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# Acute renal failure in a young female with vaginal bleeding with partial recovery

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Glomerular collapse is one of the morphological patterns of response of kidney parenchyma to a variety of noxious agents. As such, it is commonly observed on renal biopsies showing a variety of diseases. It is important to report this lesion in the context of underlying major pathology so as not to confuse it with idiopathic collapsing glomerulopathy. The prognosis of this lesion is determined in such cases by the underlying pathology.

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## Clinical presentation

An 18-year-old female presented to nephrology OPD with anuria of five days duration and acute renal failure (ARF). She gave history of profuse bleeding per vagina, followed by dilatation and curettage (D&C) immediately before the presenting complaints. Her vitals were stable with blood pressure of 110/80 mmHg. The serum creatinine at the time of presentation was 14 mg/dL. Ultrasound of the abdomen showed normal size kidneys with normal echogenicity. Hepatitis B surface antigen (HbsAg) and anti-hepatitis C virus (HCV) were negative. The relevant autoimmune serology including antiphospholipid antibodies (aPLs) was also negative. Urine analysis showed 2+ albumin, 10-12 white blood cells (WBCs)/HPF, 1-2 red blood cells (RBCs)/HPF and occasional granular casts.

No history of medications usage or family history of renal disease could be elicited. Acute tubular necrosis (ATN) was suspected clinically and she was hemodialyzed a couple of times and her most recent serum creatinine is 3.8 mg/dL. Since she did not recover her renal function completely in due course of time, a renal biopsy was performed on day 34 of admission (Figure 1).

## What are the causes of kidney dysfunction in the above setting?

The most common cause of renal failure in the above setting is the ischemic renal disease, which spans a spectrum of lesions from variable degrees of acute tubular injury to ATN to full-blown diffuse acute cortical necrosis (ACN) (1,2). The later lesion, if diffuse and of severe degree, can be picked up on imaging studies and is now rarely biopsied. However, it is still a quite prevalent cause of ARF in many developing countries (1,2). Patchy or incomplete ACN may not be readily apparent on ultrasound and in such cases, renal

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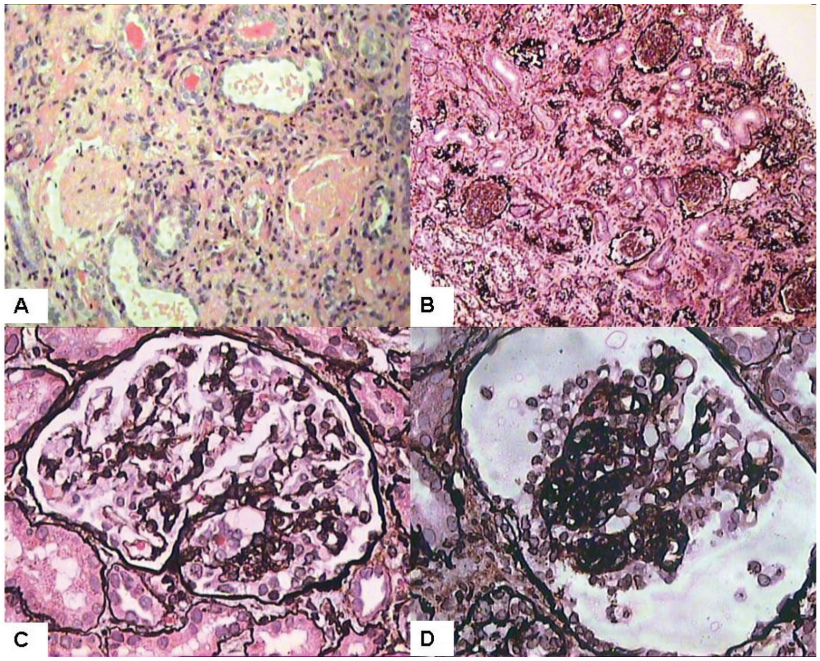


**Figure 1.** A. H&E stain showing infracted glomeruli. (H&E, ×200). B. Silver staining showing cortical infarction. (JMS, ×200). C. Segmental collapse of capillary tuft with podocyte hyperplasia. (JMS, ×400). D. Global collapse of capillary tufts with podocyte hyperplasia. (JMS, ×400).

biopsies, still continue to be performed, as in the present case. Other causes of ARF such as acute glomerulonephritis, vasculitides, and obstruction may also present in this setting but are rare.

