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CASE REPORT

Renal cell carcinoma with atypical omental metastasis: A case report and literature review

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Abstract

Background: Renal cell carcinoma (RCC) is the most common type of kidney cancer in adults, with common metastatic sites including the lungs, bones, liver, and brain. Omental metastasis is exceedingly rare and usually occurs post-operatively. Fumarate hydratase-deficient (FH-deficient) RCC, a recently classified and highly aggressive subtype of RCC, is known to show early metastatic potential but remains poorly understood. We present a rare case of synchronous FH-deficient RCC with isolated omental metastasis identified at diagnosis.

Case Presentation: We report the case of a 48-year-old man who first presented with abdominal pain, early satiety, and a 25-pound weight loss. Imaging revealed an 11.8 cm right renal mass with a separate 15.4 cm left upper quadrant mesenteric mass with no definitive evidence of lung, bone, or liver involvement. Biopsies of both renal and mesenteric masses confirmed non-clear-cell FH-deficient RCC with papillary architecture and diffuse overexpression of 2-succinyl cysteine.

Conclusion: Omental metastasis of RCC is rare, especially in the absence of prior surgery. This case is distinguished by its synchronous presentation of the primary RCC and omental metastasis with a rare histologic subtype. Unlike most documented reports of RCC with omental spread, which typically involve clear cell histology and often present years after nephrectomy, this case involves a rare, aggressive subtype with atypical metastatic behavior. This case underscores the importance of considering atypical metastatic patterns in non-clear-cell subtypes and the need for further research to inform evidence-based therapy.

KEYWORDS

atypical spread, case report, fumarate hydratase-deficient RCC, omental metastasis, renal cell carcinoma

1 | INTRODUCTION

Renal cell carcinoma (RCC) is a kidney cancer originating in the epithelial lining of renal tubules. While the classic triad is hematuria, flank pain, and/or a palpable abdominal mass, RCC is increasingly found asymptomatically due to increased cross-sectional abdominal imaging in the United States[1].

RCC accounts for over 90% of all kidney cancers with a mortality rate of 30%–40%, making it not only the most common kidney cancer but also the deadliest urogenital malignancy[1,2]. The strongest risk factors are age and sex; approximately 50% more likely to appear in men than women and in patients aged 60–70 years. Other risk factors include obesity, smoking, and hypertension[2]. RCC is most prevalent in Europe and North America

and is generally more common in developed countries, a pattern that may reflect increased routine abdominal imaging[1–3].

At diagnosis, only 20%–30% of RCC patients present with metastases[1]. After nephrectomy, 25%–50% of patients develop metastasis, usually appearing in the lungs (3%–16%), liver (1%–7%), bones (2%–8%), or brain (2%) [4]. RCC may also metastasize the adrenal glands, contralateral kidney, small intestine, or pancreas. Omental tumors, which overwhelmingly present as metastatic, very rarely originate from RCC and are more commonly associated with ovarian, endometrial, gastric, and pancreatic cancers[5–8].

2 | CASE PRESENTATION

A previously asymptomatic 48-year-old man with an insignificant past medical history presented to primary care with persistent, diffuse abdominal pain initially experienced during weightlifting that developed over 6–7 months. The patient reported early satiety without regurgitation or nausea/vomiting. He experienced an unintended 25-pound weight loss over the course of 3–4 months. He denied gross hematuria and any irritative voiding symptoms as well as dysphagia, cough, or dyspnea. He reported a smoking history of over 20 pack-years and was currently using smokeless tobacco, including snuff and chew. Physical exam was significant for palpable right-sided abdominal mass without tenderness or guarding.

