

DIAGNOSIS

Based on the clinical history of hypovolaemic shock as an obstetric complication and the characteristic findings of a hypo-intense renal cortex on computed tomography, a diagnosis of renal cortical necrosis (RCN) was made. RCN is a rare cause of acute kidney injury (AKI) in developed countries, characterised by oligo-anuria with typical computed tomography findings of a hypo-intense renal cortex in the early stages and cortical calcifications in later stages.¹ Traditionally, RCN is associated with obstetric complications such as (pre-)eclampsia, uterine haemorrhage and puerperal sepsis. Rarer causes include sepsis, hypercoagulability and major surgery.^{2,3}

Although the exact pathogenesis remains incompletely understood, a number of factors causing impaired renal cortical perfusion contribute to the development of RCN, notably arterial vasospasm, arterial thrombosis, endothelial damage and circulatory shock. Several physiological and pathophysiological changes in pregnancy explain the association between RCN and obstetric complications: the increased susceptibility of the arterial circulation to vasopressors, the hypercoagulable state and the possibility of endothelial damage due to pre-eclampsia, HELLP syndrome or exposure to foetal material during delivery or abortion.³

In a clinical setting of arterial vasoconstriction combined with endothelial damage, the renal cortex is particularly vulnerable as the renal vasculature is especially sensitive to endothelin, a potent endothelium-derived vasopressor, thus exacerbating arterial vasospasm when there is concomitant

endothelial damage. The renal medulla, which operates at low tissue oxygen levels in normal physiology, can more easily switch to oxygen-independent metabolism, which explains why the ischaemia only affects the cortex.

The incidence of RCN decreases with improving maternal care, accounting for less than 2% of AKI in Europe, compared with 7% in developing nations.^{3,4} However, as computed tomography and renal biopsy are not routinely performed in critically ill patients with AKI, RCN may be under-diagnosed. The diagnosis should be considered especially in obstetric patients, where 20% of AKI is due to RCN. Therapy for RCN is supportive. The prognosis is poor compared with many other causes of AKI, with less than one-third of patients partially recovering renal function.³ This partial recovery may be explained by sparing of the juxtamedullary glomeruli. After being dependent on haemodialysis for 36 days, our patient's renal function gradually recovered to an estimated glomerular filtration rate of 26 ml/min.

REFERENCES

1. Kim HJ. Bilateral renal cortical necrosis with the changes in clinical features over the past 15 years (1980-1995). *J Korean Med Sci.* 1995;10:132-41.
2. Gopalakrishnan N, Dhanapriya J, Muthukumar P, et al. Acute kidney injury in pregnancy--a single center experience. *Ren Fail.* 2015;37:1476-80.
3. Prakash J, Singh VP. Changing picture of renal cortical necrosis in acute kidney injury in developing country. *World J Nephrol.* 2015;4:480-6.
4. Prakash J, Vohra R, Wani IA, et al. Decreasing incidence of renal cortical necrosis in patients with acute renal failure in developing countries: a single-centre experience of 22 years from Eastern India. *Nephrol Dial Transplant.* 2007;22:1213-7.