CLINICAL PRACTICE

Exercises in Clinical Reasoning

The Importance of Framing

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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 (italics) is integrated throughout the discussion.

CLINICAL INFORMATION

A 43-year-old African-American man with a past medical history of HIV/AIDS, congestive heart failure (CHF) with ejection fraction 25–30%, prior polysubstance abuse, and a remote history of tuberculosis (TB) infection was transferred to our institution for further evaluation of worsening shortness of breath and chest pain. The patient reported chest pain and dyspnea for 2 months, which gradually worsened 48 h prior to admission. His chest pain was substernal, dull, and non-pleuritic, and was exacerbated by exertion and alleviated by rest. On full review of systems, he also had non-specific generalized intermittent epigastric abdominal pain and feeling “hot and cold” over the last several days, a productive cough for 2 months, anxiety and diarrhea for 5 months, and 60-lb unexplained weight loss over 6 months.

Clinician: Our patient is a middle-aged man with HIV/AIDS and systolic CHF who presents with sub-acute progressive dyspnea and chest pain that suddenly worsened. He also has several other sub-acute and chronic symptoms that may or may not be related to the present illness. Due to the presence of AIDS, we will need to consider a broadened differential to include conditions seen in immunocompromised patients.

Clinicians should always consider potentially life-threatening illness in the diagnostic process. If we first focus on his acute chest pain and dyspnea, we would consider acute coronary syndrome (which has a higher incidence in HIV

patients), acute pulmonary embolism, aortic dissection, pneumothorax, esophageal perforation, and pericardial disease. With his dyspnea of 2 months' duration, cough, and other symptoms, these emergent conditions seem less likely.

Pulmonary edema from a heart failure exacerbation, or other alveolar-filling processes, such as pneumonia, seem more likely. Common organisms for pneumonia, such as *S. pneumoniae*, *M. catarrhalis*, and *H. influenza*, are less likely given the sub-acute pulmonary symptoms and lack of fever. With his immunocompromised status, the etiology of pneumonia includes many pathogens that may present more slowly. For example, *Mycobacterium avium* complex (MAC) is consistent with the patient's gastrointestinal symptoms, pulmonary findings, and weight loss.

The patient's CD4 count will be a critical piece of information, as it will determine his risk for opportunistic infections (OIs), especially *Pneumocystis jirovecii* pneumonia (PJP), MAC, or fungal infections. If the patient recently started antiretroviral therapy, consequences of immune reconstitution such as immune reconstitution inflammatory syndrome could cause his symptoms. TB should be strongly considered, regardless of CD4 count, given his past history of infection, chronic cough, and significant weight loss. If he has profound immunosuppression, he could have multiple etiologies explaining the multiple symptoms.

To be complete in our thinking, other pathologies to consider that cause chest pain and shortness of breath include autoimmune disease, vascular disease, trauma, and substance abuse. He has a history of polysubstance abuse, and we need to know more about the types of substances, and whether this is still an active issue.

The differential diagnosis for chest pain and dyspnea is extensive, and is especially broad in patients with HIV/AIDS and polysubstance abuse. The clinician states that there are many variables to consider, and in doing so recognizes the challenges of composing a problem representation^{1,2} in such a complicated patient, especially early in the clinical decision-making process. A problem representation is a mental construct developed by the clinician to succinctly define the case in abstract terms¹. An accurate problem representation incorporates all key elements into an initial diagnostic frame. The frame through which clinicians view a patient will have a large impact on the formulation of their problem representation and can also lead to diagnostic errors.

Little is published in the medical literature about framing. One way to consider framing is related to the clinician's careful selection of elements to include in the problem representation. If the clinician fails to include an important piece of information in the frame, the problem representation could be wrong, and the

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diagnosis missed³. How clinicians decide what information to include in the frame as they develop diagnostic hypotheses is unknown. In 1981, Tversky and Kahneman described the psychology of framing of decisions.⁴ They defined a decision-frame as “the decision makers conception of the acts, outcomes, and contingencies associated with a particular choice.” The frame that decision makers choose is based both on their understanding of the problem and on any personal experiences, biases, or personal characteristics that they possess, including perceived probabilities of outcomes.^{4,5} Individuals are more likely to make risk-averse decisions when presented with possible negative outcomes.⁵ Thus, in this case, the clinician first considers life-threatening causes of chest pain and dyspnea, and then includes AIDS and degree of immunosuppression in the frame, as the probability of a negative outcome from an opportunistic infection is high, if not considered.