Initial blood work demonstrated microcytic anemia with mild thrombocytosis. Notably, creatinine was initially normal at 1.1. An ultrasound performed 6 months after the initial presentation demonstrated a large right renal mass and a large lobulated left abdomen mass (Figure 1). Subsequent computerized tomography (CT) imaging revealed a 10.7 cm × 10.2 cm × 11.8 cm heterogeneously enhancing right upper mass of the kidney with marked collateral vessels seen surrounding the lesion (Figure 2). A second 15.4 cm × 10.7 cm × 15.2 cm heterogeneously enhancing mass was noted in the left upper quadrant, abutting the inferior pole of the left kidney and the adjacent descending and transverse colon. An enlarged 1.5 cm left periaortic retroperitoneal lymph node was noted. Abdominal magnetic resonance imaging (MRI) further characterized the right upper pole mass as appearing inseparable from the posterior right abdominal wall and abutting the liver without local extension. The lobulated mesenteric mass, suspicious for metastasis, in the left upper quadrant extended into the left paracolic gutter and pararenal space with significant mass effect and anterior abdominal wall deformity (Figure 3). There was no hydronephrosis, obstructing renal or ureteral calculi, nor bladder anomalies. MRI of the brain and bone scan were negative for metastatic disease.

CT-guided biopsy of the right renal mass and left abdominal mass was then performed. Pathology demonstrated a non-clear cell, fumarate-hydratase-deficient RCC with diffuse overexpression of 2-succinyl cysteine, focal nuclear expression of transcription factor E3, and papillary architecture. There was no demonstrated sarcomatoid or rhabdoid morphology. Patient was ultimately lost to follow-up following initial diagnostic imaging.

3 | DISCUSSION

RCC has a well-characterized metastatic pattern, with common sites including the lung (71%), lymph nodes (49%), bone (36%), liver (21%), adrenal gland (9%), brain (9%), pancreas (5%), and pleura (4%)[9]. Metastases not localized to thoracic, skeletal, hepatic, adrenal, and encephalic sites are considered atypical; peritoneal, mesenteric, and omental spread is especially rare, representing only about 1% of metastatic RCC cases upon autopsy[10]. Peritoneal and retroperitoneal diffusion, when it occurs, is associated with poor prognosis[10].

The most common subtypes of RCC include clear cell (ccRCC) (accounting for 75% of diagnoses), papillary (pRCC) (10%), and chromophobe (chrRCC) (5%)[11]. Fumarate hydratase-deficient RCC (FH-deficient RCC) is a rare, recently classified subtype of RCC, encompassing tumors with either germline or somatic mutations in the fumarate hydratase gene. Originally classified as papillary RCC type II and defined in the context of hereditary leiomyomatosis and renal cell carcinoma (HLRCC) syndrome, FH-deficient RCC has been recognized by the WHO as a distinct histologic entity since 2016 due to its unique molecular features and highly aggressive behavior[12]. It tends to present at an earlier age, is difficult to diagnose, and is characterized by a propensity, even with small tumor size, for early distant metastasis, including to the lymph nodes and bone[13].

At the molecular level, FH-deficient RCC is characterized by biallelic inactivation of the fumarate hydratase (FH) gene, which encodes a mitochondrial enzyme responsible for catalyzing the conversion of fumarate to malate in the tricarboxylic acid (TCA) cycle. This disruption impairs normal cellular respiration and leads to intracellular accumulation of fumarate, an oncometabolite that alters cell signaling and gene regulation. Accumulated fumarate inhibits α-ketoglutarate–dependent dioxygenases, including prolyl hydroxylase domain (PHD) enzymes, resulting in stabilization of hypoxia-inducible factor 1-alpha (HIF-1α) and activation of a pseudo-hypoxic transcriptional program that promotes angiogenesis and tumor progression[14,15]. In addition, fumarate inhibits histone and DNA demethylases, inducing a CpG island methylator phenotype (CIMP) characterized by widespread promoter hypermethylation in tumor suppressor genes and

