CLINICAL PRACTICE

Exercises in Clinical Reasoning

NSTEMI or Not: A 59-Year-Old Man with Chest Pain and Troponin Elevation

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A 59-year-old African-American man with a history of hypertension, hyperlipidemia, and cerebrovascular disease presented to the emergency department complaining of chest pain. He stated that the pain began suddenly while he was seated at home several hours prior to presentation. He described the pain as throbbing, 9 out of 10 in intensity, substernal, and associated with nausea, diaphoresis, and dyspnea. He stated that the pain radiated to his left leg but not to his arm or jaw. The pain resolved spontaneously after an hour, but then recurred 3 hours later. He had never experienced pain like this before. He initially delayed coming to the hospital, expecting the chest pain to subside spontaneously again; but finally his family brought him in when he became disoriented and confused.

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One of my rules of thumb when constructing a differential diagnosis is to factor in the abruptness of symptom onset as well as pace. Symptoms that present themselves at “the snap of a finger” suggest to me an underlying vascular catastrophe, including stroke, acute myocardial infarction, aortic dissection, and pulmonary embolism. Although many

of these same entities can present in a stuttering fashion, the specificity of abrupt onset for a vascular event is high.

This patient has several risk factors for coronary artery disease, so we also need to consider coronary ischemia from the outset. Typical or definite angina is defined as being substernal pain with radiation to the arm or jaw that is exertional and is relieved within 10 minutes by rest or sublingual nitroglycerin. In this case, the pain is throbbing and occurs at rest, without mention of the effect of nitroglycerin, and therefore is atypical for angina. The associated autonomic symptoms such as nausea, vomiting, and diaphoresis indeed correlate with severe pain, but they are not specific for coronary artery disease. The duration of the pain is also worrisome if related to coronary ischemia; chest pain at rest of greater than 30 minutes duration portends a worse prognosis in patients with acute coronary syndrome. Finally, he is also at risk for other similarly worrisome entities, including aortic dissection, esophageal rupture, pericarditis, or pneumothorax. The radiation of the pain to the left leg is at this point perplexing and difficult to explain.

A careful and targeted physical examination should help us refine our differential diagnosis. Simple maneuvers, such as checking blood pressure in both arms, examining the patient both supine and sitting, auscultating for the absence of breath sounds unilaterally, and auscultating for the presence of murmurs or gallops, would be of great clinical importance.

DIAGNOSTIC REASONING

This patient presentation describes a diagnostic problem characterized by uncertainty, time pressure, and high stakes. To understand how experts solve such problems, psychologists developed the concept of Naturalistic Decision Making (NDM).¹ Gary Klein proposed one NDM model, called Recognition-Primed Decision Making (RPD). This model followed from observations of expert firefighters’ decision-making processes in the field in real

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 (italics) is integrated throughout the discussion.

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time¹ (as opposed to artificial decisions made in a laboratory). RPD explains how an expert, primed with previous knowledge and experience, solves complex, high-stakes problems under time pressure. While most NDM descriptions involve fields outside of medicine, we can apply its concepts to medicine to understand how an expert reasons through this complex, high-stakes patient presentation.

Like the heuristics and biases model of diagnostic reasoning, RPD uses the concepts of the dual process theory of cognition. In the dual process theory, diagnosticians alternate between system 1 and system 2 thinking. System 1 can be labeled intuitive thinking, allowing the immediate identification of an animal as a dog, an accent as Southern, or cloud formation as an impending thunderstorm. System 1 thinking explains quick pattern recognition. System 1 takes little energy, and is appropriate for many common diagnostic situations in medicine and elsewhere. System 2, the analytical approach, describes a slow, deliberate cognitive process that novice learners frequently use. Experienced and expert clinicians also resort to the analytical approach when confronted with complex patient presentations. Thus, system 2 thinking is useful when the problem becomes more complex, and requires careful and analytical reasoning. Most diagnostic dilemmas require system 2 thinking. Clinicians often shift from system 1 thinking to system 2 thinking when a cue raises concerns over accepting the system 1 answer.