Another way to consider framing is related to medical errors, known as the framing effect. In a classic paper on cognitive errors, Croskerry³ includes the framing effect as a cause of diagnostic error. This effect is defined as how clinicians “see things” and may be influenced by the way in which the problem is framed to them. If the information is framed incorrectly to the clinician by patients or other healthcare professionals, the physician’s perceptions of the severity of risk (including outcomes and contingencies) can lead to diagnostic error. In reality, it is difficult to separate these concepts, due to our limited understanding of what clinicians consciously and subconsciously include in their frame. Subconsciously, the framing effect may also impact what a clinician includes for framing decisions.

The patient was diagnosed with HIV in 1994. He was followed intermittently at the local HIV clinic, with inconsistent adherence to antiretroviral medications for 20 years. His nadir CD4 count was six cells/mL in 2011, and most recently was 152 cells/mL 3 months prior to presentation. He had no history of prior OIs, except for a remote TB history. Systolic CHF was diagnosed at an outside hospital 2 months prior to this admission, but no evaluation was done for ischemia. His medications included albuterol, aspirin, dapsone, darunavir, emtricitabine-tenofovir, ritonavir, and lisinopril. He reported adherence to his antiretroviral medications.

The patient was a current smoker of tobacco (12 pack-years) and marijuana, had a history of heavy alcohol and cocaine use (he reported cessation 2 months prior, following his last hospitalization). His mother had end-stage renal disease and hypertension, and grandmother had a myocardial infarction at an unknown age.

Clinician: The patient’s present CD4 count will help determine whether the dyspnea could be from pneumonia with an OI, TB, or non-infectious etiologies. Given his most recent CD4 count of 153, he is at risk for OIs, especially PJP and TB at this level. We also need to know his viral load to gain an understanding of the overall disease control. The patient’s history of CHF is unusual given his age. While cocaine and alcohol use could explain his CHF, there are other causes of non-ischemic cardiomyopathy to consider, such as viral, metabolic, infiltrative, or valve disease. The family history of CAD and his tobacco use

predispose him to premature atherosclerotic disease, but CAD does not explain his weight loss, cough, temperature dysregulation, or gastrointestinal symptoms.

Given his dramatic weight loss with history of tobacco and alcohol, lung, stomach, and esophageal cancers should be considered.

The additional information discovered in the patient’s past social and family history has added complexity to the case. The clinician continues to account for features he included in the original framing, including the patient’s degree of HIV control, etiologies for CHF in a middle-aged patient, and multiple other symptoms that seem unrelated. The clinician recognizes that polysubstance abuse may be the cause of the heart failure, but this alone does not explain the multitude of other symptoms. With the history complete, the clinician has both consciously and subconsciously framed the patient to create his problem representation of the case. The initial process of framing a new case is mostly subconscious, and occurs within the domain of intuitive or reflexive decision making, system 1.⁴ However, complicated, ill-defined clinical cases are especially challenging, even for expert clinicians. Thus, the clinician must also use analytical, slow, deliberate clinical reasoning, system 2.^{3,6} If the clinician discounts information that is relevant, and does not include it in the frame, a framing error can occur.³ Framing errors at the initiation of the cognitive process are directly related to errors at the end of cognitive process.⁷

On physical exam, the patient’s temperature was 98°F, blood pressure 114/82 mmHg, heart rate 120 beats/min, respiratory rate 22 breaths/min, and oxygen saturation 94% on 2 L nasal cannula. He was anxious, had increased work of breathing, and was in mild distress. He had dry mucous membranes with prominent jugular veins distended to the jawline when sitting up. He had decreased breath sounds at the right lung base, rhonchi throughout, and few scattered expiratory wheezes. He was tachycardic with no murmurs appreciated. His abdomen was soft, non-distended, and non-tender to palpation. He had trace lower extremity edema with normal perfusion and pulses.

Clinician: Tachycardia is a notable finding on the physical exam. The patient is not febrile and not exerting himself, yet his heart seems to be working hard at rest. Tachycardia is common in patients with CHF exacerbation. I would review prior clinic records to ensure this finding is acute. The patient is hypoxemic in conjunction with decreased breath sounds in the right lung base, supporting an alveolar filling process such as pulmonary edema or pneumonia, as we initially considered. The diffuse wheezing on pulmonary exam could suggest underlying emphysema or asthma, but could also be reactive due to his CHF with fluid overload. The focal findings of decreased breath sounds in the lung base call for differentiation between consolidation and effusion, which ideally would have been done with percussion or tactile fremitus.

The cardiac exam needs further clarification about the point of maximal impulse, distant heart sounds, and other cardiac findings to help further inform possible cardiac pathology. The benign abdominal exam suggests that this process is above the diaphragm.

