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LEFT RENAL MASS SUGGESTIVE OF RENAL CELL CARCINOMA WITH RADIOLOGICALLY SUSPECTED CONTRALATERAL ADRENAL METASTASIS: A DIAGNOSTIC CHALLENGE WITHOUT HISTOPATHOLOGIC CONFIRMATION

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ABSTRACT

Background: Renal cell carcinoma (RCC) may involve the adrenal gland, but isolated contralateral adrenal metastasis is uncommon. If metastatic, noncontiguous contralateral adrenal involvement constitutes distant (M1) rather than locally advanced (T4) disease. Imaging cannot

reliably distinguish metastasis from adenoma, adrenocortical carcinoma, or an adjacent renal mass. Delayed first presentation may preclude tissue confirmation

Case presentation: A 59-year-old man from a rural area presented with four months of progressive left abdominal and flank pain and hematuria. He had no past medical history and had never undergone previous examination. Contrast-enhanced CT demonstrated a left renal mass measuring approximately 10.6 x 14 x 14.2 cm, with heterogeneous enhancement, central necrosis, renal sinus invasion, and enlarged para-aortic lymph nodes, suggestive of RCC. A contralateral mass in the right suprarenal region, measuring approximately 7.4 x 7.6 x 10.1 cm, was interpreted as radiologically suspected metastasis versus a primary adrenal tumor. The disease was staged clinically as cT3aN1M1. Dedicated adrenal-protocol imaging, extra-abdominal staging, metabolic evaluation, and tissue sampling were planned but not completed. He developed anemia, electrolyte imbalance, and a left pleural effusion, and died on hospital day 9. Histopathology was not obtained.

Conclusion: This case illustrates an unresolved dual diagnostic problem without histologic confirmation. When a contralateral suprarenal mass is found together with a renal mass suggestive of RCC, the priority is adrenal-protocol imaging, metabolic testing, extra-abdominal staging, and tissue sampling if the patient remains a candidate. If that sequence cannot be completed, the diagnosis must remain radiologically suspected, and clinical staging should retain the “c” prefix.

Keywords: adrenal metastasis; case report; contralateral adrenal gland; diagnostic imaging; renal cell carcinoma

INTRODUCTION

Renal cell carcinoma (RCC) accounts for 2% to 3% of adult malignant neoplasms, is the most lethal of the common urologic cancers, and represents 90% to 95% of malignant renal tumors.^{1,2} Although many contemporary cases are detected incidentally, symptomatic patients may still present with flank pain, hematuria, or constitutional complaints and more often harbor advanced disease. Approximately one-third of patients have synchronous metastases at diagnosis, and 10-year survival among those with metastatic RCC is less than 5%.¹ Distant metastases most often involve the lungs, liver, bone, and brain; the adrenal gland is a less frequent site.² Contiguous extension into the ipsilateral adrenal gland is staged as T4, whereas noncontiguous adrenal involvement ipsilateral or contralateral is classified as distant metastasis (M1) and therefore

changes both prognosis and treatment intent.^{1,3} Concurrent ipsilateral adrenalectomy is no longer performed routinely and is reserved for radiographic or intraoperative suspicion of adrenal disease.^{1,3,4} Accurate characterization of a concurrent adrenal lesion is therefore essential when a renal mass is suggestive of RCC.

Adrenal metastases have been reported in 1.1% to 10% of patients with RCC.^{3,5} In a large surgical series, metastases were identified in 5.5% of adrenal glands removed at nephrectomy, predominantly among patients with pT3 or greater primary tumors.³ Isolated ipsilateral adrenal metastasis occurs in approximately 3% to 5% of patients undergoing nephrectomy, whereas isolated contralateral involvement is uncommon, with an estimated incidence of 0.7%.² Bilateral adrenal metastasis is rarer still, reported in fewer than 0.5% of cases.² Among patients with metastatic RCC, adrenal involvement is observed in approximately 6% to 10%.⁴

