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Spontaneous regression of primary renal cell carcinoma following image-guided percutaneous biopsy

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ABSTRACT

Spontaneous regression of metastatic renal cell carcinoma (RCC) is a rare but well-described clinical phenomenon; spontaneous regression of nonmetastatic RCC has been reported far less frequently. We present three cases of primary RCC that regressed spontaneously following the image-guided biopsy that established their diagnosis. We briefly review the literature describing spontaneous regression of both primary and metastatic RCC and emphasize how knowledge of this phenomenon may be useful for abdominal imagers that perform renal biopsy or interpret postbiopsy follow-up studies.

1. Case series

This report is institutional review board exempt at our institution.

1.1. Patient 1

A 78-year-old man with type I diabetes mellitus and stage V chronic kidney disease presented with weight loss. This was evaluated with an abdominal ultrasound and unenhanced magnetic resonance imaging (MRI) that demonstrated a 3.1-cm exophytic left upper pole renal mass with characteristics suspicious for a solid neoplasm (Fig. 1).

This mass was biopsied under ultrasound guidance revealing type I papillary renal cell carcinoma (RCC) with a Fuhrman nuclear grade of 2/4. A thrombotic agent (purified collagen matrix; Helitene, Integra Life Sciences, Irvine, CA) was administered through the introducer into the mass following biopsy procurement to promote hemostasis. Following the biopsy, the patient opted for active surveillance instead of extirpative therapy given his comorbidities, the size of the mass, and the intermediate-grade histology.

A follow-up noncontrast abdominal MRI performed roughly 10 months later without intervening therapy demonstrated spontaneous regression of the left upper pole RCC (Fig. 1). There was a small residual focus of susceptibility artifact in the same location that was favored to represent hemosiderin deposition related to the biopsy. The patient has developed no evidence of local or regional disease spread 18 months following his biopsy, and he remains on active surveillance.

1.2. Patient 2

A 62-year-old man with benign prostatic hyperplasia presented with symptoms of renal colic. Abdominal ultrasound followed by a renal-mass-protocol computed tomography (CT) revealed two enhancing right renal masses in the upper and lower poles (1.8 cm and 1.7 cm, respectively).

Biopsy of the larger 1.8-cm upper pole lesion revealed a type I papillary RCC with a Fuhrman nuclear grade of 2/4. The smaller 1.7-cm lower pole lesion was not sampled and presumed to be of similar histology. Following the biopsy, the patient opted for active surveillance instead of extirpative therapy given the size of the masses and the intermediate-grade histology.

A follow-up renal-mass-protocol CT performed roughly 3 years later showed interval growth of the lower pole lesion to 2.7 cm (originally 1.7 cm) and stable appearance of the previously sampled upper pole lesion; a biopsy of the growing lower pole mass was performed and demonstrated chromophobe RCC. A thrombotic agent (purified collagen matrix) was administered through the introducer into the mass following biopsy procurement to promote hemostasis. Active surveillance was continued.

A follow-up renal-mass-protocol CT obtained roughly 12 months after the second biopsy (4 years after the original CT and biopsy, without

