CLINICAL STUDY

Diffuse Renal Cortical Hemorrhage in the Setting of Subcapsular Hematoma: Diagnosis and Treatment with Embolization

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ABSTRACT

Purpose: To describe radiologic findings, embolization technique, and clinical outcomes in patients with renal subcapsular hematoma and diffuse cortical hemorrhage.

Materials and Methods: Ten patients with renal subcapsular hematoma and diffuse cortical hemorrhage were reviewed. Nine of the 10 had undergone procedures (nephrostomy, n = 4; biopsy, n = 4; embolization of a cerebral aneurysm, n = 1) and 1 patient was receiving oral anticoagulation. Computed tomography (CT), angiography, and embolization of bleeding sites were performed in all patients.

Results: CT and angiography revealed subcapsular hematoma with diffuse cortical hemorrhage at the level of the interlobar and/or arcuate branches. Total embolization of intrarenal arterial branches was required in 3 patients. Partial embolization, which also resulted in permanent functional loss, was required in 4. The functional loss was likely caused by the embolization procedure and the underlying renal disease. In these 4 patients, renal failure was demonstrated by scintigraphy in 3 cases and based on the need to start chronic hemodialysis in 1 case. In the remaining three patients, embolization did not compromise renal function.

Conclusions: Diffuse cortical hemorrhage unrelated to the site of puncture may be seen in some cases of subcapsular hematoma. The cause is likely the laceration of transcortical capsular arteries secondary to enlargement of the subcapsular hematoma. In the present case series, embolization achieved hemorrhage control, but loss of renal function was observed in patients with underlying renal disease.

Iatrogenic injuries from renal biopsies are the most common causes of renal vascular lesions, followed by percutaneous nephrostomy and percutaneous nephrolithotomy (1–3). Although most are minor and resolve spontaneously, the development of clinically significant complications such as severe bleeding, persistent hematuria, and high-flow shunting may require surgical or angiographic procedures in 0.22%–2% of cases (4).

Iatrogenic vascular lesions are directly related to the puncture site of percutaneous procedures. These lesions are most commonly found in the lower pole of the kidney, as this is the access site preferred by interventionalists (5). They are usually self-limited and resolve spontaneously, but they may gradually expand in some cases and lead to complications such as perirenal hematoma formation, severe bleeding, and even hemodynamic instability. Another complication of subcapsular hematomas is Page kidney, which refers to parenchymal compression caused by a subcapsular hematoma associated with hypertension and renal ischemia (6). A rare complication is the development of a subcapsular hematoma that secondarily results in laceration of the transcortical capsular branches. This complication has been described in patients undergoing stent placement for renal artery stenosis when guide wire perforation results in hematoma formation (7–9). To the best of our knowledge, other underlying causes of this type of complication have not been reported so far.

The present report focuses on the radiologic findings, treatment, and clinical outcomes in a series of patients in whom diffuse cortical hemorrhage developed as a complication of a subcapsular hematoma. The proposed mechanism and risk factors for this complication, as well as the embolization procedure, are discussed.

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None of the authors have identified a conflict of interest.

Figure E1 is available online at *www.jvir.org*.

J Vasc Interv Radiol 2017; ■:1–6

http://dx.doi.org/10.1016/j.jvir.2017.06.017

MATERIALS AND METHODS

This retrospective review of a hospital database was granted exemption from full review by the institutional review board. Patients included were treated between January 2005 and December 2013. Clinical data, laboratory data, imaging studies, treatment, and outcomes in a series of 10 consecutive patients in whom diffuse cortical hemorrhage developed as a complication of a subcapsular hematoma were retrospectively reviewed.

The series included eight male patients and two female patients with a mean age of 60.2 years (range, 29–83 y). Four patients had urinary tract obstruction secondary to retroperitoneal fibrosis, ureteral tumor, vesical tumor, or acute pyelonephritis caused by upper urinary tract calculi, respectively; four patients presented with impaired renal function, increased serum creatinine level, and decreased glomerular filtration rate secondary to acute tubular necrosis after transplantation, HIV-related nephropathy, graft dysfunction, and systemic vasculitis, respectively; one patient was receiving oral anticoagulation for cardiac arrhythmia; and one patient had undergone embolization of a cerebral aneurysm 6 hours before the onset of hemorrhage symptoms.

The procedures that caused renal hemorrhage were nephrostomy in four patients, renal biopsy in four, and embolization of a cerebral aneurysm in the patient receiving dual antiplatelet therapy. The 10th patient presented with spontaneous renal bleeding related to oral anticoagulation, which led to a diagnosis of Wunderlich syndrome.