Imaging cannot reliably distinguish adrenal metastasis from a benign adenoma, adrenocortical carcinoma, or an adjacent renal mass.^{1,2,5} Adrenal incidentalomas are found in approximately 3% to 7% of adults, and most are benign non-functioning adenomas.^{5,6} In patients with RCC, roughly half of adrenal masses may represent metastasis; however, the probability that an adrenal abnormality on computed tomography (CT) or magnetic resonance imaging is metastatic has been reported across a wide range, from 11% to 93%.⁵ Lipid-rich adenomas typically measure 10 Hounsfield units or less on unenhanced CT, but hypervascular RCC metastases may exhibit washout similar to lipid-poor adenoma on delayed contrast-enhanced imaging.^{1,5,7} Large size, heterogeneity, and necrosis increase concern for malignancy: the likelihood of cancer has been reported as 10%, 19%, and 47% for adrenal tumors of 4 cm or greater, 6 cm or greater, and 8 cm or greater, respectively.⁷ Lesions identified during staging of a known extra-adrenal cancer are not classified as incidentalomas and require oncologic interpretation rather than a purely incidentaloma pathway.⁶ Even when metastasis is the leading radiologic concern, a metabolic evaluation remains indicated, because imaging cannot exclude a functioning lesion and percutaneous sampling of an unrecognized pheochromocytoma is hazardous.^{1,6} Histopathology remains the definitive means of distinction.²

These uncertainties have direct therapeutic implications. In highly selected patients, isolated adrenal metastasis may be considered for metastasectomy as part of a cytoreductive

strategy; however, a survival benefit has not been established in prospective trials.¹ Solitary adrenal involvement is nevertheless associated with a more favorable outlook than disseminated disease.^{3,4} Multidisciplinary review is required when imaging is not consistent with a benign lesion or when resection is contemplated.^{6,7} When biopsy or resection cannot be performed, the diagnosis remains presumptive. Delayed first presentation from a rural area, without previous examination, may allow tumors to enlarge until systemic complications preclude tissue confirmation.

We report a 59-year-old man from a rural area who presented with four months of progressive left abdominal and flank pain and hematuria. Contrast-enhanced CT showed a large left renal mass suggestive of RCC, with renal sinus invasion and enlarged para-aortic lymph nodes, and a large contralateral mass in the right suprarenal region interpreted as radiologically suspected metastasis versus a primary adrenal tumor. The disease was staged clinically as cT3aN1M1 according to the AJCC Cancer Staging Manual, 8th edition.⁸ Dedicated adrenal-protocol CT, extra-abdominal staging, metabolic evaluation, and tissue sampling were planned but not completed. No histopathology was obtained, and the patient died on hospital day 9. This case illustrates the diagnostic challenge of suspected contralateral adrenal metastasis when histologic confirmation is unavailable, and the impact of delayed presentation on definitive management.

CASE PRESENTATION

A 59-year-old man from a rural area was admitted with progressive left abdominal and flank pain of four months' duration, which had intensified over the past week. The pain radiated around the left flank, was graded 7 to 8 on a numeric scale, and was accompanied by hematuria, right flank discomfort, weakness, and nausea. Bowel habit was unchanged. He had no past medical history and had never undergone previous examination.

On examination he appeared moderately ill and was fully conscious. Blood pressure was 130/80 mmHg, heart rate 128 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 96% on room air. Both flanks were examined: there was no bulging, and no mass was palpable on either the left or the right side. Admission laboratory studies demonstrated anemia, hyponatremia, hyperkalemia, and elevated urea and creatinine (Table 1). Random blood glucose and coagulation times were within the reference range. Random blood glucose and coagulation times were within the reference range. Initial management comprised intravenous 0.9% sodium

chloride, transfusion of one unit of packed red cells, and intravenous cefoperazone and omeprazole. Hyperkalemia was treated with slow intravenous 10% calcium gluconate.

Table 1. Serial laboratory findings during hospitalization

Hospital day	Hemoglobin (g/dL)	Sodium (mmol/L)	Potassium (mmol/L)	Urea (mg/dL)	Creatinine (mg/dL)
Day 1	9.3	126	5.2	70	3.20
Day 2	9.6	128	5.2		
Day 3	9.5	129	5.1		
Day 4	9.5	129	5.4	79	2.61
Day 8	-	-	-	155	3.93

Contrast-enhanced abdominal CT with axial, coronal, and sagittal reconstructions showed an inhomogeneous hypodense mass in the left kidney (73 HU), with well-defined but irregular (ball-type) margins and central necrosis, measuring approximately 10.6 x 14 x 14.2 cm. The lesion enhanced to 95 HU on post-contrast images, occupied the upper to mid poles of the left kidney, invaded the renal sinus, and displaced the pelvicalyceal system. The right kidney was not enlarged and had a normal contour (fig.1). A hypodense mass (30 HU) with well-defined, irregular margins, measuring approximately 7.4 x 7.6 x 10.1 cm, was identified in the right suprarenal region. Enlarged para-aortic lymph nodes were present (fig.2). A left pleural effusion was visible on CT. There was no free intraperitoneal fluid. Visualized bone showed no lytic or blastic lesions. Renal-vein or inferior vena cava thrombus was not described. Chest radiography showed a pleural effusion in the left lower lung field (fig.3).