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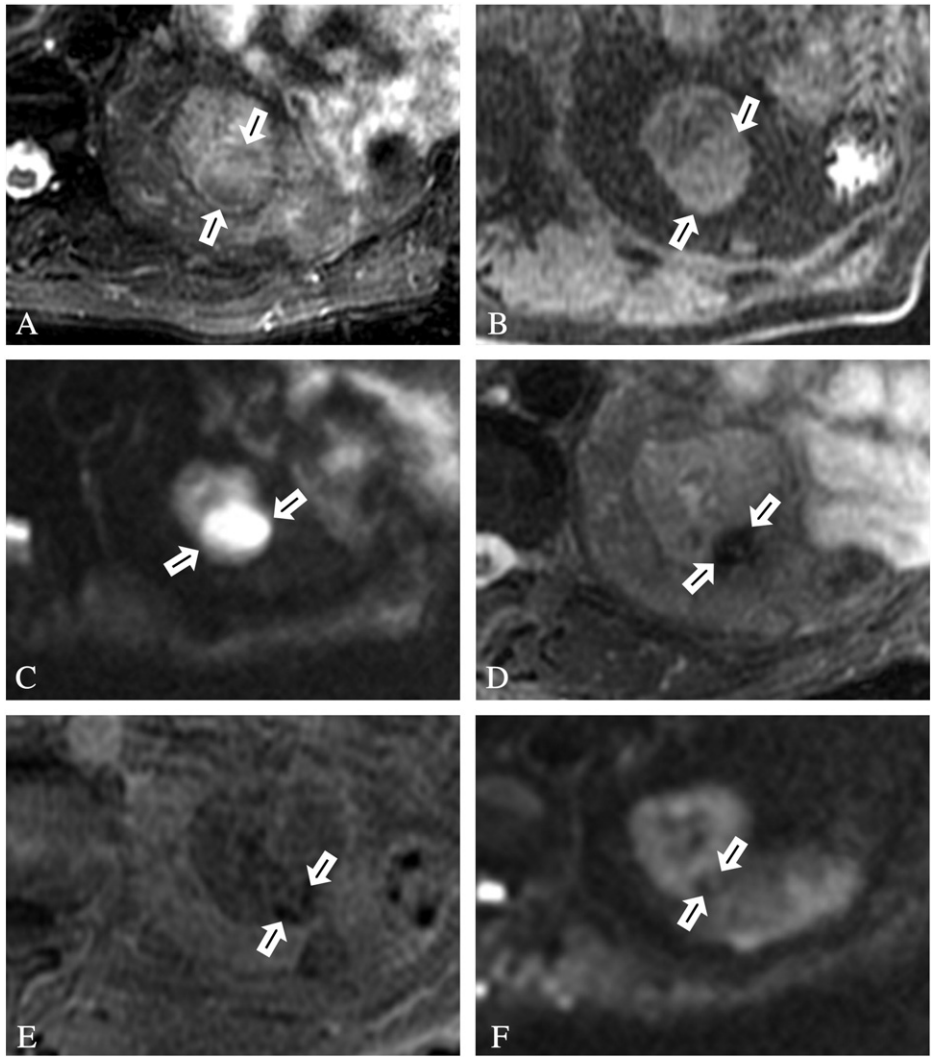

Fig. 1. A 78-year-old man with type I diabetes mellitus and stage V chronic kidney disease undergoes an unenhanced MRI of the abdomen, revealing a 3.1-cm solid-appearing mass arising from the upper pole of the left kidney. (A) Axial T2-weighted fast spin echo with fat saturation, (B) axial T1-weighted gradient echo with fat saturation, (C) axial diffusion-weighted imaging (*b* value=800). Percutaneous biopsy was performed under ultrasound guidance (not shown). Prothrombotic collagen matrix was introduced into the mass postprocedure to promote hemostasis. Follow-up imaging performed approximately 10 months later shows regression of the mass and increase in the apparent diffusion coefficient. (D) Axial T2-weighted fast spin echo with fat saturation, (E) axial T1-weighted gradient echo with fat saturation, (F) axial diffusion-weighted imaging (*b* value=800).

intervening therapy) demonstrated spontaneous regression of the recently biopsied lower pole chromophobe RCC (Fig. 2).

The patient has developed no evidence of local or regional disease spread 19 months following biopsy of the lower pole lesion and he remains on active surveillance.

1.3. Patient 3

A 58-year-old woman with pulmonary sarcoidosis and recent colonic diverticulitis presented with symptoms of renal colic. Renal stone CT followed by a renal-mass-protocol MRI revealed an enhancing renal mass in the interpolar region of the left kidney measuring 1.4 cm (Fig. 3).

The mass was biopsied under CT guidance revealing papillary RCC with a Fuhrman nuclear grade of 2/4. A thrombotic agent (purified collagen matrix) was administered through the introducer into the mass following biopsy procurement to promote hemostasis. The patient opted for active surveillance due to her recent illness (diverticulitis), the size of the masses, and the intermediate-grade histology.

A follow-up renal-mass-protocol MRI performed roughly 6 months later showed interval spontaneous decrease in size of the recently biopsied interpolar papillary RCC to 0.6 cm. The patient has developed no evidence of local or regional disease spread 6 months following her biopsy and she remains on active surveillance.

2. Discussion

RCC is responsible for 2–3% of all cancers; worldwide, there are approximately 209,000 cases and 102,000 deaths annually due to RCC [1]. Risk factors include smoking, obesity, hypertension, and end-stage renal disease. It is associated with several genetic syndromes including von Hippel-Lindau, tuberous sclerosis, Birt-Hogg-Dubé, and hereditary papillary renal carcinoma [1]. RCCs are classically divided into histologic descriptions such as clear cell, papillary, and chromophobe, although more modern descriptions have extended to genetic descriptors such as Xp11 translocations [2]. Surgical treatments include local ablative therapies, partial nephrectomy, and radical nephrectomy. In addition to traditional chemotherapies, more specific medical treatments include