CLINICAL PRACTICE

*Exercises in Clinical Reasoning***Split Decision**Karthik V. Giridhar, MD¹, Gurpreet Dhaliwal, MD^{1,2}, and Lawrence Tierney, MD^{1,2}¹Department of Medicine, University of California San Francisco, San Francisco, CA, USA; ²Medical Service, San Francisco VA Medical Center, San Francisco, CA, USA.

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In this series, a clinician extemporaneously discusses the diagnostic approach (regular text) to sequentially presented clinical information (bold). Additional commentary on the diagnostic reasoning process (italic) is interspersed throughout the discussion.

A 51-year-old man presented to the emergency department with several hours of right-sided flank pain. The pain was moderate, sharp, radiated to the inguinal region, and was associated with nausea. He had no fevers, chills, emesis, dysuria, or macroscopic hematuria. One month earlier, he had experienced similar left-sided flank pain and presented to another facility where nephrolithiasis was suspected after findings on a noncontrast computed tomography (CT) scan were interpreted as being consistent with passage of a left kidney stone.

Flank pain may be due to renal stones, pyelonephritis, renal carcinoma, or any process involving the retroperitoneum, such as hematoma occurring after cardiac catheterization. Nephrolithiasis can be recurrent and occasionally bilateral. In addition, abdominal conditions, such as cholecystitis, hepatitis, colitis, peptic ulcer disease, and pancreatitis can present with flank pain, although bilaterality is unusual.

His medical history included hypertension, hyperlipidemia, benign prostatic hyperplasia, and a Leydig testicular tumor that was treated by orchiectomy ten years earlier. His medications were pantoprazole, atorvastatin, atenolol, tadalafil, etodolac, and a nicotine patch. He had a 25-pack-year history of smoking, consumed three alcoholic beverages per week, and did not use illegal drugs. On physical exam, he was afebrile; his blood pressure was 161/67 mm Hg; heart rate was 79 beats per minute; respiratory rate was 16 breaths per minute; oxygen saturation was 98 % on room air. He was a thin man in mild pain. His heart had a regular rate and rhythm without murmurs or extra heart

sounds. His lungs were clear to auscultation. He was tender over the right lower quadrant of the abdomen and at the right costovertebral angle. He had strong symmetric dorsalis pedis pulses without lower extremity edema. The white blood cell count was 13,000 per cubic millimeter. The hemoglobin was 15.3 grams per deciliter. The platelet count was 255,000 per cubic millimeter. The creatinine was 0.9 mg per deciliter. Coagulation parameters were normal. Urine dipstick testing was negative for blood and protein.

Leydig cell tumors are typically curable, and because a decade has passed without any evidence of recurrence, it is unlikely that retroperitoneal metastases account for his flank pain. Most patients with nephrolithiasis have at least microscopic hematuria. In a patient with vascular risk factors, flank pain raises the possibility of an expanding aortic aneurysm; however, this is typically felt in the left flank, rather than the right, because of a tendency for aneurysms to expand leftward and posteriorly. Furthermore, a symptom-free interval of a month would be unlikely if such a serious vascular event were at play. It is important not to overlook a common abdominal condition, such as appendicitis or cholecystitis, which may be unrelated to his previous presentations.

Frequently, clinicians must contemplate two or more versions of a problem representation¹ early on in a presentation. Here, the discussant considers a middle-aged man with acute flank pain in two iterations—one that labels the event as recurrent (based on a similar but contralateral presentation one month earlier) and one that considers the current presentation in isolation. Infection, nephrolithiasis, and retroperitoneal metastases are potential causes of recurrent but alternating flank pain, while appendicitis and aortic dissection are more applicable to the monophasic event formulation.

A contrast-enhanced computed tomography scan of his abdomen and pelvis revealed multiple wedge-shaped opacities in the right kidney with perinephric stranding consistent with renal infarctions. Wedge-shaped hypoenhancement and lobulation of the left renal cortex suggested a previous left kidney infarct (Fig. 1). The serum lactic dehydrogenase was 304 units/L (normal range of 100–190).