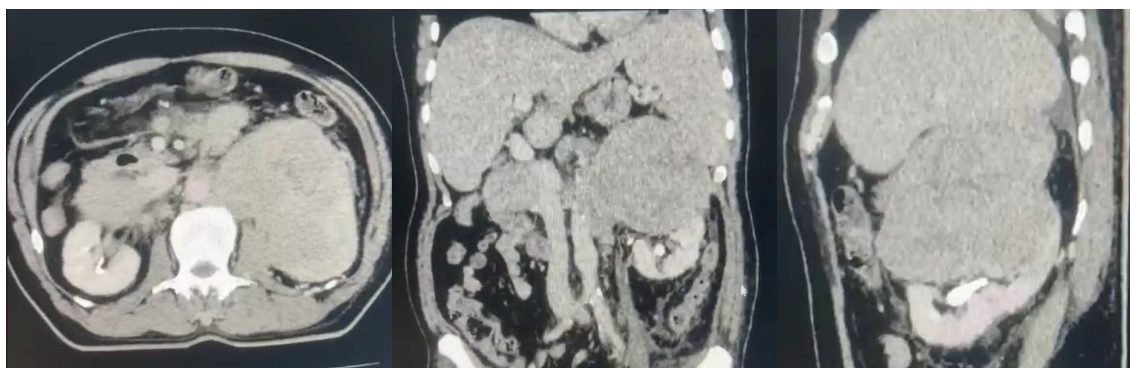


Figure 1. Abdominal CT scan with axial, coronal, and sagittal reconstructions showing the left renal mass suggestive of RCC; enlarged para-aortic lymph nodes



Figure 2. Right suprarenal mass and enlarged para-aortic lymph nodes

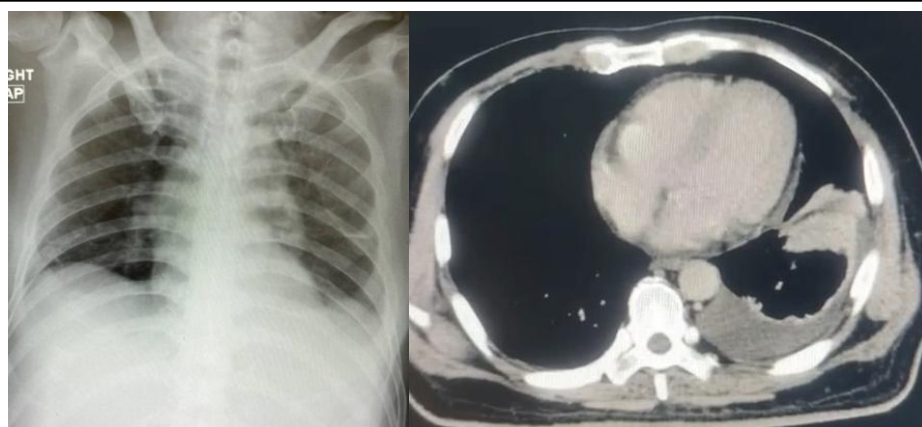


Figure 3. Left pleural effusion in the left lower lung field

The diagnosis was (1) a left renal mass suggestive of RCC, staged clinically as cT3aN1M1 according to the AJCC Cancer Staging Manual, 8th edition, and (2) a right suprarenal mass, radiologically suspected metastasis versus a functional or non-functional primary adrenal tumor. Further investigations were planned to support the diagnosis: dedicated adrenal-protocol CT (unenhanced attenuation and washout), chest CT for complete staging, bone scintigraphy, metabolic evaluation of cortisol, catecholamine, and aldosterone excess, and tissue sampling for histopathology. These studies were not completed.

