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Case Report

Papillary renal cell carcinoma with massive hematoma mimicking hemangioma

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ABSTRACT

It is extremely rare that papillary renal cell carcinoma has a massive hemorrhage. We report a case of papillary renal cell carcinoma with a massive hemorrhage which showed hemangioma-like imaging findings such as a globular discontinuous enhancement on the corticomedullary phase with a gradual centripetal fill-in pattern on the excretory phase on computed tomography and heterogeneously hyperintensity on T2-weighted magnetic resonance imaging. We also discuss a plausible mechanism explaining such imaging findings, with reference to pathological findings.

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Introduction

The papillary renal cell carcinoma (PRCC) is a histological subtype of RCC and constitutes approximately 13%–15% of all RCC [1]. Microscopically, PRCC is predominantly papillary or tubulopapillary, often with a thick fibrous capsule, foam cells,

necrosis, hemorrhage, and multifocality [2]. Delahunt and Eble defined 2 morphological subtypes of PRCC. Type 1 PRCC consists of papillae and tubular structures enclosed with a single layer of small cells with pale cytoplasm and low-grade nuclei. In type 2 PRCC, the cells are often of higher nuclear grade compared to the cells of type 1 PRCC with abundant eosinophilic cytoplasm and pseudostratified nuclei on papil-

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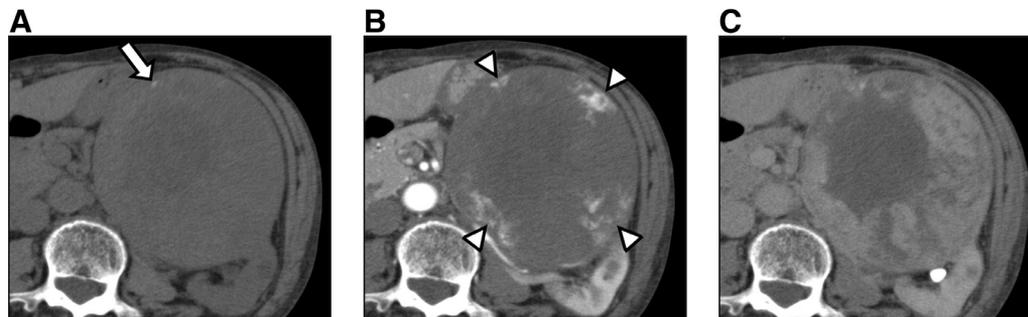


Fig. 1 – (A) On unenhanced CT, a large mass (11 cm in diameter) in the left kidney has spotty calcification (arrow). **(B)** On the corticomedullary phase, the peripheral parts (arrowheads) of the mass were focally enhanced as strongly as arteries. **(C)** A gradual centripetal fill-in enhancement was seen through the excretory phase.

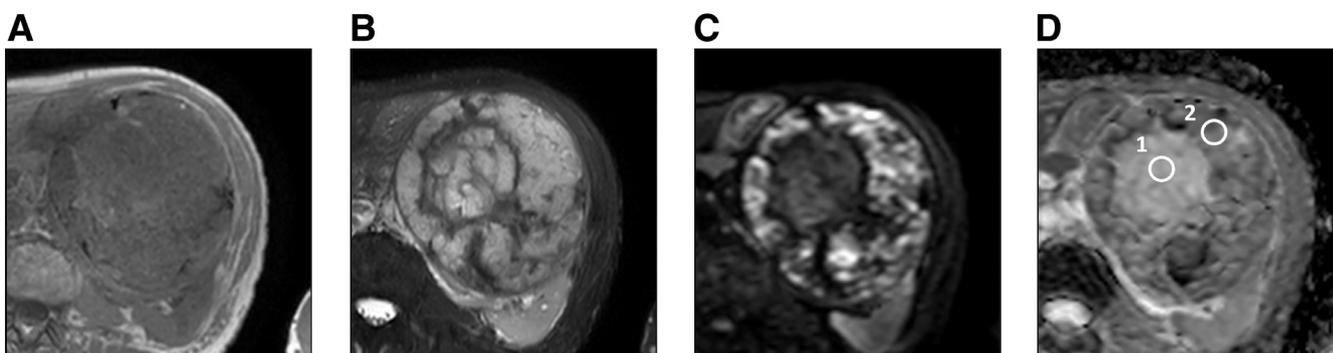


Fig. 2 – (A) In-phase chemical shift gradient-echo image, **(B)** fat-suppressed T2-weighted image, **(C)** diffusion-weighted image, and **(D)** apparent diffusion coefficient (ADC) map. Most parts of the mass showed isointensity to the renal parenchyma on the in-phase of the chemical shift gradient-echo image **(A)**, while they were hyperintense on the fat-suppressed T2-weighted image **(B)**, and hyperintense on the diffusion-weighted image **(C)**. Some parts of the mass were hypo- to hyperintense on the in-phase of the chemical shift gradient-echo image **(A)**, hypointense on the fat-suppressed T2-weighted image **(B)**, and hypointense on the diffusion-weighted image **(C)**. **(D)** The ADC of region of interest 1 (circle with number 1) was $2.3 \times 10^{-3} \text{ mm}^2/\text{s}$ on the central part of the mass. The ADC of region of interest 2 (circle with number 2) was $1.2 \times 10^{-3} \text{ mm}^2/\text{s}$ on the peripheral part.

lary cores. PRCC is usually associated with a more favorable prognosis compared to clear cell RCC, and type 2 PRCC carries a worse prognosis compared to type 1 PRCC [3].