Klein's model incorporates three approaches. Any given decision may require the expert to use one or more approaches. The first approach employs intuitive or system 1 thinking.^{2,3} In medicine, this approach involves pattern recognition compared against a fully formed illness script. What distinguishes experts in this case is that they have more completely developed illness scripts than do experienced non-experts.² Experts not only match more points of the presentation against their illness scripts, but notice more discrepancies. In this discussion, our expert starts with intuition, concluding that the patient is likely having a vascular event.

The second approach resembles system 2 thinking. In RPD, the expert develops a story using his diagnosis, identifies missing data, and then seeks those data.⁴ In this presentation, our expert focuses on developing a story based on the possibility that the patient has acute coronary syndrome, but is uncomfortable using only the intuitive approach because he cannot match the left leg pain to an illness script and does not yet have complete data. He then asks for specific physical examination data. If those data are complete and match his initial thought of coronary artery disease, he will likely revert to the first approach and proceed with this diagnosis.

The third approach involves the use of a mental "pre-mortem" examination.^{2,4} In this approach, our expert

imagines choosing a given diagnosis with its resultant treatment. The expert then mentally simulates the potential consequences. If the expert still has doubts because the mental simulation raises cautions, he will seek more information before committing to the diagnosis.

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(Initial Evaluation in the Emergency Department)

The patient was afebrile with a heart rate of 62 beats per minute, blood pressure of 96/47 mmHg, respiratory rate of 18 breaths per minute, and oxygen saturation of 100 % on ambient air. He was well appearing, in no acute distress. Heart was regular in rate and rhythm and without murmurs, rubs, or gallops. Lungs were clear to auscultation. Abdomen was normal. The extremities were warm, with no edema. The patient had word-finding difficulty attributed by the family to a prior stroke, but no other deficits.

Initial laboratory evaluation: sodium 141 mEq/L, potassium 3.5 mEq/L, chloride 106 mEq/L, bicarbonate 26 mEq/L, glucose 121 mg/dL, blood urea nitrogen 16 mg/dL, creatinine 1.7 mg/dL, creatinine kinase (CK) 82 units/L, CK-MB fraction 1.2 ng/mL, troponin I 0.154 ng/mL (upper limit of normal is 0.039 ng/dL); white blood cell count 12,800 per cubic mm with 85 % neutrophils, 9 % lymphocytes, and 5 % monocytes; hematocrit 44 %, prothrombin time 14 s (normal), partial thromboplastin time 35 s; urine drug screen was negative for barbiturates, benzodiazepines, cannabinoids, and cocaine metabolites. Dipstick urinalysis only demonstrated 2+ proteinuria.

A 12-lead electrocardiogram (ECG) is shown in Figure 1. Chest radiograph is shown in Figure 2. Computed tomography (CT) of the head without contrast demonstrated old lacunar infarcts in the bilateral basal ganglia.

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The physical examination is helpful, but unfortunately incomplete in terms of refining our original differential diagnosis. There is no evidence of a pneumothorax, and the patient does not have findings consistent with left-sided or right-sided heart failure. Absence of a pericardial rub is helpful, but not all positional maneuvers were done to exclude pericarditis. More importantly, there was no measurement of blood pressure in both arms or assessment for symmetry between the radial and femoral pulses. More detailed examination of the left lower extremity would help,

Figure 1. Electrocardiogram on presentation to the emergency department. Note sinus rhythm, rate of 57 per minute, PR interval of 160 ms, QRS duration of 78 ms, and symmetric T wave inversions in leads V3 through V6 with evidence of left ventricular hypertrophy.

considering the radiation of pain there. The vital signs are worrisome in someone with a history of hypertension, although the patient appeared hemodynamically stable upon arrival at the emergency department.