All patients presented with pain, clinical signs of hypotension, and low hematocrit level. Patients required a mean of 5 U of packed red cells (range, 0–12 U). Six of the 10 patients showed clinical signs of hemodynamic instability, defined in our institution as systolic blood pressure < 80 mm Hg. All four patients with a percutaneous nephrostomy showed bleeding from the nephrostomy tube. Ultrasound (US) images showed renal subcapsular hematoma in all patients. **Table 1** shows the baseline characteristics of the patients.

The mean time between clinical onset and CT examination was 3.9 days (range, 6 h to 14 d). A subcapsular hematoma measuring 3–5 cm in thickness was seen in all patients. The hematomas compressed the renal parenchyma, flattening or deforming the renal contour. In all cases, signs of capsular rupture and bleeding into the perirenal space or the retromesenteric or retrorenal planes were present. Intravenous iodinated contrast medium was not administered to two patients because of renal failure. A multiphasic CT scan including a late arterial and/or venous phase was performed in the remaining eight patients. When the contrast agent could be administered, all patients except one showed small (< 1 cm) pseudoaneurysms located in the subcapsular hematoma, adjacent to the parenchymal edge. The number of pseudoaneurysms identified on CT images ranged from 1 to 11 per patient, and they were best seen on late arterial images. When multiple foci could be seen, they showed a diffuse distribution (**Fig 1**). Three patients, including the one in whom a pseudoaneurysm could not be demonstrated on imaging, exhibited active bleeding.

Within 24 hours after the CT scan, angiography was performed in eight patients with CT signs of active bleeding and in one patient with enlargement of the subcapsular and perirenal hematomas (nonenhanced CT) and hemodynamic instability. In the remaining patient with acute tubular necrosis after kidney transplantation, angiography was performed 7 days after the nonenhanced CT scan because there was a chance to salvage the transplant.

The angiography and embolization procedures were performed by using an Axiom Artis system (Siemens, Erlangen, Germany). The patient's right femoral artery was accessed percutaneously. A 0.035-inch hydrophilic guide wire (Radiofocus; Terumo Europe, Leuven, Belgium) and a 5-F Sidewinder or 5-F Cobra catheter (Radifocus; Terumo Europe) were used for selective catheterization of the renal arteries. Digital subtraction angiography was performed with the use of nonionic iodinated contrast medium (370 mg/mL) at volume and injection rates of 12 mL and 3 mL/s, respectively, when injected in the main renal artery and 6–8 mL and 1.5–2 mL/s, respectively, when injected in the interlobar arteries. Superselective catheterization of the bleeding vessels was performed by using a 2.7-F (Progreat; Terumo Europe) or 2.4-F (Excelsior; Stryker, Kalamazoo, Michigan) microcatheter. Microcoils 2–2.5 mm in size (VortX or Interlock-18; Boston Scientific, Marlborough, Massachusetts) were used for selective embolization of the bleeding vessel. Polyvinyl alcohol particles 150–250 μm (Contour; Boston Scientific) or 100–300 μm in size (BeadBlock; Terumo Europe) were used when diffuse bleeding was observed. When total embolization was performed, 3–5-mm microcoils (VortX or Interlock-18; Boston Scientific) were deployed after the embolization particles.

Angiographic images demonstrated multiple bleeding points of nodular shape (ie, pseudoaneurysms) or a "watering-can" appearance (7) at the level of the arcuate and interlobular branches (**Fig 1**). Bleeding from the directly injured artery associated with diffuse cortical bleeding was seen in four patients. In one patient, round contrast-enhancing lesions that were considered microaneurysms were not treated. An angiogram obtained 6 days later showed complete resolution of the lesions (**Fig E1** [available online at *www.jvir.org*]). Final levels of embolization were as follows: main renal artery in two patients, segmental branches in one, interlobar in five, and arcuate branches in two.

RESULTS

Total embolization with permanent embolic agents that resulted in organ loss was performed in three patients. A more selective embolization of the interlobar arteries was performed in five patients, but the procedure, together with the underlying renal disease, resulted in permanent functional organ loss in four of the five. This was confirmed by renal scintigraphy in three cases, and the fourth patient started dialysis. In the fifth patient, a follow-up CT scan showed that embolization resulted in 50% infarction of the renal parenchyma and 50% preservation of renal function.