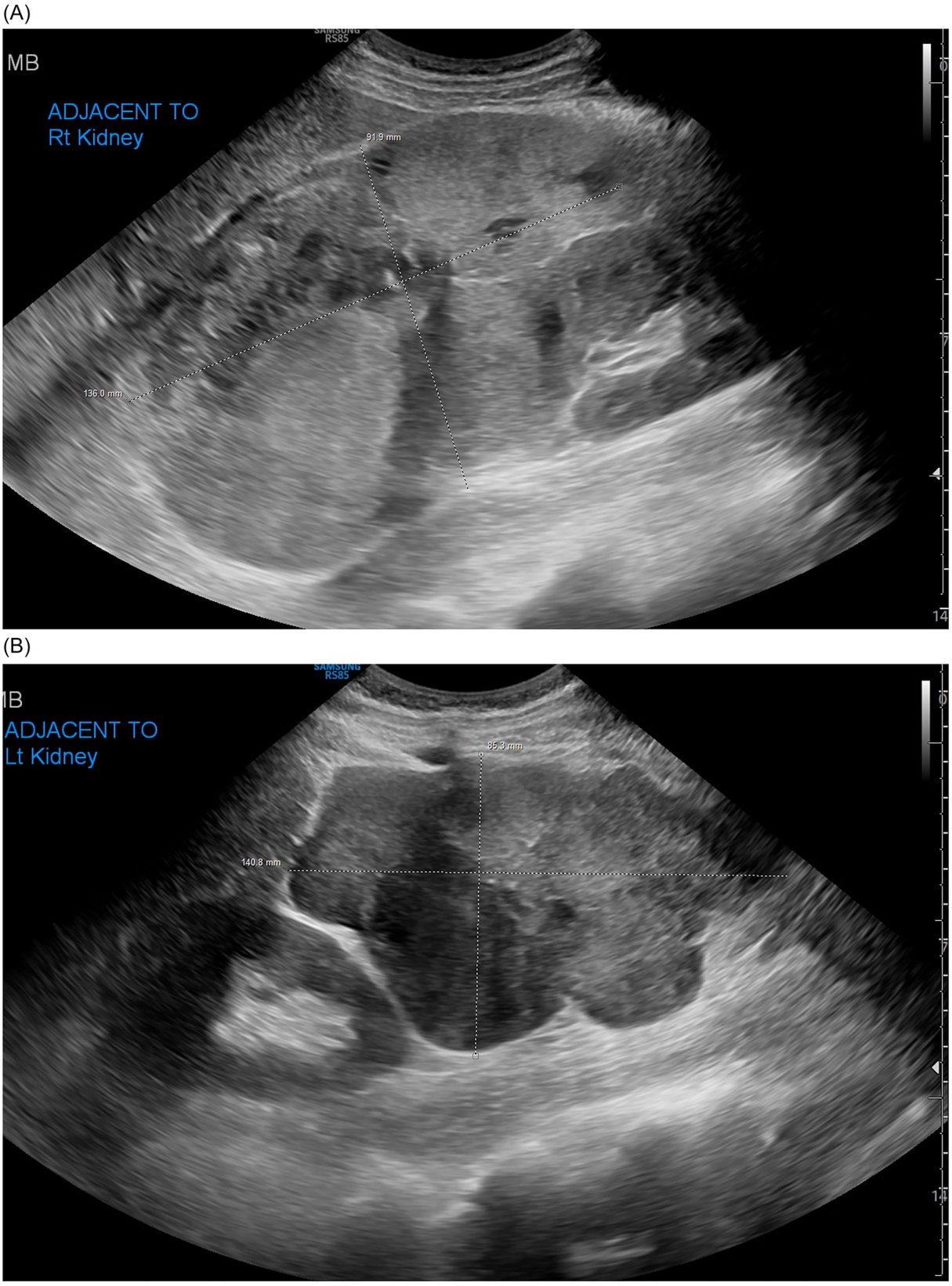

FIGURE 1 Ultrasound of the right abdomen showed a large mass in the upper pole right kidney (A) and a lobulated mass in the left abdomen (B) indicating bilateral abdominal pathology suggestive of multifocal disease.

