobe. **b** Serum creatinine levels during follow-up **c** Light microscopy (H&E stain, ×200) shows diffuse acute tubular injury with detachment of tubular epithelial cells from the basement membrane and sloughing of epithelial cells into the tubular lumen. **d** Light microscopy (H&E stain, ×200) demonstrates red blood cell casts within the tubular lumen. No glomerular abnormalities are observed.