The findings suggestive of a renal infarction surely come as a surprise to the clinician. Renal infarction ordinarily takes a place lower on the differential diagnosis than the entities noted above and may often be overlooked. One

Figure 1. a A contrast enhanced computed tomography scan shows a wedge-shaped area of hypoattenuation throughout the parenchyma of the right kidney (arrow) with perinephric stranding. b The left kidney shows an evolved lower pole infarction with cortical retraction (arrow). The right kidney wedge-shaped hypoattenuation remains.

first thinks of cardiac emboli, although there is no history of atrial fibrillation or valvular disease. In situ thrombosis of the renal artery, or of one of its branches, is another cause, and warrants consideration in this patient with abundant risk factors for vascular disease, although obstructive processes of the renal arteries typically produce unilateral renal infarctions. Aortic dissection, particularly in a hypertensive patient, may be responsible, but aortic dissections that present with intra-abdominal or retroperitoneal symptoms without thoracic symptoms are uncommon. So, one must then consider a cardiac embolus first, followed by in situ thrombosis/atherosclerosis, and finally, a highly atypical iteration of aortic dissection. The lactic dehydrogenase is often extremely elevated in patients with renal infarction.

The discussant reveals important elements of his illness script¹ for renal infarction. He acknowledges that renal

infarction is frequently unsuspected and easily overlooked—and understandably so, given how it is dwarfed in frequency by more common conditions such as nephrolithiasis or infection. As he outlines the conditions that underlie renal infarction, it becomes clear that this patient had no factors that amplified its very low pre-test probability and that the presentation has some features, such as bilaterality, which lower the likelihood even further.

The patient was admitted to the hospital. He remained in normal sinus rhythm while undergoing telemetry monitoring. A transthoracic echocardiogram showed normal left ventricular size and function with no thrombus or valvular abnormalities. Saline contrast showed no evidence of right-to-left atrial shunting. A testicular ultrasound showed a right-sided varicocele and a surgically absent left testicle.

Blood cultures to exclude endocarditis would be reasonable, although the absence of fever and murmur would temper any expectations of a positive result. A period of monitoring to determine if the patient had intermittent atrial tachyarrhythmias was sensible, as those rhythms may come and go. Cardiogenic embolus, whether from a mural thrombus or a more rare cause such as an atrial myxoma, is now quite unlikely.

There is no demonstrated peripheral vascular disease that would predispose him to an in situ thrombosis. Although hypercoagulability warrants consideration, the appreciable majority of patients who present with an arterial thrombosis are not found to have any specific syndrome leading to hypercoagulability. Nonetheless, given the unusual clinical presentation, it would not be unreasonable to screen for hypercoagulability, although one gets the feeling that absent other evidence of pathologic blood clotting, no answer will be forthcoming.

The CT might have been expected to disclose an aortic dissection on careful inspection. Thus, we are pointed to an intrinsic process of the renal arteries, such as polyarteritis nodosa or fibromuscular dysplasia (more commonly seen in young women). There is no report of the typical features of Marfans syndrome or Ehler Danlos syndrome (vascular type), which are two heritable disorders of connective tissue that can weaken the integrity of the blood vessels. When a vessel dissects, the media of the vessel is generally to blame. Such is the case in a typical aortic dissection, where cystic medial necrosis, a somewhat ambiguous and nonspecific pathological term, weakens the wall of the media, resulting in an intimal tear allowing a column of blood to enter the media. Might this process be confined to renal vessels?

Having found no evidence of disease elsewhere in the body, the discussant directs his attention entirely to the renal arteries. The discussant makes an inference that the pathophysiology of an aortic dissection could unfold exclusively in the renal arteries. This is an example of analogical reasoning, where knowledge about a known problem or issue (pathophysiology of aortic dissection) is

used to infer understanding of a new problem (renal artery dissection). In analogical reasoning, the brain must retrieve an example from prior experience to which the new problem can be related, then map a connection between the two problems, and then evaluate the suitability and credibility of the transferred knowledge. Analogical reasoning only establishes a hypothesis, but not a proven fact.²

A renal angiogram demonstrated a focal dissection of a major branch of the right renal artery supplying the upper pole with post dissection aneurysmal dilatation. The left renal artery also showed a major branch vessel with a dissection flap in the area of the previous infarct. The angiographic appearance of the renal arteries was not suggestive of fibromuscular dysplasia, segmental arterial mediolysis, or vasculitis. The aorta, celiac artery, superior and inferior mesenteric arteries appeared normal (Fig. 2). He was diagnosed with spontaneous renal artery dissection.

This idiopathic condition, which describes his renal artery dissections and accounts for his renal infarctions and presenting symptoms, would lead me to ask the following questions: Is this condition likely to occur again? Is renal function threatened? Should the patient be anticoagulated?

His primary care physician held discussions with vascular surgery, cardiology, radiology, and the anticoagulation service. Anticoagulation was advised, but the patient declined it. He was prescribed aspirin. Carotid Doppler ultrasounds showed moderate stenosis but no evidence of dissection. CT angiography of the neck did not show any dissection, occlusion, or aneurysm. One month after discharge, he underwent scheduled interval CT scanning of his chest, abdomen, and pelvis, which excluded vasculopathy elsewhere in the body and demonstrated resolution of his renal infarctions.