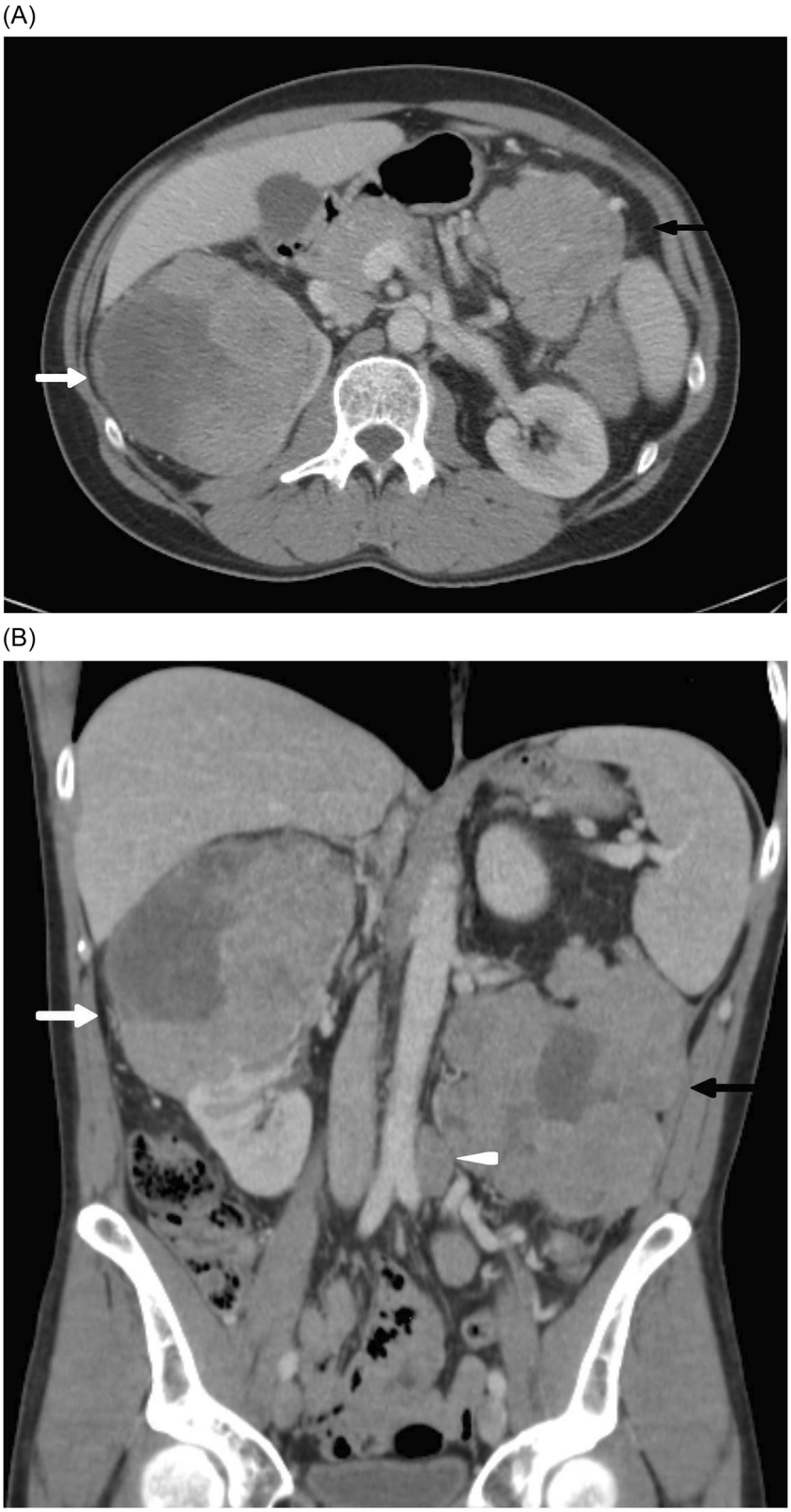

FIGURE 2 Post-contrast axial (A) and coronal (B) computed tomography images of the abdomen demonstrate a heterogenously enhancing mass (white arrow) in the upper pole right kidney abutting the inferior right hepatic lobe and a lobulated mass (black arrow) in the left upper abdominal mesentery. An enlarged left periaortic lymph node is indicated by the white arrow head.