The ECG and laboratory data are rather concerning. The deep T wave inversion in V3 through V6 would suggest

ischemia of the anterior and lateral walls of the left ventricle. It is important to look carefully at the shape of the T waves, with symmetric inversion being more specific for myocardial ischemia. The mild elevation of cardiac biomarkers suggests myocardial injury. The elevated troponin could also constitute “troponin leak,” although there is not a

Figure 2. Chest radiograph in emergency department (portable). Note clear lung field, possible cardiomegaly and a tortuous aorta.

readily identifiable cause, such as markedly elevated blood pressure. The mild elevation of creatinine could represent chronic kidney disease; but if this is a new finding, we would need to reconcile the chest pain with a condition causing acute kidney injury. The patient does not appear to be in shock. There is no good reason in the history for volume depletion, which raises the possibility that the renal insufficiency could reflect an ischemic insult as well. Measurement of blood pressure in both arms appears to be rather important, if not critical, since it is urgent to decide whether or not to initiate heparin to manage acute coronary syndrome.

DIAGNOSTIC REASONING

Now our expert continues to consider a diagnosis of acute coronary syndrome. In his RPD research, Klein found that experts focus on one diagnosis and accept or exclude that diagnosis before looking for a second diagnosis.² While much of the data supports the acute coronary syndrome diagnosis, our expert needs some additional data before crossing the required diagnostic threshold and initiating a potentially harmful therapy. His illness script does not let him proceed with treatment based on the current data. By using the third RPD approach, he mentally simulates giving anticoagulation to this patient. Since he has not excluded aortic dissection, he remains uncomfortable and unwilling to declare a diagnosis. Thus, he requires additional information.

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(Reassessment in the Emergency Department)

The patient received 324 mg of aspirin and 1 L of 0.9 % sodium chloride with subsequent improvement of blood pressure. The chest pain resolved, and a repeat neurologic exam was unchanged. A second set of cardiac biomarkers showed a CK-MB of 5.0 ng/mL and troponin I of 0.643 ng/mL. Repeat ECG demonstrated resolution of the T wave inversions. The patient received heparin drip and was admitted to a telemetry monitored bed with the diagnosis of non-ST segment elevation myocardial infarction (NSTEMI).

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Dynamic changes in an ECG coupled with an increase in cardiac biomarkers are, at first glance, robust evidence that we are dealing with acute coronary ischemia. From this

perspective, the initiation of antiplatelet and anticoagulant therapy would appear to be correct, although I am still not sure we have excluded conditions for which heparin could be potentially devastating, such as acute aortic dissection. More time and effort should have been devoted to exploring the possibility of dissection.

DIAGNOSTIC REASONING

Our expert continues his mental simulation and continues to describe his discomfort. This discomfort stems from both an incomplete match to his illness script and the mental simulation warning of dire consequences from anticoagulation for acute coronary syndrome if the patient in fact has aortic dissection. Without the particular data he has requested, he cannot yet cross the threshold to begin treatment for acute coronary syndrome.

CLINICAL INFORMATION

The patient's chest pain recurred upon arrival to the medical floor. The pain was similar but less intense. The blood pressure was 172/86 mm Hg. Lungs were clear to auscultation. He had a crescendo-decrescendo III/VI systolic murmur at the right upper sternal border and left lower sternal border, and a decrescendo II/VI diastolic murmur at the left lower sternal border. No carotid bruits were noted. Abdomen was non-tender with no bruits. He had a subtle left-sided facial weakness and 4/5 strength on the left upper and lower extremities. Speech was consistent with expressive aphasia. At this time, troponin I had elevated to 1.726 ng/mL. He received high-dose atorvastatin, low-dose oral metoprolol, and a nitroglycerin drip; the heparin drip was continued. He was not given clopidogrel out of concern that surgery would be required to treat his possibly acute valvular dysfunction.