Four months later the patient experienced mild flank pain and underwent a CT that did not reveal new intra-abdominal pathology. Three months later, he developed flank pain while working in the yard. His primary care physician examined him and provided reassurance regarding the musculoskeletal etiology for his pain.

CLINICAL REASONING DISCUSSION

Clinical reasoning is typically conceptualized as the cognitive processes that underlie the clinician's ability to elicit and analyze patient data in order to determine a diagnosis and initial treatment plan. The elements of this process were fully displayed in the discussant's analysis. Examination of the patient's current condition—asymptomatic with a vasculopathy of unknown prognosis—exemplifies how clinical reasoning can be a higher order process that considers how *future* encounters can take place in the safest, most efficient, and most cost-effective manner. This broader lens on reasoning and decision making requires that the physician not only be

Figure 2. a Angiography of the right renal artery shows a major branch supplying the upper pole has a focal dissection evidenced by a linear filling defect (arrow). In addition, there are post-dissection luminal filling irregularities, aneurysmal dilatation, and occlusion of the false lumen. b Angiography of the left renal artery reveals a focal dissection flap (small arrow) in a main artery supplying the inferior pole. In addition, there is post-dissection aneurysmal dilatation and distal occlusion of false lumen (large arrow).

well-versed in the canon of medical knowledge, but also be able to engage in shared decision making with the patient and view decisions from the perspective of other providers.

This patient underwent three imaging studies in order to receive the diagnosis of a renal artery dissection. While the CT scans and renal angiogram were medically indicated, it is worth examining the value of *subsequent* imaging studies. In the absence of symptoms, the patient underwent carotid ultrasound and CT imaging studies to assure resolution of his infarctions and exclude clinically silent vasculopathy elsewhere. The additional surveillance imaging studies may have been necessary to assign a label of “spontaneous” (synonymous with “idiopathic”) to his condition so that a systemic condition that required different treatment or monitoring was not overlooked.

Given his predilection to arterial dissections, he is now at risk of undergoing a CT scan every time he has pain (or other symptoms) referable to his head, neck, chest, abdomen, or extremities. Whenever there is unexplained pain or dysfunction, the possibility of an arterial dissection will lurk. The treating physician—particularly one who does not know the patient—may experience powerful incentives (patient safety and liability concerns) that are satisfied with a negative imaging study. Balancing the short-term diagnostic benefits of CT scans with the long-term risks^{3,4} is difficult because physicians (and frequently patients) prefer the increased diagnostic certainty afforded by imaging. Given his decades of remaining expected life, it is plausible that the overuse of imaging may confer greater risk (through contrast and radiation exposure and incidental findings) than the disease itself.

This patient has a well-established relationship with his primary care physician, which is the ideal setting for shared decision making. Open conversations about the patient's perceptions of risks and benefits of future symptom-driven imaging that also prepare him for how other doctors might view the risks and benefits of the same imaging studies will allow the patient to partner with his physicians in navigating both his and their diagnostic uncertainty at scheduled and urgent medical encounters in the future.

CLINICAL TEACHING POINTS

1. Renal infarctions typically present with abdominal pain, hematuria, and elevated serum lactate dehydrogenase (LDH).^{5,6} The most common cause of renal infarction is a thromboembolus from the heart or aorta.⁶ Renal artery dissection is a rare cause of renal infarction.
2. Conditions that predispose to renal artery dissection include extension of aortic dissection, connective tissue disorders, atherosclerotic disease, extreme physical exertion, intravascular cocaine use, malignant hypertension, and segmental arterial mediolysis. The gold standard for evaluating anatomic causes of renal infarction is arteriography.^{7,8}
3. When these conditions are absent, patients are assigned the diagnosis spontaneous renal artery dissection (SRAD).^{7,9,10} The pathophysiology underlying SRAD may be related to local breakdown of the intima or an

intramural hematoma from ruptured vasa vasorum, which causes severe stenosis of the renal artery.¹¹

4. SRAD has a male predominance and typically presents in the fourth to sixth decade of life. SRAD is bilateral in 10–15 % of cases.^{9,10}
5. Options for managing renal artery dissection include blood pressure control, anticoagulation, endovascular stenting, open vascular surgery, and nephrectomy.^{7,9,10,12}

Conflict of Interest: The authors declare that they do not have a conflict of interest.

Corresponding Author: Karthik V. Giridhar, MD; Department of Medicine University of California San Francisco, 505 Parnassus Avenue, San Francisco, CA 94143, USA (e-mail: Karthik.giridhar@ucsf.edu).

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