Chest x-ray (CXR) showed right pleural effusion with bilateral alveolar opacities and cardiomegaly (Fig. 1). A computed tomography angiography (CTA) done at the transferring hospital showed no evidence of pulmonary embolism, but did show the right pleural effusion and probable compressive atelectasis in the right lower lobe. His electrocardiogram (ECG) showed sinus tachycardia of 116 beats/min, left atrial enlargement, Q waves in V5/V6, and T-wave inversion in V6. The chemistry panel was unremarkable. His white blood count was 3970/mm³ (26% neutrophils, 59% lymphocytes, 12% monocytes), hemoglobin 11.3 g/dL, normal mean cytoplasmic volume, and platelet count of 113,000/mm³. His aspartate transaminase (AST) was 58 units/L, alanine transaminase (ALT) 47 units/L, and alkaline phosphatase 157 IU/L. The patient had B-type natriuretic peptide (BNP) of 2526 pg/mL and troponin 0.081 ng/mm.

The CD4 count this admission was 351 cells/mL. The urine drug screen had cannabinoid, benzodiazepines, and buprenorphine. The patient was empirically started on vancomycin and cefepime for possible pneumonia given his dyspnea, tachycardia, and CXR findings. Review of outpatient records did indicate a persistent tachycardia prior to this admission.

Clinician: The CD4 count is greater than 200 cells/mL, making OIs less likely. The CXR is consistent with the physical exam findings and our prior suspicion of CHF or pneumonia. Given the information at hand, there is not a clear reason for CHF exacerbation in this patient. His cardiac enzymes and ECG are not consistent with acute coronary syndrome, he is not anemic, and the urine drug screen excludes recent stimulant use. The patient's persistent unexplained tachycardia is intriguing, given that it is found in a patient with significant weight loss and hyperdefecation over the past few months. Connecting these

symptoms into an illness script would make it appropriate to order a thyroid-stimulating hormone (TSH). Hyperthyroidism is a possible cause of CHF in a young patient. He should undergo thoracentesis to further evaluate the pleural effusion.

His TSH came back undetectable, with a high T4 of 2.75 ng/dL. Echocardiogram once again revealed decreased ejection fraction with a dilated left ventricle. Thoracentesis yielded transudate fluid. Blood cultures were negative.

Closer physical exam found a non-tender, enlarged thyroid with audible thyroid bruit, exophthalmos, lid lag, and pretibial myxedema. Evaluation confirmed the diagnosis of Graves' disease. The patient was started on methimazole, his heart failure regimen was optimized, and his clinical symptoms resolved.

The clinician identified a key finding in the physical exam, tachycardia. Review of the prior clinic records showed that this was present at multiple prior clinic visits, and was not an acute finding. As he considered the causes of persistent tachycardia, hyperthyroidism entered into his differential diagnosis. Once the clinician added persistent tachycardia to his frame and developed a more accurate problem representation, the illness script of hyperthyroidism-induced CHF was recognized, and in doing so, yielded the correct diagnosis. Although not noted on the initial physical exam, findings of hyperthyroidism (tachycardia, thyroid bruit, lid lag), and more specifically Graves' disease (diffuse symmetric goiter, exophthalmos, and pretibial myxedema), were evident. Unfortunately, they were initially not recognized by the treating physicians, likely due to the complexity of other symptoms and signs. This case demonstrates how the elements included in (or excluded from) the initial frame can affect diagnostic reasoning, and how reframing a problem representation may be needed in complicated patients.

Figure 1 Chest x-ray on admission with right pleural effusion, bilateral opacities, and cardiomegaly.

DISCUSSION

The dual process theory is the leading model for explaining clinician decision-making.⁸ The theory proposes two cognitive systems used during reasoning. The first is described as intuitive and reflexive, occurring as an automatic process derived from the clinician's illness scripts and past experiences. The second is an analytical process which involves slow and deliberate thinking and thus increased cognitive effort.^{8,9} This case reinforces the notion that the two systems are complementary, not opposing,^{6,10} and highlights how framing is important for both systems. The initial process of framing a new case is mostly subconscious, occurring within the domain of system 1, and is key to hypothesis generation and developing a problem representation. It also allows clinicians to consider more elements as they shift to system 2 with slow, analytical thinking. Failure to include the correct initial framing for a problem representation, or allowing one to be biased by the framing effect, may result in a missed diagnosis or treatment, demonstrating that the two concepts are interdependent.

Little is known about the process physicians use when selecting a frame.¹¹ Clinical variables are provided in unorganized, uncertain, and possibly irrelevant ways.¹² Clinicians organize these variables into a mental construct based on existing knowledge, experience, clinical judgment, and social context.⁷ It occurs without effort and often in system 1 thinking. In complicated patients with multiple clinical variables, the elements included in, and those excluded from, the frame can affect diagnostic reasoning.¹ Framing a patient correctly is necessary to efficiently develop the correct differential diagnosis; incorrect framing can lead the clinician astray. In Table 1, we show how different frames lead to different diagnostic considerations.