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The presence of a diastolic murmur in a patient with chest pain should alert the clinician to the possibility of a proximal aortic dissection. Acute mitral regurgitation or a ventricular septal defect (VSD) murmur would be suggestive of coronary ischemia; however, both of these murmurs have different acoustic characteristics from those found in this patient. Both mitral regurgitation and VSD murmurs are holosystolic in nature; in addition, acute mitral regurgitation is usually associated with pulmonary edema, and a VSD murmur is frequently accompanied by a thrill. The

Figure 3. Coronal image from the computed tomography angiogram of the chest, demonstrating a Stanford type A aortic dissection extending down the length of the thoracic descending aorta.

possibility of this diastolic murmur representing acute aortic insufficiency makes a proximal arch dissection a more likely explanation than myocardial ischemia. Other findings, including the neurological deficits and the possible acute kidney injury, suggest simultaneous ischemia in

several vascular territories, making acute coronary syndrome an incomplete explanation.

Finally, and perhaps most telling, the blood pressure measured on the medical floor raises a rather alarming possibility. On the medical floor, the blood pressure could

Figure 4. Transverse image from the computed tomography angiogram of the chest, demonstrating the involvement of the left main coronary artery and the aortic valve annulus. The dissection flap in the descending aorta is also seen.

have been checked in the arm opposite to the one checked in the emergency department, thus suggesting the possibility of discrepant perfusion to the upper extremities. At this point, I would suggest that the data make aortic dissection the most likely diagnosis. It would be intriguing to speculate that the leg pain is related to an extensive dissection down to the left femoral artery, which would also affect the renal arteries. Immediately, I would stop the heparin drip and do a confirmatory test: either CT angiography despite the abnormal creatinine, or a trans-esophageal echocardiogram, depending on the institution and the time of day.

DIAGNOSTIC REASONING

These new data allow our expert to reject the initial diagnosis and proceed to a new diagnosis. The murmur and the discrepant blood pressures support the possibility of aortic dissection. He can now explain the confusing history of leg pain as well as the elevated creatinine. He then mentally simulates this new diagnosis and asks for definitive proof because the treatment for aortic dissection requires anatomical confirmation. His mental simulation indicates that the potential harm from obtaining the anatomical information (contrast nephropathy) is outweighed by the potentially fatal consequence of leaving aortic dissection undiagnosed. He asks to stop the anticoagulation now that he can abandon acute coronary syndrome as a possible diagnosis. Now that he has all the relevant data, he can proceed rapidly down the path of confirming the diagnosis and treating the patient.

We can also explain our expert's approach based on how his doubt informed his desire to "slow down." In a parallel approach to Klein's, Carol-anne Moulton argues that experts differ from experienced non-experts because they know when to slow down and reconsider the diagnosis or treatment.² Our expert quickly shifts from system 1 thinking to system 2 thinking because the presentation has incomplete and confusing data. Experts do not require comprehensive data, but they do require all relevant data.

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Three hours later, the patient's nurse notified the admitting physician that the patient was having chest pain again. The physician noted a difference in blood pressure between arms (left, 180/60 mmHg; right, 120/40 mmHg). An emergent CT angiogram of the chest demonstrated an extensive, complex Stanford type A aortic dissection extending from the aortic valve root

Table 1. Examples of Recognition-Primed Decision Making (RPD) in this Clinical Scenario^{4,6}

