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From the Clinic

CKJ

Collapsing glomerulopathy with patchy acute cortical necrosis secondary to postpartum hemorrhage

Sir,

Collapsing glomerulopathy (CG) represents a distinct pattern of renal response to injury characterized by segmental to global collapse of capillaries in association with hyperplasia and hypertrophy of the visceral epithelial cells (VECs) associated with marked tubulointerstitial damage [1]. The reporting of CG in the literature has increased with the growing awareness among the nephrologists and pathologists of its association with disorders other than human immunodeficiency virus-1 infection [1–3]. Currently, it is classified as a variant of focal segmental glomerulosclerosis (FSGS) [3]. However, more recently, some authors have suggested that this relationship with FSGS may not last longer, and sooner or later it may be classified as a separate nosologic entity [2, 3].

With increasing awareness, the vascular lesions and thromboembolic phenomenon with consequent ischaemia have also emerged as important etiopathogenetic mechanisms in the development of CG in both the native and the transplanted kidneys [4–7]. More recently, the direct causal relationship between patchy infarction and *de novo* CG in transplanted kidneys has been reported [8]. However, no such link with acute cortical necrosis (ACN) secondary to post-partum haemorrhage (PPH) and hypovolaemic shock in native kidneys of young patients with no vasculopathy has been reported till date. We herein report two cases of CG involving the glomeruli in the vicinity of patchy ACN found on biopsies from native kidneys in two patients.

Both patients were young females, 17 and 26 years, respectively, and presented with acute renal failure (ARF) following PPH. No history of drug intake or past medical illness of note was elicited. Ultrasound findings were not typical of ACN. Urine analysis was non-contributory. Relevant viral and autoimmune serology was negative. Both patients required dialysis initially, but one is off dialysis and maintaining serum creatinine at 221 μmol/L 8 months post-biopsy, whereas the other is on haemodialysis, waiting for kidney transplantation 10 months after diagnosis.

Renal biopsies in both cases showed patchy infarction. In addition, both biopsies showed variable numbers of glomeruli in the vicinity of infarction, with segmental to global collapse of capillaries associated with hyperplasia and hypertrophy of VECs (Figure 1). There was moderate mixed inflammatory cell infiltration in the interstitium. However, no vasculopathy or thrombotic lesions were noted. Immunofluorescence was performed on snap-frozen tissue and showed segmental positivity of immunoglobulin M (IgM) and C3 in areas of collapsed tufts of viable glomeruli, whereas IgG, IgA and C1q were negative. Thus, both cases showed typical glomerular changes of CG involving the glomeruli in close proximity to patchy ACN.

A report of three cases of zonal distribution of CG in the vicinity of patchy infarction secondary to severed accessory renal vessels in the transplanted kidneys has recently been published [8]. Similarly, occasional reports are also available in the literature, in which an association of FSGS and CG in the native kidneys with the vascular lesions has been observed [4–6]. However, this is the first report of CG in association with ischaemic ACN secondary to hypovolaemia resulting from PPH in native kidneys in two young females and provides evidence for the broad etiopathogenetic pathways leading to the final common pattern of CG. We also believe that this lesion is of secondary or reactive nature, rather than primary or idiopathic CG [9]. There is very little information in literature on the clinical behaviour, treatment and prognosis of secondary forms of CG, but these might be determined by the underlying disease. In conclusion, this report highlights the close association of ischaemia with CG and further expands the spectrum of associations of this renal lesion.

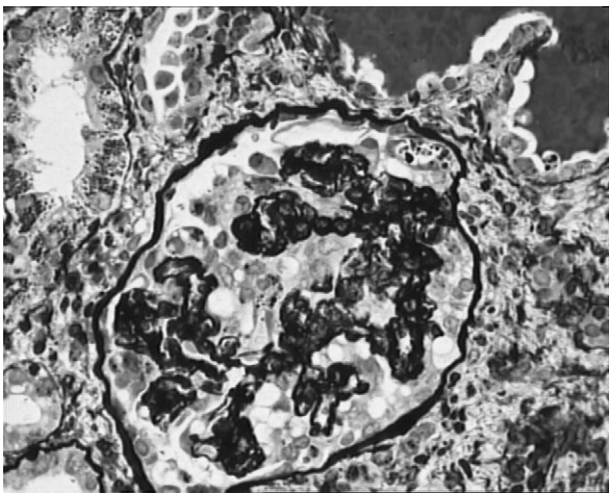

Fig. 1. High-power view showing a glomerulus from one of the renal biopsy specimens showing global collapse of capillary tufts associated with marked hypertrophy and hyperplasia of the podocytes. These show marked cytoplasmic vacuolization and protein resorption droplets in some cells. There is moderate tubulointerstitial inflammation in the background and one tubular lumen contains proteinaceous cast with scalloped margins (silver stain, x400).

Conflict of interest statement: None declared.

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