## What are the kidney biopsy findings in the present case?

Her renal biopsy showed two cores of renal parenchyma with up to 24 glomeruli. Of these, 7 along with a significant part of the cortex were severely ischemic and hyalinized consistent with glomerular infarction (Figure 1A,B), five glomeruli in the immediate vicinity of the ischemic area showed segmental to global collapse of capillary tufts associated with marked hypertrophy and hyperplasia of visceral epithelial cells (VECs), as shown in Figure 1C, D. A high-power view in Figure 2 shows the lesion of CG in greater detail, with global collapse of capillary tufts, podocyte hyperplasia and hypertrophy, hyaline droplets in the cytoplasm of podocytes and formation of pseudo-crescent. Rest of the glomeruli appeared unremarkable. No vasculopathy was noted. There was moderate to severe degree of acute tubular injury and moderate tubular atrophy associated with interstitial inflammation and scarring in the background. Immunoflourescence (IF) showed trace and segmental positivity of immunoglobulin (Ig) M only, rest of the Igs and complement components were negative.

## What is the diagnosis?

A diagnosis of patchy ACN complicated by secondary collapsing glomerulopathy (CG) was rendered in this case.

## Describe the management and prognosis of this condition.

This is a case of patchy ACN secondary to hypovolemia caused by profuse vaginal bleeding in a young female. ACN represented an important cause of ARF of obstetrical origin in many developing countries (1,2). However, recent studies have shown a declining trend of this diagnosis in the setting of ARF (2). When ACN is global or diffuse, end-stage renal disease (ESRD) is inevitable. However, recovery of renal function is possible in cases of patchy ACN depending on the extent of cortical infarction (3). Renal biopsy at a later stage will disclose bands of fibrosis rep-

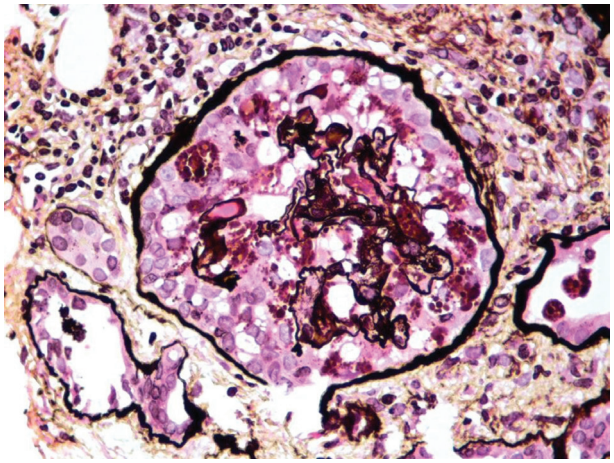

**Figure 2.** Global collapse of capillary tufts with marked podocyte hyperplasia resulting in pseudocrescentic appearance. There is moderate interstitial inflammation in the background. (JMS, ×400).

resenting healed infarction (3). The management of ACN has changed little over the years and depends on general supportive measures. Another important finding in this case consists of the changes of CG in the glomeruli in the vicinity of infarction. This is an interesting phenomenon and has only occasionally been described in literature (4,5). We have earlier described two such cases on native renal biopsies with different outcomes, as has been done by Canaud *et al.* on renal allograft biopsies (4,5). CG in this case represents a secondary change with different implications than those observed in the idiopathic form presenting with heavy proteinuria and needs no treatment (4). In fact, the prognosis of CG in this setting is determined by the extent of the underlying ischemic damage in the renal parenchyma.

This case highlights the importance of recognizing the lesions in their proper context and adds to the growing list of secondary CG in different settings.

## Final diagnosis

Patchy acute renal cortical necrosis complicated by secondary collapsing glomerulopathy.

## Authors' contributions

All authors wrote the manuscript equally.

## Conflict of interests

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