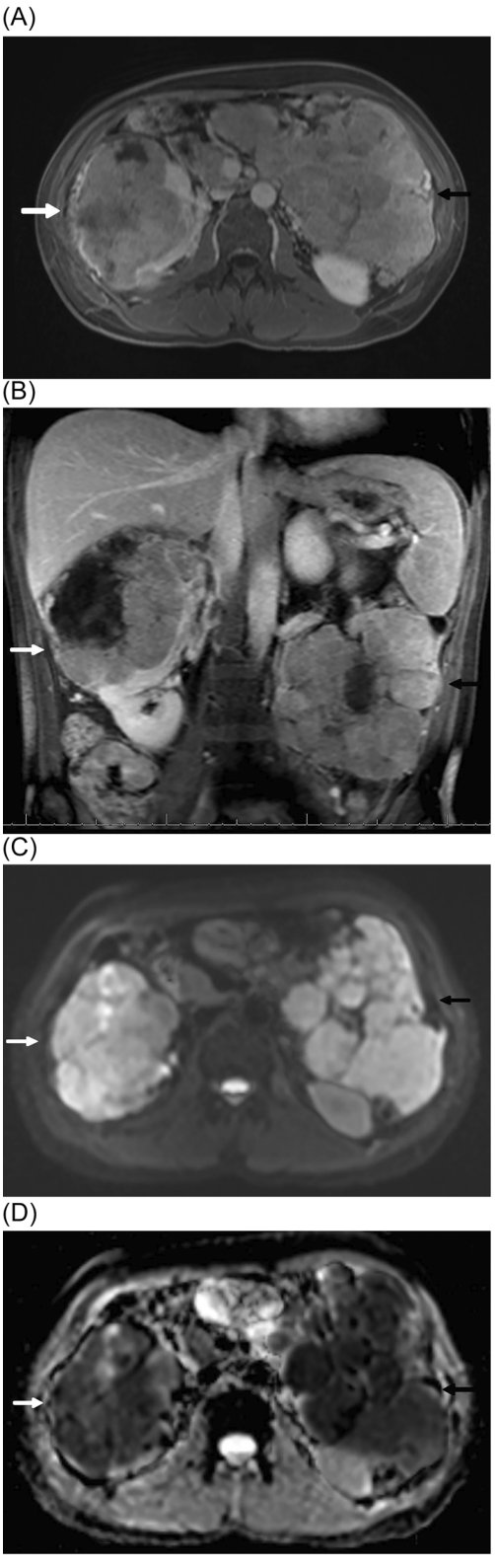

FIGURE 3 Post-contrast axial (A) and coronal (B) T1-weighted images demonstrate a complex enhancing mass in the upper pole of the right kidney (white arrow) and a lobulated mass in the left mesentery (black arrow). Diffusion-weighted imaging (DWI) (C) and the apparent diffusion coefficient (ADC) map (D) images demonstrate restricted diffusion in both the right renal mass (white arrow) and the mesenteric mass (black arrow).

transcriptional regulators, which contributes to increased invasiveness and metastatic potential[14]. Despite a relatively low tumor mutation burden, FH-deficient RCC demonstrates high immunogenicity, with marked tumor-infiltrating lymphocytes and upregulation of immune checkpoint molecules such as PD-L1, TIGIT, and BTLA—features that may support immune evasion and suggest vulnerability to immunotherapy[16,17]. In addition, emerging evidence implicates collaborative genomic alterations, such as concurrent NF2 mutations, in influencing metastatic behavior and site-specific tropism, particularly toward bone[16]. Together, these findings highlight the complex and multifactorial pathogenesis of FH-deficient RCC, driven by metabolic, epigenetic, and immune dysregulation.

