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# Kidney in chronic uncontrolled hypertension; mind the dual pathology

Dorsa Jahangiri<sup>1</sup>, Mohammadreza Ardalan<sup>2\*</sup>, Muhammed Mubarak<sup>3</sup>, Shahrzad Alimohammadi<sup>4,5</sup>,  
Hamid Reza Jahantigh<sup>6,7</sup>, Sanam Saeifar<sup>8</sup>

<sup>1</sup>Independent Researcher, 43185 Cardston Place Leesburg Virginia, 20176, USA

<sup>2</sup>Kidney Research Center, Tabriz University of Medical Sciences, Tabriz, Iran

<sup>3</sup>JIK Department of Histopathology, Sindh Institute of Urology and Transplantation, Karachi, Pakistan

<sup>4</sup>Doctoral School of Molecular Medicine, University of Debrecen, Debrecen, Hungary

<sup>5</sup>Department of Immunology, Faculty of Medicine, University of Debrecen, Debrecen, Hungary

<sup>6</sup>Interdisciplinary Department of Medicine - Section of Occupational Medicine, University of Bari, Bari, Italy

<sup>7</sup>Animal Health and Zoonosis PhD Course, Department of Veterinary Medicine, University of Bari, Bari, Italy

<sup>8</sup>Buchmann Institute for Molecular Life Sciences (BMLS), Cluster of Excellence Frankfurt Macromolecular Complexes (CEF-MC), Goethe University Frankfurt am Main, Frankfurt am Main, Germany

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### Implication for health policy/practice/research/medical education:

A patient with history of uncontrolled hypertension and frequent analgesic usage reported in this article. Based on clinical and paraclinical manifestations and renal biopsy, the diagnosis was thrombotic microangiopathy (TMA).

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## Kidney in chronic uncontrolled hypertension; mind the dual pathology

A 47-year-old male with a long-standing past medical history of uncontrolled hypertension and frequent analgesic usage was presented by nausea and headache as his first clinical manifestations. On admission, his blood pressure was 180/120 mm Hg, that was controlled by intravenous anti-hypertensive medication. The terminal examination revealed cotton exudates. The peripheral blood examination showed fragmented red blood cells (Schistocytes). Further laboratory investigations presented hemoglobin, 8.0 g/dL, lactate dehydrogenase, 950 IU/L, platelets, 117,000/ $\mu$ L, and serum creatinine level, 4.5 mg/dL. The diagnosis was thrombotic microangiopathy (TMA), since malignant hypertension could be associated with shear stress-induced endothelial damage and TMA (1). Renal biopsy demonstrated features of subacute TMA, chronic hypertensive vasculopathy, and chronic tubule-interstitial changes (Figure 1).

### Authors' contribution

Conceptualization: MA. Methodology: MM, ShA and HRJ.

Validation: DJ. Formal analysis: MA. Investigation: MA. Resources: DJ and SS. Data curation: DJ and SS. Writing—original draft preparation: MA and SS. Writing—review and editing: DJ and SS. Visualization: MA. Supervision: MA. Project administration: MA.

### Conflicts of interest

The authors declare that they have no conflicts of interest.

### Ethical issues

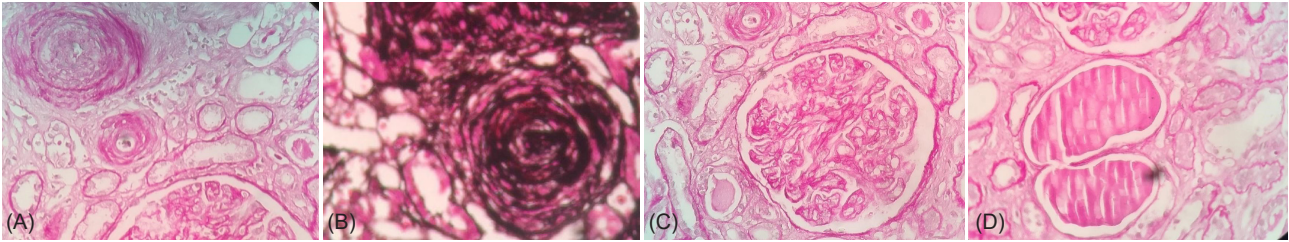
Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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### References

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**Figure 1. Renal biopsy revealed features of subacute TMA, chronic hypertensive vasculopathy, and chronic tubulointerstitial changes.** Two types of lesions were detected, those of nephrosclerosis and TMA, and thrombi are not needed to diagnose TMA. (A) Mild to moderate intimal fibroplasia of an arteriole, and intimal proliferation and mucoid changes markedly narrowing the arterial lumen. The former change signifies typical vascular lesion of benign nephrosclerosis and the later, subacute change is seen in TMA. Mild tubular atrophy is also seen in the background. (PAS,  $\times 400$ ). (B) Well established concentric "onion skinning" intimal fibroplasia of severe degree with almost complete occlusion of the lumen of a small artery. This lesion is characteristically by or with the malignant form of hypertension. (Jones' silver stain,  $\times 400$ ). (C) The glomerulus shows mesangiolytic, segmental reduplication of glomerular basement membranes (double contouring), and near total occlusion of the capillary lumens, signifying persistent endothelial injury of some durations. Hyperplastic arteriosclerosis and mild tubular atrophy are seen in the backdrop (PAS  $\times 400$ ). (D) Mild tubular atrophy. Two atrophic and dilated tubules are filled with hyaline casts (PAS  $\times 400$ ).

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