In general, PRCC is reported to be hypovascular and homogenous on contrast-enhanced computed tomography (CT) [4]. On magnetic resonance imaging (MRI), most PRCCs show hypointensity on both T1- and T2-weighted images due to the deposition of hemosiderin and calcium or the densely collagenous nature of PRCC [5]. Based on these findings, PRCC is distinguished from other subtypes of RCC in daily practice.

We present a case of PRCC which showed atypical imaging findings, namely a “hemangioma-like appearance,” and also discuss a plausible mechanism to explain such imaging findings.

Case report

A 74-year-old female was referred to our hospital for evaluation of a left renal mass detected by unenhanced CT, which

had been administered to evaluate extraluminal compression found by screening gastroscopy at her previous hospital. She had neither clinical symptoms nor a history of abdominal trauma. She did not take anticoagulants. There were no abnormal values in her laboratory data (WBC $4730/\text{mm}^3$, Hb 12.0 g/dL, Plt $160 \times 10^4/\text{mm}^3$, BUN 18 mg/dL, Cr 0.79 mg/dL, AST 14 IU/L, ALT 8 IU/L, LDH 203 IU/L, CRP 0.11 mg/dL, PT 12.4 sec, PT-INR 1.02, APTT 28.8 sec).

Triple-phase CT revealed a mass of 11 cm in diameter in the left retroperitoneum (Fig. 1). The beak sign between the mass and left kidney suggested left renal origin. Calcification was seen on the peripheral part of the mass on unenhanced CT. On the corticomedullary phase, the peripheral parts of the mass were focally enhanced as strongly as arteries. A gradual centripetal fill-in enhancement was seen through the excretory phase.

On MRI, chemical shift gradient-echo imaging showed an absence of fat in the mass. Most parts of the mass showed hypointensity on chemical shift gradient-echo images and hyperintensity on fat-suppressed T2-weighted images. Some parts of the mass were hyperintense on chemical shift

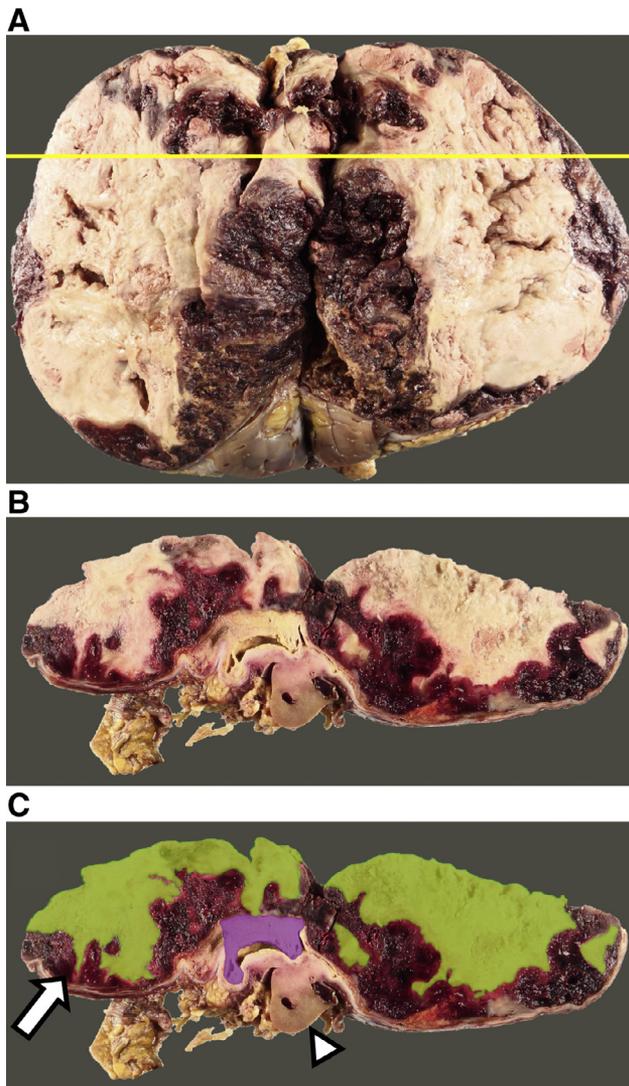


Fig. 3 – (A) Photomicrograph of the parting plane of formalin-fixed specimen. The black line represents cut surface B. (B) The mass contained a large amount of necrosis in the center (C, green area) surrounded by a large amount of hematoma (arrow). Only a small amount of PRCC (C, purple area) was found in the peripheral part adjacent to the normal kidney (arrowhead).

