Clinical Image

Lack of Blood Flow in Bilateral Renal Cortex

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CLINICAL IMAGE

Renal cortex-to-medulla blood flow redistribution is rarely observed on clinical images [1], and when observed, it could be taken as a sign of bilateral renal cortical necrosis, a rare form of acute kidney injury (AKI) [2]. We present a clinical example of redistribution.

The pictures presented are computed tomographic (CT) images of an 84-year-old woman who developed acute kidney injury (AKI) after undergoing surgical resection for gastric cancer. She was anuric with a serum creatinine level of 469 µmol/L (177 µmol/L, preoperatively) after an episode of postoperative hypotension, and was suspected to have hypovolemic shock-induced AKI. A CT scan performed to check for intra-abdominal bleeding detected neither hematoma nor active bleeding. However, a contrast-enhanced CT scan of the parenchymal phase demonstrated enhancement of the renal pyramids, but not the renal cortex, though renal cortex enhancement is normally expected. Volume resuscitation by hydration recovered the patient’s renal function. After several intermittent hemodialysis sessions, serum creatinine levels of approximately 265 µmol/L were sustained without further dialysis.

Lack of blood flow in the renal cortex on both sides is a characteristic sign of bilateral renal cortical necrosis, which is often noted in patients with systemic hypotension in the context of hemorrhagic shock [3]. Because of the occurrence of AKI after hypovolemic shock, bilateral renal cortical necrosis was suspected in the current case, and the CT scan findings supported the diagnosis. However, in contrast to the reported cases of bilateral renal cortical necrosis, in which chronic renal replacement therapy was needed [2], the current case had renal function recovery.

The recovery of renal function indicates that this case is a clinical example of renal cortex-to-medulla blood flow redistribution. In experimental renal cortex-to-medulla blood flow redistribution, ischemia-induced intrarenal hemodynamic changes, which are reversible by volume resuscitation, cause the redistribution [4]. The hemodynamic changes can conserve body fluid by preventing blood flow from reaching glomeruli in the renal cortex, and constriction of subcortical arterioles by vascular nerve activation may cause such intrarenal hemodynamic changes. Although constriction is a physiological response to hypovolemia, vascular nerve overstimulation during severe hypovolemia can induce vasospasm of the arterioles. On renal images, the vasospasm presents as a characteristic sign of bilateral renal cortical necrosis, occurring due to a lack of blood flow in the renal cortex on both sides, as shown in the current case. Therefore, proactive volume resuscitation is recommended in cases under suspicion of bilateral renal cortical necrosis. Restoring intrarenal hemodynamics can recover renal function in cases of renal cortex-to-medulla blood flow redistribution.

REFERENCES


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