RPD Variation	Description	Example from Clinical Scenario
Simple Match	Intuitive approach	Abrupt onset of symptoms suggests a vascular problem
	Pattern recognition	Risk factors suggest coronary disease is a highly likely cause of this patient's chest pain ECG suggests coronary ischemia Elevated biomarkers suggest myocardial injury New diastolic murmur is concerning for proximal aortic dissection
Diagnose the Situation	Create stories explaining the situation, develop expectancies, identify relevant cues, and describe goals and actions Identify any anomalies in the present situation	Identifying the pain as atypical for angina Identifying symptoms inconsistent with acute coronary syndrome (duration of pain, radiation to leg, low blood pressure with no signs or symptoms of cardiogenic shock) Detailing focus of further evaluation Neurologic symptoms and renal insufficiency are not explained by acute coronary syndrome Elevated blood pressure on general medical floor is possibly consistent with discrepant perfusion to the upper extremities
Evaluate Course of Action	Mental simulation of current course of action "Pre-mortem" analysis of potential causes of failure	Appropriate to initiate antiplatelet and anticoagulant therapy for acute coronary syndrome; however, treating acute coronary syndrome if acute aortic dissection has not been excluded would result in devastating consequences
	Redirecting course of action if necessary	Once other data suggest aortic dissection, the need for anatomic data outweighs the risk of contrast nephropathy sufficiently to order an emergent CT angiogram in the setting of renal insufficiency

beyond the renal arteries to the common iliac arteries (Fig. 3). It involved the left coronary artery origin, with decreased flow in the left coronary system (Fig. 4). There was a wedge-shaped infarct in the left kidney. An emergent echocardiogram confirmed severe aortic regurgitation with a dilated aortic root and a dissection flap. The patient underwent emergent surgical repair. He was eventually discharged to inpatient rehabilitation

to recover from the embolic strokes he had sustained during the dissection.

DISCUSSION

Undergraduate and graduate medical education should foster critical thinking with the ultimate goal of developing diagnostic expertise. Learners develop expertise through the experience of seeing patients and thinking about their diagnoses systematically. Expertise requires more than knowledge; it requires the development of fully formed illness scripts.⁵ These illness scripts can be used to develop a basis for system 1 thinking in simple scenarios, but they also provide a way of analyzing more complicated presentations so that an expert can quickly decide when to switch to system 2 thinking.³⁻⁷ Upon switching to system 2 thinking, RPD provides a framework for mentally simulating a diagnosis and treatment, and for predicting potential adverse outcomes.^{1-3,6}

Aortic dissection is a complex disease that does not always present as a “tearing chest pain radiating to the back.” This patient’s presentation can be explained retrospectively. The radiation of the pain to the left leg likely indicated the extension of the dissection into the iliac artery; the pulse deficit usually assists with the diagnosis and portends worse outcomes.⁸ The ECG abnormalities, troponin elevation, renal failure, and neurologic deficits were likely the result of arterial occlusion (from either dissection or embolism). The management of aortic dissection is reviewed in detail elsewhere.⁹ Anticoagulants inadvertently given to patients with aortic dissection have been associated with a worse outcome,^{10,11} but fortunately, this patient did not suffer a bleeding complication.

In Stanford type A aortic dissection (involvement of ascending aorta), prompt surgical treatment is essential.¹² A delay in diagnosis has been noted in patients presenting with ischemic changes on ECG.¹³ Acute coronary syndrome-like ECG findings occur in 26 % of Type A aortic dissections.¹⁴ Guidelines provide additional recommendations for imaging.⁹ Our expert was able to correctly identify the relevant information that would lead to the proper diagnosis and treatment in a timely manner. His use of the reasoning steps modeled by RPD is depicted in Table 1. The reasoning techniques described by RPD kept him focused on obtaining all the relevant information, rather than prematurely accepting the initial diagnosis of NSTEMI. Consequently, the RPD framework provides a context that enables the clinician to make a correct diagnosis under time pressure while dealing with an uncertain, but high-stakes, problem.

Clinical Teaching Points

- Atypical features or signs can lead to delays in diagnosis of aortic dissection. An ischemic pattern on ECG is not uncommon in aortic dissection.
- Aortic dissection should be considered while assessing a patient with sudden onset chest, back, or abdominal pain; new neurologic deficits with acute pain; new aortic insufficiency or hypotension.
- Diagnoses should be compared with fully formed illness scripts, not only to determine how closely they match, but also to identify missing data, which should be promptly gathered.
- Mentally simulating possible effects of therapy is an effective way to conduct a “pre-mortem analysis” to weigh the consequences of a decision.