Case reports in the literature demonstrate heterogeneity in timing, histology, and anatomic distribution of RCC omental metastases, summarized in Table 1. Acar et al. describe the first reported case of a metachronous, isolated omental metastasis arising 13 years after a partial nephrectomy for T1 clear cell RCC[18]. The patient, a 62-year-old man with a history of pulmonary sarcoidosis, presented with constitutional symptoms. Imaging initially revealed a supravesical fluorodeoxyglucose (FDG)-avid lesion, as well as FDG-avid lesions with widespread osseous, renal, pleural, and lymphatic distribution. Biopsy of the supravesical specimen confirmed metastatic ccRCC, while biopsy of the most hypermetabolic extravesical lesion revealed sarcomatoid reaction. The patient underwent near-total omentectomy and was subsequently treated with sunitinib. Notably, the omental lesion was clearly demarcated without peritoneal or intestinal involvement, and the patient's symptoms as well as his non-omental lesions were suspected to be due to sarcomatoid paraneoplastic syndrome. Similarly, Chung et al. report a case involving a 44-year-old man who presented with hematuria and flank pain and was found to have an 8.8 cm left renal mass[19]. He underwent laparoscopic radical nephrectomy and adrenalectomy for pT3a clear cell RCC (Fuhrman grade III), which demonstrated lymphovascular invasion and tumor necrosis. At 6 months postoperatively, surveillance imaging revealed a 12-mm enhancing omental nodule in the left lower peritoneum later confirmed as metastatic ccRCC. Laparoscopic removal of the lesion was performed one month later.

Other reports describe more diffuse metastatic patterns. Bruckschen et al. documented a 79-year-old man with a history of nephrectomy 17 years prior, who presented with biopsy-proven clear cell metastases to the liver, omentum, thyroid, and mediastinum[20]. Kuzgunbay et al. presented a case of a 72-year-old woman whose initial Bosniak II renal lesion progressed within six months into a semi-solid RCC with an extensive, inoperable omental metastasis forming an “omental cake”[21]. Win and Aparici presented a 69-year-old Caucasian man who had undergone laparoscopic total

TABLE 1 Summary of reported cases of renal cell carcinoma with omental metastasis.

	Year	Site of metastasis	Timing	Histology	Treatment
Acar et al.[18]	2016	Isolated omentum	Metastasis occurred 13 years post pNx	ccRCC with a supravesical FDB-avid lesion	Near-total omentectomy with adjuvant sunitinib
Chung et al.[19]	2023	12-mm omental nodule in left lower peritoneum	Metastasis occurred six months post laparoscopic rNx and adrenalectomy	pT3a ccRCC (Fuhrman grade III)	Laparoscopic removal of the omental lesion one month after confirmed metastasis
Bruckschen et al.[20]	2021	Omentum, liver, thyroid, and mediastinum	Metastases occurred 17 years post-nephrectomy	ccRCC	Chemotherapy with the tyrosine kinase inhibitor pazopanib. Abdominal progression after one year, so therapy was changed to nivolumab. Six months later, more abdominal progression, so cabozantinib was started.
Kuzgunbay et al.[21]	2011	Extensive, inoperable omental cake	Six months after initial Bosniak II lesion	unknown	Surgical exploration led the omental mass to be deemed inoperable. Patient died before immunotherapy.
Win and Aparici[22]	2014	Interaortocaval mass and mesenteric stranding	6 months after laparoscopic rNx	T3aN0M0 chromophobe RCC	Lymph-node dissection and removal of omental implant
Goncalves et al.[23]	2014	Widespread peritoneal implants in omentum, liver, spleen, small and large bowel, mesocolon, abdominal wall	Synchronous metastatic presentation at the time of initial RCC diagnosis	ccRCC	unknown

