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# Patient cueing, a type of diagnostic error

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## ABSTRACT

Diagnostic failure can be due to a variety of psychological errors on the part of the diagnostician. An erroneous diagnosis rendered by previous clinicians can lead a diagnostician to the wrong diagnosis. This report is the case of a patient who misdiagnosed herself and then led an emergency room physician and subsequent treating physicians to the wrong diagnosis. This mechanism of diagnostic error can be called patient cueing.

**Keywords**
Diagnostic error; Cognitive aspects; Error sources; Misdiagnosis; Autopsy

## CASE REPORT

A 70-year-old white female former 9-1-1 emergency telephone operator had a history of hypertension, depression, gallstones, hysterectomy (at age 48) and a 3 cm tubulovillous adenoma of the sigmoid colon removed endoscopically 8 years ago. She had severe chronic obstructive pulmonary disease and a 50 pack-year history of smoking, ending 12 years ago. The patient lived alone.

Four years earlier, she had been diagnosed with a well differentiated adenocarcinoma of left lower lobe lung with a bronchioloalveolar appearance. Full-body positive emission tomography/computerized tomography (PET/CT) scan did not suggest any spread to the brain, bones, liver or adrenal glands.

The patient had such severe lung disease that she required home oxygen at 2 L/minute at rest to maintain saturations over 90%. The risk of perioperative death or incapacitation if she were to have a left lower lobectomy was very significant, so she received only therapy with pemetrexed and bevacizumab, which continued over the next four years. CT a month ago showed unchanged residual treated tumor in the left lower lobe with no evidence of thoracic metastases.

The patient was brought to the emergency department, 14 days after her last chemotherapy, for weakness, fatigue, and intermittent nausea and vomiting for the past 10 days and diarrhea for the past 2 days. Her oral intake had been decreased. The patient also mentioned an incidental symptom of left shoulder pain, which started after she carried her oxygen tank on her shoulder when she was out for the first time since heavy snowfall about 6 weeks prior had rendered her housebound, so she was not used to carrying something so heavy. The patient attributed her shoulder pain to carrying her oxygen tank on it. She denied chest pain, dyspnea, fevers or chills. She was on numerous medications including an oral opioid and prednisone.

On admission her temperature was 36.7 degrees C, heart rate 126/minute, blood pressure

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100/69 mm Hg, respirations 20/minute and saturation 97% on supplemental oxygen at 3 L/minute via nasal cannula. She was anxious, alert and oriented. She had decreased breath sounds at the bases. Her heart had a regular rhythm. Her abdomen was soft and non-distended. She had no edema. Her skin was dry, with poor turgor. Electrocardiogram (EKG) showed sinus tachycardia at a rate of 121/minute, with occasional premature ventricular complexes and ST depressions in anterior leads (new since the last EKG); the PR interval was 130 ms and QTc 479 ms. Chest x-ray showed diffuse emphysema, normal heart size and a right peripherally inserted central venous catheter (PICC) with the tip in the superior vena cava near the right atrium. Left shoulder x-ray was unremarkable. Blood testing showed: potassium 2.7 mmol/L (reference value [RV]: 3.5-5 mmol/L), glucose 153 mg/dL (RV: 80-100 mg/dL), white blood cell count 17,100/cu mm (RV: 4,500-11,000/cu mm) with 62% neutrophils (RV: 40-70%), 19% lymphocytes (RV: 20-50%) and 19% monocytes (RV: 2-12%), hemoglobin 14.9 g/dL (RV: 12-15 g/dL), and platelets 219,000/cu mm (RV: 150,000-400,000/cu mm).

The emergency department (ED) physician recorded weakness as the chief complaint, but focussed on diarrhea in her assessment. The ED physician noted the patient's leukocytosis and stated: "It is unclear what this is coming from, but I suspect it is the diarrhea." The patient was admitted to the oncology service and the admission history and physical examination was performed by a nurse practitioner, who followed the emergency department physician's diagnostic lead. The patient's hypokalemia was corrected with intravenous replacement.