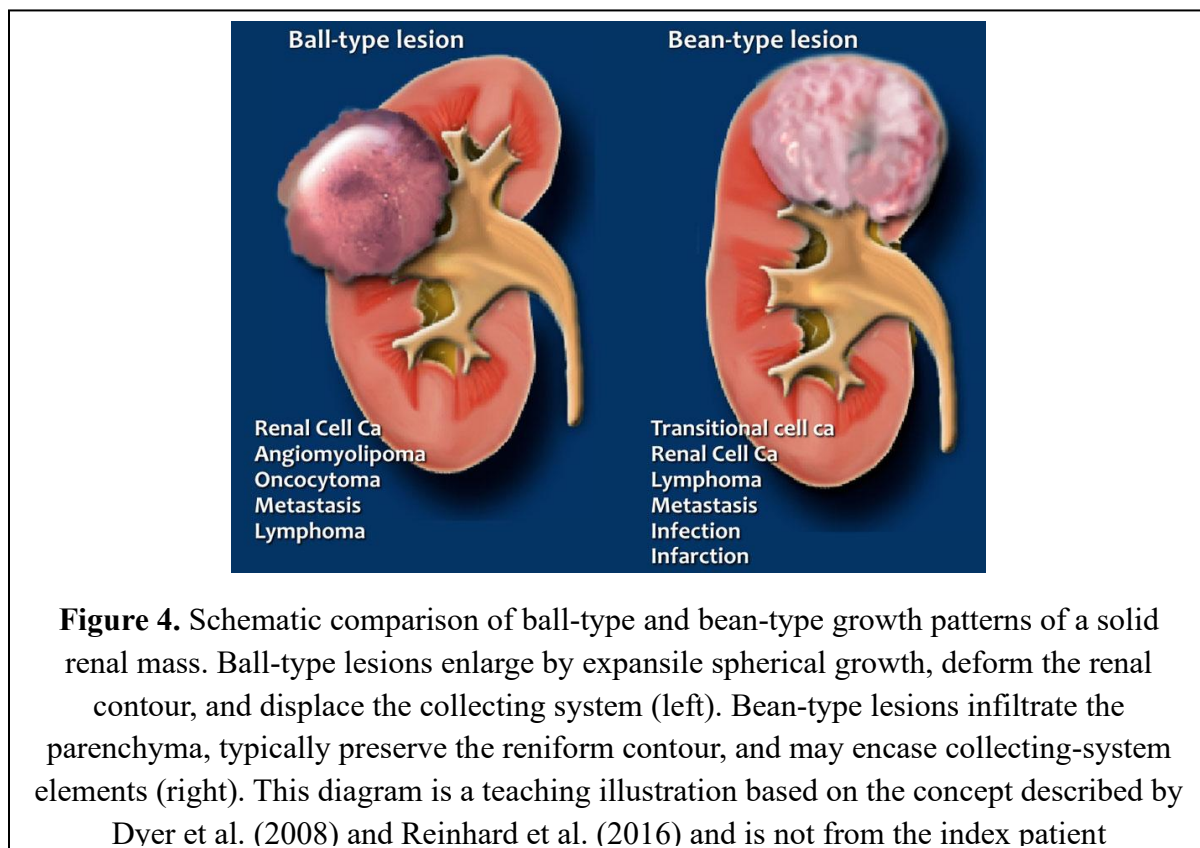
During hospitalization, the left flank pain improved, but dyspnea subsequently developed. Serial laboratory measurements showed persistent hyponatremia and serum potassium at or above the upper reference limit; hemoglobin remained in the anemic range after transfusion, and azotemia worsened on hospital day 8 (Table 1). Oxygen was administered via nasal cannula. Intravenous furosemide was added for the left pleural effusion. Hyponatremia was corrected with 3% sodium chloride, and recurrent hyperkalemia required repeated intravenous calcium gluconate. Anemia was managed with oral iron supplementation after the initial transfusion. Intravenous levofloxacin was initiated, and thoracentesis was planned but deferred because the effusion was small. As his clinical condition worsened, further diagnostic and surgical procedures, including percutaneous biopsy, adrenalectomy, and nephrectomy, could not be performed before he died on hospital day 9. Histopathologic confirmation was not obtained.