In a qualitative study of cases of error, Balla et al. found that cognitive biases developed at initial framing were related to diagnostic errors later in care.⁷ Of course, the initial problem representation will affect subsequent diagnostic evaluation. Our patient was initially viewed in the context of his immunocompromised status and heart failure. Thus the initial physical exam focused on signs related to these conditions. As the case unfolded, our clinician identified a key finding in the physical exam, persistent tachycardia. Although a non-specific finding, the clinician recognized this as a symptom he could not otherwise explain. When he considered the causes of tachycardia, hyperthyroidism entered into his differential diagnosis. With this simple exam finding, the clinician shifted from system 2 back to system 1. He subconsciously readjusted his frame to include tachycardia, and in doing so discovered the correct

Table 1 Differences in Differential Diagnosis with Changing Frames

Key elements in the frame	Differential diagnosis
HIV (CD4 < 200 units), subacute cough, dyspnea, and hypoxia	Pneumonia from atypical or opportunistic pathogens, such as <i>Pneumocystis jirovecii</i> pneumonia, fungal, or mycobacterial (tuberculosis)
HIV, hyperdefecation, abdominal pain	Bacterial or viral gastroenteritis, <i>Legionella</i> Opportunistic infections: <i>Mycobacterium avium</i> complex, cryptosporidium, cytomegalovirus colitis Mesenteric vascular occlusion
Tobacco abuse, cough, dyspnea, and weight loss	Lung cancer, esophageal cancer with thoraco-esophageal fistula, emphysema
Substernal chest pain, dyspnea	Congestive heart failure exacerbation, acute coronary syndrome, pneumothorax, pulmonary thromboembolism
Polysubstance abuse and weight loss	Malnutrition, wasting syndrome, withdrawal symptoms, drug toxicity
Weight loss, anxiety, hyperdefecation, tachycardia	Hyperthyroidism

Table 2 Common Presenting Findings with Graves' Disease

Symptoms	Palpitations, weight loss, hyperdefecation, heat intolerance, perspiration, irritability, anxiety
Physical exam	Ophthalmopathy: proptosis, chemosis, periorbital edema, tachycardia, lid lag, goiter, tremor, moist skin, thyroid dermopathy
Lab values	TSH low, T4 high, thyroid peroxidase enzyme antibodies, homogeneous, increased radioiodine uptake

Adapted from Jameson, J, Mandel SJ, Weetman AP. Disorders of the Thyroid Gland¹³

diagnosis. Although not noted on the initial physical exam, findings of hyperthyroidism, and specifically Graves disease (Table 2), were evident on admission, and may have been missed at several prior healthcare visits. The patient's history of past drug and alcohol use, as well as his intermittent compliance with care, may have impacted prior framing, and specifically caused a delay in treatment due to framing effect. Especially pertinent in this case, clinicians should be aware that physical appearance and behavioral cues can make a powerful impact on the framing of a case.⁷

Once tachycardia was added to the frame, hyperthyroidism-induced CHF was considered. His problem representation evolved and a correct diagnosis was made. His symptoms of weight loss, anxiety, diarrhea (actually hyperdefecation), non-ischemic CHF causing pulmonary edema, and pleural effusion causing dyspnea all fit this singular diagnosis. By excluding HIV/AIDS from the frame, chronic tachycardia, hyperdefecation, and unexplained weight loss in isolation would likely elicit an intuitive, system 1, diagnosis of hyperthyroidism in practicing clinicians.

How physicians view a patient can be shaped and influenced by several different elements in the clinical environment.³ Tversky and Khaneman likened different frames of the same clinical decision to different perspectives of the same image.⁴ In medicine, the patient's clinical presentation is our image, and how clinicians frame patients can ultimately change our perspective of the case. Framing is partially inherent, based on our own biases, patterns, and experiences. Therefore, as clinicians, we must be vigilant in understanding the rationality of a frame in which we view the patient.

CLINICAL TEACHING POINTS

1. Graves disease is the most common autoimmune disease and causes 50–80% of hyperthyroidism cases. Thyroid-stimulating antibodies result in hypersecretion of the thyroid hormone as well as hypertrophy and hyperplasia of the thyroid follicles. The buildup in the thyroid follicles results in the characteristic goiter seen in Graves' disease patients.¹⁴
2. Physical exam findings of ophthalmopathy can be seen in up to 50% of Graves' patients. Ophthalmopathy most commonly presents as eyelid lag and periorbital edema;

however, patients may also present with exophthalmos, diplopia, and other less common ocular symptoms.¹⁴

3. In a study of 591 patients with hyperthyroidism, 6% presented with CHF. Of these 34 patients, 47% had systolic left ventricular dysfunction. Patients in this trial experienced improvement in left ventricular function upon reaching a euthyroid state.¹⁵

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