Page Kidney in a Hemodialyzed Patient

Dear Sir,

Perirenal hematoma is the most common cause of Page kidney [1,2]. Compression of the parenchyma by a perirenal hematoma decreases blood flow through the kidney. Consequent to the parenchymal compression, the renin-angiotensin-aldosterone system is activated, resulting in hypertension [3,4].

We recently experienced a new case of Page kidney seen in a patient undergoing hemodialysis. As far as we know, although there have been numerous reports dealing with Page kidney, no case in hemodialyzed patients has ever been reported. The paucity of such case reports prompted us to describe our experience.

A 45-year-old man was admitted to our hospital due to sudden-onset right hypochondriac pain. For 4 years prior to admission, he had been undergoing regular hemodialysis for chronic renal failure, and he was anuric.

Computed tomography (CT) on admission suggested that a right perinephric hematoma was expanding to the retroperitoneal space (fig. 1). Emergent renal angiography revealed that the atrophic right kidney was compressed by an avascular component (fig. 2). On the basis of the CT findings and the decline in his hematocrit, the bleeding volume was estimated to be approximately 1,000-1,500 ml. A detailed history failed to identify the cause of the perirenal hematoma.

On his seventh day in hospital, a right nephrectomy was electively carried out. The removed kidney was atrophic, compatible with chronic renal failure, and consisted of multiple acquired cysts. An organizing hematoma was found to be compressing the renal parenchyma without involvement of the renal hilar vessels. On microscopic examination of the nephrectomized kidney, no neoplasm was revealed in either the cysts or the remnant renal tissue. Spontaneous rupture of an acquired cyst seemed the most likely cause of the perirenal hematoma in this patient.

Before admission, a long-acting angiotensin-converting enzyme (ACE) inhibitor and a calcium antagonist had been prescribed for control of blood pressure which was maintained at around 150/90 mm Hg. His blood pressure after admission was 200/100 mm Hg, despite the massive
bleeding. In addition, an alternative short-acting calcium antagonist was also necessary, because his blood pressure often became elevated to 230/110 mm Hg. After the nephrectomy, his blood pressure gradually decreased to 160/95 mm Hg. Plasma renin activity (PRA) fell from 3.35 before to 1.95 ng/ml/h (normal, 0.5-2.0) after the operation. During admission, his dry weight and the regime of reular hemodialysis were not changed.

At present (4 months after the nephrectomy), his blood pressure is easy to control with a long-acting ACE inhibitor and a calcium antagonist, as it was before admission. These findings indicated that the compression of the atrophic kidney by the perirenal hematoma exacerbated his hypertension.

Even if a hemodialyzed patient is anuric, some renal function, especially endocrine activity is sometimes conserved. For example, it is well known that bilateral nephrectomized patients experienced worse renal anemia. It is thought that the kidneys can produce natural erythropoietin after renal replacement therapy is initiated.

Therefore, it is not surprising that when the kidney of our patient was compressed by the perirenal hematoma, the renin-angioten-sin-al-dosterone system could be activated in the kidney. Unfortunately, we did not do renal vein renin sampling, which is often helpful for an accurate diagnosis [5].

In addition, in this context, patients with end-stage renal disease (ESRD) can develop Goldblatt’s hypertension as well as Page kidney. Therefore, physicians should be aware that these complications can occur in patients with ESRD, and that hypertension can be reversible if appropriate medical or surgical treatment is given.

References

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