DISCUSSION

This case illustrates a dual diagnostic problem that could not be resolved before death: a large symptomatic left renal mass with imaging features suggestive of RCC, and a concurrent contralateral suprarenal mass that was interpreted as radiologically suspected metastasis versus a primary adrenal tumor. Isolated contralateral adrenal metastasis is uncommon, with an estimated incidence of 0.7% among patients undergoing nephrectomy, and imaging cannot reliably distinguish metastasis from adenoma, adrenocortical carcinoma (ACC), or an adjacent renal mass.^{2,5} The patient presented from a rural area after four months of progressive pain and hematuria, without prior examination, and died on hospital day 9 before dedicated adrenal-protocol imaging, extra-abdominal staging, metabolic testing, or tissue sampling could be completed. The clinical stage of cT3aN1M1 therefore remains presumptive.⁸ The case is instructive because large

size, heterogeneity, and a known extra-adrenal mass increase concern for malignancy, yet none of these features establishes histology, and several alternative diagnoses remain open.^{6,7}

A structured imaging approach to a renal mass begins by classifying the lesion as cystic, fat-containing, or solid.^{9,10} Most cystic masses can be characterized with established CT criteria and are benign.⁹ When a mass is not cystic, the next step is to search for macroscopic fat, typically attenuation less than -20 HU, which identifies a fat-containing lesion.¹⁰ If neither a cystic architecture nor macroscopic fat is demonstrated, the lesion is treated as a solid mass.^{9,10} Solid masses are then further divided by growth pattern into ball-type and bean-type lesions (Fig. 4).^{9,10} Ball-type masses enlarge by expansile spherical growth, deform the renal contour, and may displace the collecting system; bean-type lesions use the renal parenchyma as a scaffold for infiltrative growth, typically preserve the reniform contour, and may encase or obliterate collecting-system elements rather than displace them.^{9,10} Recognition of this pattern provides a starting point for characterization before a specific histologic diagnosis is assigned.⁹



The left renal lesion was solid, without described macroscopic fat, and occurred in a patient without fever or a known extra-renal primary, so a cyst, classic angiomyolipoma, pyelonephritis, and metastasis were not favored.¹⁰ The mass occupied the upper to mid poles, displaced the pelvicalyceal system, and enlarged by expansile growth that deformed the renal contour, matching a ball-type pattern (Fig 4).^{9,10} Conventional (clear-cell) RCC is the prototypic ball-type mass; the prototypic bean-type lesion is the aggressive infiltrative form of urothelial carcinoma (transitional cell carcinoma [TCC]).⁹ Because the present lesion displaced collecting-system elements rather than encasing them and did not preserve a reniform contour, infiltrative TCC was not the leading consideration, although infiltrative RCC, collecting-duct carcinoma, and lymphoma may assume a bean-type pattern, and lymphoma may be either ball-type or bean-type.^{9,10} Size is the most important imaging predictor of malignancy: only a minority of resected masses of 6 to 7 cm prove histologically benign, and the present lesion measured approximately 10.6 x 14 x 14.2 cm.¹⁰

Heterogeneous enhancement from 73 to 95 HU with central necrosis further supports a hypervascular solid neoplasm, most often clear-cell RCC, although oncocytoma, lipid-poor angiomyolipoma, and lymphoma may produce overlapping ball-type morphology.^{1,9,10} Invasion of the renal sinus without described renal-vein or inferior vena cava thrombus fulfills the eighth edition criterion for T3a disease and does not require venous tumor thrombus.⁸ Enlarged para-aortic nodes were classified as regional (N1) rather than distant disease, because aortic and para-aortic nodes drain the kidney; nodal enlargement on CT remains a clinical assignment, because reactive hyperplasia is common and some metastases measure less than 1 cm.^{1,8}

Table 2. TNM Classification of Renal Cell Carcinoma According to the AJCC 8th Edition. Adapted from Amin et al. (2017).