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

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The case presentation and clinical discussion were presented at a monthly Clinical Problem Solving conference at the University of Alabama at Birmingham; hence, the case discussion closely represents the actual discussion. Dr. Schleifer is now a cardiology fellow at Mayo Clinic Scottsdale, Arizona.

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REFERENCES

1. Klein G. Naturalistic decision making. *Hum Factors*. 2008;50:456-60.
2. Klein GA, Calderwood R, Clinton-Cirocco A. Rapid decision making on the fire ground. *Proc Hum Factors Ergon Soc Annu Meet*. 1986;30:576-80.
3. Kahneman D, Klein G. Conditions for intuitive expertise: a failure to disagree. *Am Psychol*. 2009;64:515-26.
4. Zsombok CE, Klein G, eds. *Naturalistic Decision Making*. Lawrence Erlbaum Associates, Mahwah NJ, 1997. “The recognition-primed decision model: looking back, looking forward,” 285-92.
5. Klein G. The Recognition-Primed Decision (RPD) Model: Looking Back, Looking Forward. In: (Zsombok CE, Klein G, eds.) *Naturalistic Decision Making*. Mahwah, NJ: Lawrence Erlbaum Associates; 1997:285-92.
6. Klein G. “The power of mental simulation.” In: *Sources of power: how people make decisions*. Boston: Massachusetts Institute of Technology; 1998: 45-74.
7. Moulton C-a, Regehr G, Lingard L, Merritt C, MacRae H. “Slowing down when you should”: initiators and influences of the transition from the routine to the effortful. *J Gastrointest Surg*. 2010;14:1019-26.
8. Bossone E, Rampoldi V, Nienaber CA, Trimarchi S, Ballotta A, Cooper JV, Smith DE, Eagle K, Mehta RH. Usefulness of pulse deficit to predict in-hospital complications and mortality in patients with acute type A aortic dissection. *Am J Cardiol*. 2002;89:851-5.
9. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, Eagle KA, Hermann LK, Isselbacher EM, Kazerooni EA, Kouchoukos NT, Lytle BW, Milewicz DM, Reich DL, Sen S, Shinn JA, Svensson LG, Williams DM. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/

- SVM Guidelines for diagnosis and management of patients with thoracic aortic disease. *Circulation*. 2010;121:e266–369.
10. **Hansen MS, Nogareda GJ, Hutchinson SJ.** Frequency of and inappropriate treatment of misdiagnosis of acute aortic dissection. *Am J Cardiol*. 2007;99:852–6.
 11. **Davis DP, Grossman K, Kiggins DC, Vilke GM, Chan TC.** The inadvertent administration of anticoagulants to ED patients ultimately diagnosed with thoracic aortic dissection. *Am J Emerg Med*. 2005;23:439–42.
 12. **Tsai TT, Nienaber CA, Eagle KA.** Acute aortic syndromes. *Circulation*. 2005;112:3802–13.
 13. **Harris KM, Strauss CE, Eagle KA, Hirsch AT, Isselbacher EM, Tsai TT, Shiran H, Fattori R, Evangelista A, Cooper JV, Montgomery DG, Froehlich JB, Nienaber CA.** Correlates of delayed recognition and treatment of acute type A aortic dissection. The International Registry of Acute Aortic Dissection. *Circulation*. 2011;124:1911–18.
 14. **Biagini E, Lofiego C, Ferlito M, Fattori R, Rocchi G, Graziosi M, Lovato L, di Diodoro L, Cooke RMT, Petracci E, Bacchi-Reggiani L, Zannoli R, Branzi A, Rapezzi C.** Frequency, determinants, and clinical relevance of acute coronary syndrome-like electrocardiographic findings in patients with acute aortic syndrome. *Am J Cardiol*. 2007;100:1013–9.