Kidney in chronic uncontrolled hypertension; mind the dual pathology

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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).

Keywords: Histopathology, Hypertension, Kidney

A R T I C L E  I N F O

Article Type: News and Views

Article History:
Received: 2 July 2022
Accepted: 18 August 2022
ePublished: 18 September 2022

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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/μL, 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.

Funding/Support
None.

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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, ×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, ×400). (C) The glomerulus shows mesangiolysis, segmental reduplication of glomerular basement membranes (double contouring), and near total occlusion of the capillary lumens, signifying persistent endothelial injury of some durations. Hyperplastic arteriolosclerosis and mild tubular atrophy are seen in the backdrop (PAS ×400). (D) Mild tubular atrophy. Two atrophic and dilated tubules are filled with hyaline casts (PAS ×400).

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