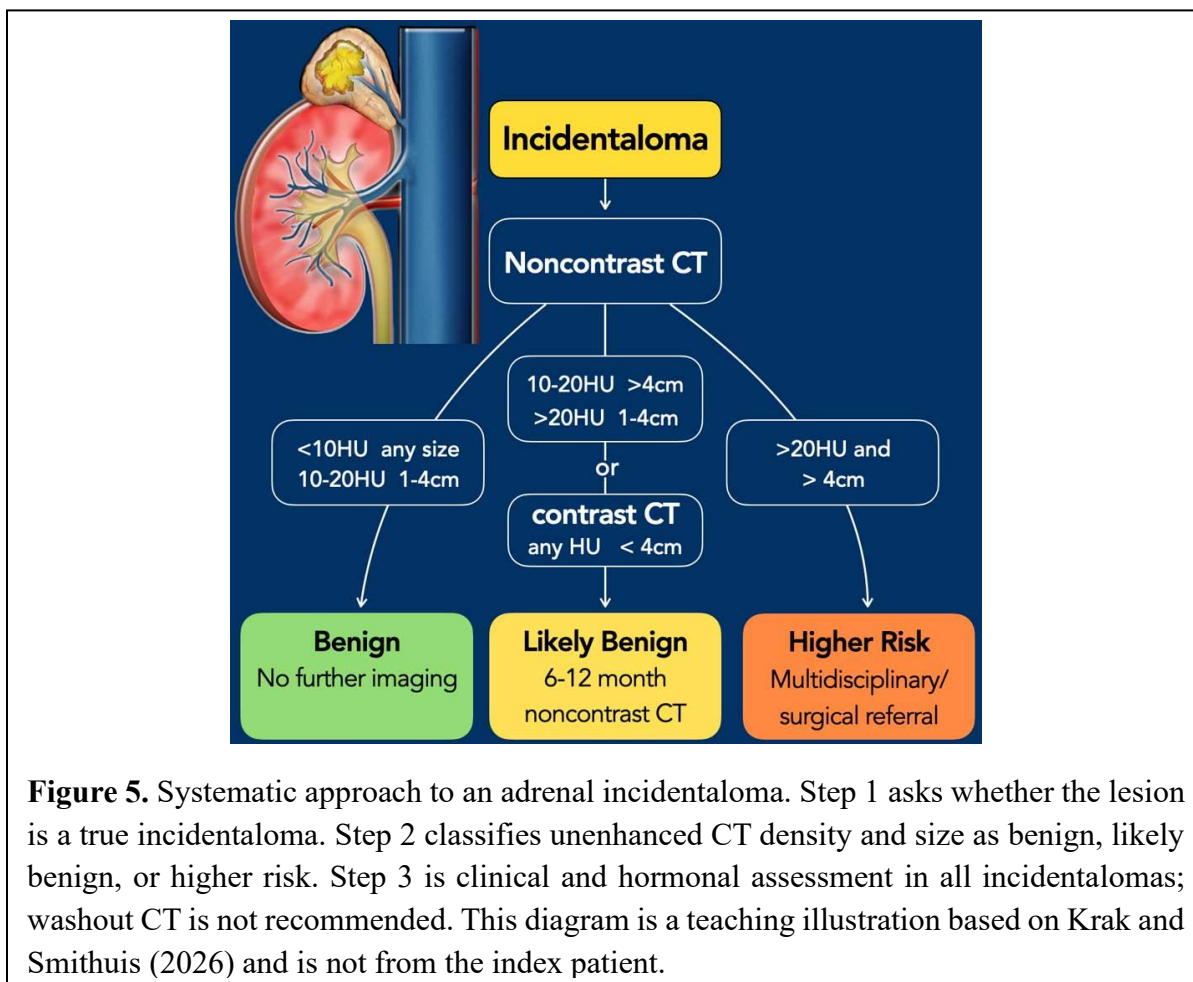
Primary Tumor (T)	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Tumor ≤ 7 cm in greatest dimension, limited to the kidney
T1a	Tumor ≤ 4 cm in greatest dimension, limited to the kidney
T1b	Tumor > 4 cm but ≤ 7 cm in greatest dimension, limited to the kidney

T2	Tumor > 7 cm in greatest dimension, limited to the kidney
T2a	Tumor > 7 cm but ≤ 10 cm in greatest dimension, limited to the kidney
T2b	Tumor > 10 cm, limited to the kidney
T3	Tumor extends into major veins or perinephric tissues, but not into the ipsilateral adrenal gland and not beyond Gerota's fascia
T3a	Tumor extends into the renal vein or its segmental branches, or invades the pelvicalyceal system, or invades perirenal and/or renal sinus fat but not beyond Gerota's fascia
T3b	Tumor extends into the vena cava below the diaphragm
T3c	Tumor extends into the vena cava above the diaphragm or invades the wall of the vena cava
T4	Tumor invades beyond Gerota's fascia (including contiguous extension into the ipsilateral adrenal gland)
Regional Lymph Node (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in regional lymph node(s)
Distant Metastasis (M)	
M0	No distant metastasis
M1	Distant metastasis