Abbreviations: ccRCC, clear cell renal cell carcinoma; pNx, partial nephrectomy; rNx, radical nephrectomy.

right nephrectomy for T3aN0M0 chromophobe RCC[22]. Six months later, a surveillance CT revealed a new inter-aortocaval mass and mesenteric stranding, prompting lymph-node dissection and removal of a suspicious omental implant, which tested positive for chromophobe RCC—an uncommon and typically less aggressive histologic subtype. Follow-up FDG-PET/CT in 2014 showed new FDG-avid omental nodules and osseous metastases. Goncalves et al. describe a 58-year-old man who presented at initial workup with acute abdominal symptoms and CT evidence of a large left renal mass with widespread peritoneal implants involving the omentum, liver, spleen, small bowel, colon, mesocolon, and abdominal wall[23]. Biopsy confirmed metastatic ccRCC.

Compared to these cases, our current report exhibits several novel and distinguishing features. First, it represents a synchronous presentation of RCC with omental metastasis, whereas most previously reported cases were metachronous, often with long intervals of more than a decade between nephrectomy and subsequent delayed metastasis[18,20]. Second, the current case involves a rare histological subtype, fumarate hydratase–deficient RCC, for which few, if any, comparable cases of omental or peritoneal metastasis have been described. Third, the patient exhibited a focal omental metastasis in the absence of brain, lung, bone, or widespread visceral involvement. This apparent isolation of the omental metastasis further distinguishes the pattern from some of those described in prior reports. The existing literature on FH-deficient RCC offers no clear precedent for a synchronous presentation of focal omental metastasis in the absence of brain, lung, bone, or widespread visceral involvement.

While peritoneal and omental metastases from RCC are rare overall, they are most often reported in the context of prior surgical intervention, particularly laparoscopic nephrectomy. Port-site and peritoneal seeding following minimally invasive urologic oncology procedures are very uncommon, with incidence rates comparable to those of open surgical wound metastases—making them rare iatrogenic complications[24]. A review by Song et al. identified 16 reported cases of port-site metastasis after surgery for RCC, most involving high-grade tumors and concurrent distant metastases. These events were not typically attributable to technical errors during surgery but instead reflected aggressive underlying tumor biology. Importantly, port-site metastasis was associated with poor prognosis, with an overall 1-year survival rate of just 31.8%, making them more suggestive of progressive disease rather than simply localized surgical failure[25]. Given that port-site metastasis is already an exceedingly uncommon complication of RCC surgery, the occurrence here of spontaneous peritoneal dissemination without prior surgical manipulation renders this case even more exceptional and underscores the aggressive, non-iatrogenic behavior of this metastatic disease.

Theories of metastatic spread in RCC include direct seeding/invasion, lymphatic dissemination, and hematogenous routes. Direct seeding, in which tumor cells breach the renal capsule and disseminate into the peritoneal cavity, is unlikely in this case given the contralateral distribution of disease—the primary tumor arose in the right kidney while the omental metastasis was located in the left lower abdomen. Lymphatic spread is a possible, but less likely, mechanism, given the presence of left periaortic adenopathy is in the retroperitoneum, a separate compartment from the peritoneum. The mechanism of spread is uncertain. This atypical metastatic site is not inconsistent with prior reports describing RCC's unpredictable spread via angioinvasion and complex lymphatic drainage[10]. Sites of metastatic involvement vary by histological subtype and are associated with overall survival; peritoneal and retroperitoneal involvement in particular is linked to poor prognosis[10,26]. The metastatic presentation in our case contrasts with the patterns seen in the more common RCC subtypes. In ccRCC, for example, lung, adrenal, brain, and pancreatic metastases are more common, while papillary RCC more frequently involves lymph nodes, and liver metastases have high incidence in chromophobe RCC[26].

