

Journal of Nephrology

Hypertension and renal failure with right arm pulse weakness in a 65 years old man

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ARTICLE INFO

Article type:

Short Communication

Article history:

Received: 1 June 2012

Revised: 20 June 2012

Accepted: 3 August 2012

Published online: 1 October 2012

DOI: 10.5812/nephrologist.8265

Keywords:

Antiphospholipid syndrome

Hypertension

Renal failure

Antiphospholipid syndrome

nephropathy

Anti- β 2 glycoprotein

Focal cortical atrophy

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

Antiphospholipid syndrome (APS) is being increasingly recognized as an important cause of kidney injury due to thrombosis at any location within the renal vasculature. The term antiphospholipid syndrome nephropathy (APSN) refers to the kidney damage caused by vascular lesions in the glomeruli, arterioles and/or interlobular arteries in patients with antiphospholipid antibodies. APSN manifests with hypertension, acute and/or chronic kidney failure, and often a low-grade proteinuria.

Please cite this paper as: Nasri H. Hypertension and renal failure with right arm pulse weakness in a 65 years old man. J Nephrology.2012; 1(3): 130-133. DOI: 10.5812/nephrologist.8265

Short
Communication

Case

A 65-year-old man was referred for evaluation of renal failure, detected during laboratory evaluation. Patient had a history of hypertension for 3 years before referral and a history of decreased right arm pulses too. In laboratory examinations, estimated glomerular filtration rate (eGFR) was 45 ml/min; urine sediment was bland and 24-hr urine protein excretion was 400 mg. For further evaluation, a kidney biopsy was performed. In the renal biopsy of 18 glomeruli, seven were totally sclerosed. There were 4 glomeruli devoid of tuft (glomerular bal-

looning) too. Of 11 live glomeruli, six had very retracted tuft with wrinkling of glomerular basement membrane (GBM) in Jones staining with cystic dilatation of Bowman's capsules. (figure: 1A-D). There was moderate interstitial fibrosis (around 50%), associated with tubular atrophy. There was a zone of tubular thyroidization too (figure 2A). There was tubular cell flattening and tubular cell vacuolization too. In vessels, arteriosclerosis, arteriolar occlusion by thrombosis and intimal fibrosis with hyperplasia (fibrose intimal hyperplasia; FIH) in interlobular arteries was seen (figure 2 B-D). Considering the above

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mentioned morphologic features, renal biopsy was reported consistent with antiphospholipid syndrome nephropathy (APS-nephropathy). The autoantibody panel (p-ANCA, c-ANCA, anti-nuclear antibody, anti-ds-DNA) was negative except for positive anti- β 2 glycoprotein 1(anti- β 2GPI) (total), which was 55.8 IU/mL (>18 positive). After 12 weeks, the tests re-checked. The result of measurement was as follows; anti- β 2GPI (total) was 74 IU/mL, Anti-phospholipid antibody (IgM)=22.5 IU/mL (>15positive), thus confirming primary APS- nephropathy.

Pathologic descriptions of APS- nephropathy (APSN)

Antiphospholipid syndrome is being increasingly recognized as an important cause of kidney

injury due to thrombosis at any location within the renal vasculature (1-3). The term antiphospholipid syndrome nephropathy (APSN) refers to the kidney damage caused by vascular lesions in the glomeruli, arterioles and/or interlobular arteries in patients with antiphospholipid antibodies (2-4). In nephrology point of view, this small-vessel vaso-occlusive nephropathy, may presented with hypertension acute and/or chronic kidney failure, and often a low-grade proteinuria clinically. The disease may be acute as thrombotic microangiopathy (TMA), and a spectrum of chronic lesions, such as arteriosclerosis, fibrous intimal hyperplasia (FIH), tubular thyroidization and focal cortical atrophy (FCA) (1-5). In acute phase, fibrin thrombi consisting fragmented RBCs, along with edema of endothelial cells, narrow or occlude the