Accurate assignment of the right suprarenal mass determines whether the patient has locally advanced or metastatic disease. Contiguous extension into the ipsilateral adrenal gland is staged as T4, whereas noncontiguous adrenal involvement ipsilateral or contralateral is distant metastasis (M1) and places the patient in stage group IV.^{1,3,8} The present lesion lay in the contralateral suprarenal region and was separate from the left renal mass, so if it represents metastatic RCC it fulfills the M1 criterion rather than T4.^{1,8} Isolated contralateral adrenal metastasis is uncommon, with an estimated incidence of 0.7% among patients undergoing nephrectomy.² Adrenal metastases overall have been reported in 1.1% to 10% of patients with RCC and cluster among pT3 or greater primary tumors, a category that the left lesion would occupy if sinus invasion is confirmed.^{3,5} Imaging nevertheless cannot distinguish metastasis from adenoma, adrenocortical carcinoma, or an adjacent renal mass, and an abnormal gland is not histologic proof

of metastasis.^{2,3,5} The M1 assignment in this case therefore remains clinical and radiologically suspected.⁸

A structured approach to an adrenal lesion begins by determining whether it is a true incidentaloma: a mass of 1 cm or greater, detected on imaging not performed for suspected adrenal disease, in a patient without prior or current extra-adrenal malignancy.^{6,11} Characterization then proceeds by unenhanced CT density and size (Fig. 5). Homogeneous lesions measuring less than 10 HU at any size, or 10 to 20 HU and 1 to 4 cm, can be considered benign and need no further adrenal imaging. Lesions of 10 to 20 HU larger than 4 cm, or greater than 20 HU and 1 to 4 cm, are likely benign and are followed at 6 to 12 months. Higher-risk morphology comprises attenuation greater than 20 HU together with size greater than 4 cm, or heterogeneity, and warrants multidisciplinary discussion and surgical referral. Washout CT is no longer recommended for true incidentalomas.¹¹ Clinical and hormonal assessment is indicated for every incidentaloma.^{6,7,11}



The right suprarenal mass fulfills the definition of an adrenal incidentaloma: it measured greater than 1 cm, was clinically unsuspected, and was detected on CT performed for left flank pain and hematuria rather than for adrenal symptoms or for staging of a previously known extra-adrenal cancer.^{6,7,11} Concurrent discovery of a left renal mass suggestive of RCC raises oncologic concern but does not convert that examination into a staging study of a previously established malignancy.^{6,11} The lesion measured approximately 7.4 x 7.6 x 10.1 cm with irregular margins and a recorded attenuation of 30 HU, which would place it in the higher-risk category if that value were a validated unenhanced measurement (Fig. 5).¹¹ The available study nevertheless lacked a documented unenhanced adrenal-protocol acquisition, so 30 HU is not a lipid-content measurement, and washout could not be calculated; delayed washout can also misclassify

hypervascular RCC metastases as lipid-poor adenomas.^{5,11,12} Clinical and hormonal assessment was indicated—fractionated plasma or urinary metanephrines, a 1-mg overnight dexamethasone suppression test or an equivalent test for autonomous cortisol secretion, and, if hypertension or hypokalemia is present, an aldosterone-to-renin ratio—but none of these tests was completed.^{6,7,11}

The higher-risk category overlaps with typical imaging of ACC: unenhanced density greater than 30 HU, size usually greater than 6 cm, irregular margins, and heterogeneous or necrotic architecture.^{11,13} The present lesion exceeded 8 cm with irregular margins, which is in the size range associated with ACC, but the recorded 30 HU cannot be used as supporting unenhanced evidence.¹¹ Size alone correlates with cancer risk (10%, 19%, and 47% for adrenal tumors of 4 cm or greater, 6 cm or greater, and 8 cm or greater, respectively) and does not establish histology.⁷ Given a contralateral renal mass suggestive of RCC, metastasis remains the leading oncologic consideration, yet a synchronous primary adrenal tumor is not excluded.^{2,5} Adrenal ganglioneuroma can exceed 6 to 9 cm, remain metabolically silent, and be indistinguishable from ACC until resection, although it typically affects younger patients than this 59-year-old man.¹⁴ Collision tumors, in which metastatic RCC coexists with adenoma, may retain lipid signal and still harbor metastasis.¹⁵ Malignant considerations on this pathway therefore include ACC, metastasis, lymphoma, malignant pheochromocytoma, and, rarely, collision tumor.¹¹

Because imaging cannot establish histology, tissue sampling remained the only method of distinction.² Percutaneous biopsy may be considered when the result would change management, particularly to distinguish metastasis from a primary adrenal tumor.^{1,6} It does not reliably separate adenoma from ACC and carries a risk of needle-tract seeding in suspected ACC.^{6,13} Biochemical exclusion of pheochromocytoma is required before needle sampling or resection, because percutaneous biopsy or unprepared instrumentation of an unrecognized catecholamine-secreting tumor can precipitate a hypertensive crisis, arrhythmia, or cardiovascular collapse.^{1,6,7} None of these steps was completed.