gradient-echo images and hypointense on fat-suppressed T2-weighted images, suggesting hemorrhage. The apparent diffusion coefficient (ADC) value was slightly low ($1.2 \times 10^{-3} \text{ mm}^2/\text{s}$) on the peripheral enhancing part of the mass, and high ($2.3 \times 10^{-3} \text{ mm}^2/\text{s}$) on the central part of the mass (Fig. 2). From these findings, differential diagnosis included hemangioma, angiosarcoma, and chronic expanding hematoma. The possibility of malignancy could not be excluded because of the large size of the mass, and left nephrectomy was performed. On gross pathological examination, the mass was encapsulated, and necrosis was seen at the center of the mass surrounded by a large amount of hematoma (Fig. 3). Microscopic examination revealed a minute amount of carcinoma cells on

the border between the mass and the normal kidney. The carcinoma cells contained abundant and eosinophilic cytoplasm and proliferated in a papillary fashion. Diagnosis of type 2 PRCC was made. In the hematoma, there were many vascular channels lined by a single layer of endothelial cells as seen in a hemangioma (Fig. 4).

Discussion

In previous reports, PRCC has usually appeared as a mildly high density mass on unenhanced CT, occasionally with calcification (7%-32%) [4,6]. On double-phase CT, PRCC shows a gradual and homogenous enhancement pattern [7]. On MRI, PRCC exhibits hypointensity with a homogeneous pattern on T2-weighted images [5]. Taouli et al. reported that the mean ADC value of PRCC ($1.12 \pm 0.18 \times 10^{-3} \text{ mm}^2/\text{s}$) was significantly lower than that of nonpapillary RCC (mostly clear cell carcinomas) ($1.62 \pm 0.73 \times 10^{-3} \text{ mm}^2/\text{s}$) [8].

Overall, the renal mass of our case showed imaging findings quite different from the typical ones described above and was difficult to diagnose as PRCC preoperatively. Histologically, the mass in our patient contained a large amount of hematoma and only a small amount of cancer cells. We considered that this large region of hematoma caused the hemangioma-like imaging findings, since the hematoma contained many vascular channels lined by a single layer of endothelial cells as seen in a hemangioma. Although PRCC is well known to include hemorrhage, it is extremely rare that PRCC involves massive hematoma. Yamamuro et al. reported a case of PRCC mimicking a hemorrhagic renal cyst [9]. However, the authors did not show the contrast enhancement pattern of the tumor. On the other hand, Ishigami et al. reported a case of adrenal adenoma, not PRCC, with an organizing hematoma mimicking hemangioma [10]. Our case had imaging findings similar to both these hemorrhagic tumors. Thus the observation of such imaging findings may not be limited to PRCC but may also occur in other tumors. How does a tumor showing such a hemangioma-like imaging appearance occur? One plausible mechanism would involve the “negative spiral theory” of wound repair. This process is initiated by a blood clot accumulating in the closed space due to various causes of bleeding. Next, necrosis, fibrosis, hyalinization, neovascularization, and vascular dilatation occur in turn. Finally, rebleeding is again observed [11,12]. In the present case, we speculated that repeated bleeding from a PRCC might have formed a large organizing hematoma according to the negative spiral theory. The neovascularization and vascular dilatation would contribute to the observation of imaging findings like those of a hemangioma.

The ADC on the peripheral enhancing part of the mass ($1.2 \times 10^{-3} \text{ mm}^2/\text{s}$) was similar to that of PRCC reported previously. However, this value should represent organizing hematoma itself in the present case. The ADC of organizing hematoma would change according to its age. Therefore, we do not consider that ADC may be useful to diagnosis of this type of PRCC. High ADC on the central part of the mass should derive from necrosis observed pathologically.

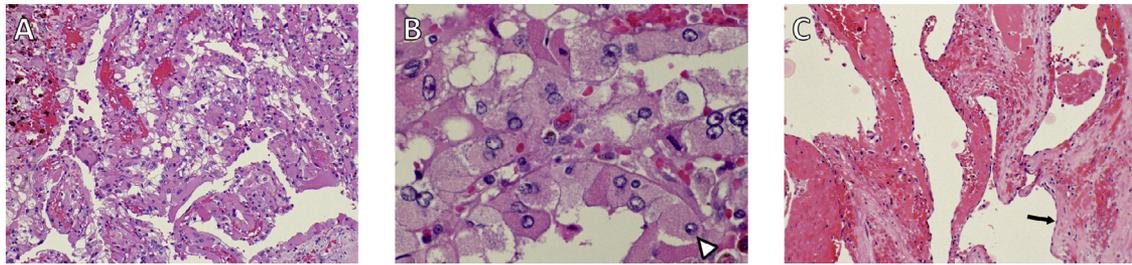


Fig. 4 – Photomicrograph of hematoxylin-eosin stained specimens. (A) Tumor cells proliferated in papillary fashion (magnification, $\times 100$). (B) Tumor cells (arrowhead) had abundant and eosinophilic cytoplasm (magnification, $\times 400$). (C) Vascular channels lined by a single layer of endothelial cells (arrow) proliferated as in a hemangioma (magnification, $\times 100$).

We reported a case of PRCC with massive hematoma, which mimicked a hemangioma radiologically. Radiologists should consider PRCC as a differential diagnosis for a renal tumor showing a hemangioma-like appearance.

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