Further research is needed to characterize the metastatic patterns of rarer subtypes like FH-deficient RCC. In particular, multi-omics approaches, including whole-exome sequencing, RNA sequencing, and DNA methylation profiling, may clarify the molecular features driving atypical metastatic tropism (including rarer sites like the peritoneum). Comparative analyses of primary and metastatic lesions could reveal key regulators of peritoneal dissemination, such as CpG methylation changes and immune-modulating chemokines. In addition, immune microenvironment profiling, including the presence of memory and effector T-cell populations, may help identify predictors of immunotherapy response and mechanisms of immune evasion in metastatic FH-deficient RCC[14,16,17].

Unfortunately, our patient was lost to follow-up before definitive treatment could be discussed. For poor-risk metastatic ccRCC, there are currently four FDA-approved first-line drug combinations: ipilimumab–nivolumab, pembrolizumab–axitinib, nivolumab–cabozantinib, and pembrolizumab–lenvatinib, as validated by the CheckMate 214 trial[27]. However, treatment for non-clear cell RCC must be tailored to the specific histologic subtype. In FH–deficient RCC, systemic treatment remains limited by a lack of large-scale prospective trial data, and current regimens are often adapted from ccRCC protocols. Despite this, several therapies have shown promise in retrospective and early-phase studies. The bevacizumab–erlotinib (E–B) combination has demonstrated objective remission rates of 60%–64% and remains guideline-recommended despite limited validation[13]. More

recently, combinations of immune checkpoint inhibitors (ICIs) and antiangiogenic agents, such as sindilizumab–axitinib or pembrolizumab-based regimens, have achieved encouraging disease control rates and extended progression-free survival[13,28]. One recent retrospective study suggests that antiangiogenic agents may be superior to ICI/mTOR combinations for first-line treatment in FH-deficient RCC, with a higher objective response rate and significantly longer time to treatment failure[29]. Given the aggressiveness and rarity of FH-deficient RCC, clinical trial enrollment is strongly encouraged.

Follow-up protocols for FH-deficient RCC reflect its aggressive behavior. Imaging is recommended every three months for the first two years after surgery, then every six months thereafter[13]. Surveillance should include CT imaging of the neck, chest, and abdomen, supplemented by MRI for better characterization of brain, liver, bone, and cystic metastases. PET and SPECT scans may miss osteolytic bone lesions, so MRI is preferred when those are suspected[13].

4 | CONCLUSION

Omental metastasis of RCC is rare and is typically reported only after surgical intervention. This case highlights an exceptionally rare presentation of a synchronous FH-deficient RCC with focal omental metastasis, in the absence of more commonly involved metastatic sites such as the lung, brain, and bone. To our knowledge, this is the first reported case of FH-deficient RCC with peritoneal dissemination at the time of initial diagnosis. Given the aggressive nature and poorly defined metastatic behavior of the FH-deficient histologic subtype, early recognition through comprehensive abdominal imaging is essential. This case underscores the importance of maintaining a high index of suspicion for atypical metastatic patterns as well as conducting further research on the metastatic tendencies of FH-deficient RCC to guide further therapy.

AUTHOR CONTRIBUTIONS

Andrew Jing was the lead author for the discussion section and contributed to the literature review and to manuscript drafting and revision. Zachary Guo wrote the introduction and contributed to the literature review and to manuscript drafting and revision. Katherine Wu performed the chart review and contributed to the literature review and to manuscript drafting and revision. Wei Wang contributed to the literature review and provided pathology consultation. James Jing conceived the original idea for the paper, reviewed and revised the manuscript, and prepared the final version for submission. All authors made substantial contributions to the conception, interpretation, and execution of the study and approved the final manuscript.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

This case report adheres to the highest standards of ethical conduct in research and publication. Patient information has been appropriately de-identified or excluded to protect privacy. This study conforms to the ethical guidelines of the Declaration of Helsinki and relevant institutional standards for the research and publication of anonymized patient data.

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