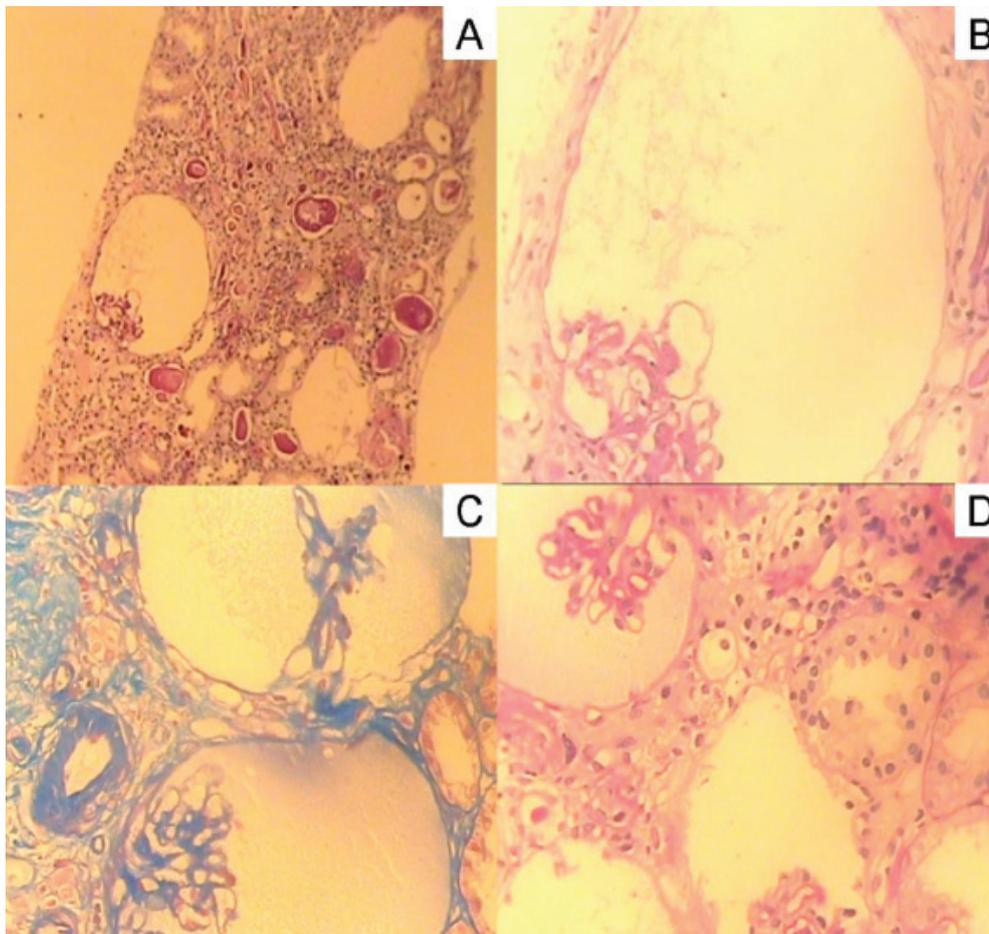


Figure 1A-D: Sever retracted glomerular tufts with cystic dilatation of Bowman's capsules'glomerular ballooning'. In figure1A, there was also aspect of tubular thyroidization in the interstitial area too. (H&E, PAS and Masson trichrome stains x400).

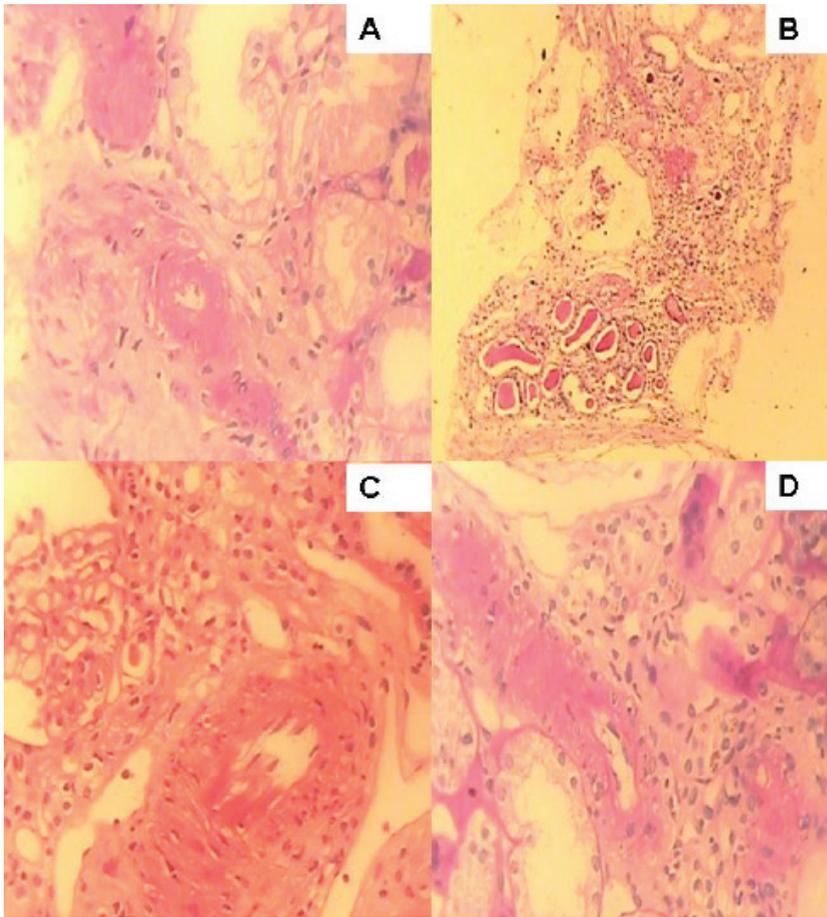


Figure 2A-D: Arteriosclerosis, arteriolar occlusion by thrombosis and intimal fibrosis with hyperplasia (fibroelastic intimal thickening) in interlobular arteries (H&E and PAS stain x400).

vascular lumen (5, 6). Thrombi finally organize into fibro-cellular and fibrous vascular occlusions, which can be recannulated resulting in onion-skin appearance of intimal fibrosis (3-6). Among the chronic morphological aspects, arteriosclerosis is typically seen associated with fibrous intimal hyperplasia, intimal thickening of the arteries and arterioles primarily by myofibroblastic cellular proliferation, with consequent lumen restriction and ischemia. Focal cortical atrophy involves the superficial cortex under the kidney capsule, as foci or triangles with sharp borders with the rest of the normal cortex, accompanied by depression of the contour of the renal capsule. In these atrophic areas, all elements of the renal parenchyma were altered in a pattern considered to be very typical of APSN (3-7). The glomeruli appear either small and sclerotic or voluminous, but may

be devoid of glomerular tuft named as 'glomerular ballooning'. The tubules change to atrophic packed with eosinophilic casts, resembling thyroid tissue, named a tubular thyroidization (2-8).

Acknowledgments

The author expresses his sincere appreciation to the staffs of division of renal immunopathology unit of Dr. Baradaran laboratory for performing and preparing the biopsy.

Financial Disclosure

The author declared no competing interests.

Funding/Support

None declared.

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