If isolated contralateral adrenal metastasis had been confirmed in a patient with good performance status, metastasectomy combined with cytoreductive nephrectomy would have been a reasonable multidisciplinary option. Complete resection of isolated or oligometastatic adrenal disease has been associated with more favorable survival than disseminated metastasis.²⁻⁴

Contemporary surgical series report durable overall survival after adrenalectomy in selected patients, although larger primary tumor diameter independently predicts worse outcome.⁴ Ficarra and Crestani (2016) argued for cytoreductive nephrectomy plus metastasectomy in a good-performance-status patient with a large primary tumor and limited metastatic volume, consistent with guideline selection rather than routine surgery for all M1 disease. A survival benefit of adrenal metastasectomy has not been established in prospective trials, and cytoreductive nephrectomy is not uniformly beneficial in the targeted-therapy era, particularly in poor-risk patients.^{1,16}

The present patient was not a surgical candidate. He appeared moderately ill with tachycardia and tachypnea at admission and had established renal impairment (creatinine 3.20 mg/dL), anemia, hyponatremia, and hyperkalemia.¹ He developed a left pleural effusion and progressive azotemia and died on hospital day 9, before nephrectomy or adrenalectomy could be considered. Isolated adrenal metastatic disease was also not established: chest CT and bone scintigraphy were not obtained, so pulmonary or osseous metastases were neither confirmed nor excluded. The left pleural effusion cannot be equated with M1 lung disease.⁸

Several limitations follow from the incomplete diagnostic evaluation. First, both the left renal diagnosis and the M1 assignment rest on CT morphology without histopathology; suggestive imaging is not confirmation.^{2,8} Second, 30 HU was recorded without a documented unenhanced adrenal-protocol acquisition, so adenoma, pheochromocytoma, ACC, ganglioneuroma, and hypervascular metastasis remain formally open.^{7,12,14} Third, delayed first presentation from a rural area, without previous examination, allowed both masses to reach dimensions at which systemic complications precluded tissue diagnosis. The case therefore demonstrates that when a contralateral suprarenal mass is found together with a renal mass suggestive of RCC, the priority is a time-sensitive sequence of adrenal-protocol imaging, metabolic testing, complete extra-abdominal staging, and, if the patient remains a candidate, biopsy or resection. When that sequence cannot be completed, the diagnosis must remain radiologically suspected, and clinical staging should retain the "c" prefix.⁸

CONCLUSION

This case illustrates an unresolved dual diagnostic problem: a large symptomatic left renal mass with imaging features suggestive of RCC, and a concurrent contralateral suprarenal mass

interpreted as radiologically suspected metastasis versus a primary adrenal tumor. Isolated contralateral adrenal metastasis is uncommon, and imaging cannot reliably distinguish metastasis from adenoma, adrenocortical carcinoma, or an adjacent renal mass.^{2,5} Metastasis remains the leading oncologic consideration, yet a synchronous primary adrenal tumor is not excluded. The clinical stage of cT3aN1M1 remains presumptive, because dedicated adrenal-protocol imaging, extra-abdominal staging, metabolic testing, and tissue sampling were not completed before death on hospital day 9.⁸ Large size, heterogeneity, and a known extra-adrenal mass increase concern for malignancy, yet none of these features establishes histology.^{6,7} Delayed first presentation from a rural area, without previous examination, allowed both lesions to reach dimensions at which systemic complications precluded biopsy or resection.

When a contralateral suprarenal mass is found together with a renal mass suggestive of RCC, the priority is a time-sensitive sequence of adrenal-protocol imaging, metabolic testing, complete extra-abdominal staging, and, if the patient remains a candidate, biopsy or resection. Tissue sampling remains the only method of distinction between metastasis and a primary adrenal tumor.² If isolated contralateral adrenal metastasis had been confirmed in a patient with good performance status, metastasectomy combined with cytoreductive nephrectomy would have been a reasonable multidisciplinary option.^{1-3,14} That pathway did not apply to the present patient: he was not a surgical candidate, and isolated metastatic disease was not established. A left pleural effusion cannot be equated with pulmonary M1 disease. When the diagnostic sequence cannot be completed, the diagnosis must remain radiologically suspected, and clinical staging should retain the “c” prefix.⁸

DISCLOSURE OF POTENTIAL CONFLICT OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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