The following day, hospital day 2, the patient's temperature was 38.5 degrees C, heart rate 102/minute, blood pressure 105/70 mm Hg, respirations 18/minute and saturation 97% on oxygen at 2 L/minute. She told a nurse, but no doctor that she felt horrible and complained of severe (8/10) left shoulder pain. She denied nausea or dyspnea. She had had no diarrhea. Chest x-ray showed left lower lobe atelectasis. The general internal medicine service had been consulted to direct the patient's care overall and assessed the patient's problems as "1- Nausea and vomiting; 2- Hypokalemia; 3- Probable reaction to chemotherapy; 4- Lung cancer; 5- Chronic obstructive pulmonary disease; 6- History of nicotine addition; 7- Anxiety; 8- Chronic pain; 9- Steroid dependence; 10- Hypertension; 11- Tendency towards supraventricular tachycardia; 12- Fluid overload; 13- Shoulder pain, could be related to carrying the oxygen; 14- Temperature spike." The general internist focused on the possibility of infection and suggested that prednisone could be masking the symptoms, but he also noted that prednisone itself could have caused the patient's leukocytosis.

An infectious disease specialist had been consulted, and suggested the possibility of catheter-related infection. A gastroenterologist had been consulted, and diagnosed "diarrhea of unknown etiology." A cardiologist had been consulted and he noted that the patient had pain in the left shoulder with tenderness to touch. She did not have any chest pain. EKG showed resolution of the ST segment depressions in anterior leads; QTc was 435 ms. The cardiologist did not diagnose myocardial ischemia or infarction. In the evening, the patient continued to have left shoulder pain, with tenderness to touch, but no chest pain and no dyspnea at rest. The next morning, on hospital day 3, the patient suffered a sudden unexpected cardiac arrest from which she could not be resuscitated.

## AUTOPSY

Postmortem examination revealed 430 mL of partially clotted blood in the pericardium. The lower lateral left ventricle showed a transmural acute myocardial infarction, measuring 6 × 4 cm, approximately 3 days old, with a hemorrhagic tract of rupture. Examination of the coronary arteries demonstrated severe atherosclerosis with thrombosis of the mid left circumflex coronary artery. The lungs had severe emphysema. The left lower lobe had residual adenocarcinoma. There were findings of heart failure (cardiac dilatation, pleural effusions and passive congestion of the liver) and a recently formed thrombus in the right atrium. The liver showed moderate steatohepatitis. The only disease in the gastrointestinal tract was sigmoid colonic diverticulosis.

## DISCUSSION

Numerous factors contributed to the failure to diagnose the acute myocardial infarction in this case. None of the patient's five complaints that prompted

her hospital admission (weakness, fatigue, nausea, vomiting or diarrhea) is suggestive of an acute coronary syndrome and, collectively, they shift focus away from the chest and towards the abdomen. Physical examination and laboratory testing revealed dehydration and potassium depletion, further directing diagnostic thinking toward the abdomen and, specifically, the gastrointestinal tract. The patient's chemotherapy provided a potential explanation for her chief complaints. Pemetrexed is an antifolate agent that inhibits folate-dependent enzymes necessary for the production of some nucleotides; fatigue, nausea and vomiting are among the side effects of pemetrexed. Bevacizumab is an antiangiogenesis agent that interferes with the function of vascular endothelial growth factor; generalized weakness, nausea, vomiting and diarrhea are among the side effects experienced by more than 30% of patients receiving it. The patient was on outpatient opioid therapy, which likely blunted the pain of her myocardial infarction. The patient had tenderness on palpation of her left shoulder, which strongly favors the diagnosis of mechanical injury over myocardial infarction. ST-segment depression is an electrocardiographic hallmark of hypokalemia.[1] The ST-segment depression present on the patient's admission electrocardiogram resolved the following day in association with correction of her hypokalemia, providing solid ground for not regarding the admission ST-segment depression as evidence of myocardial ischemia.

Another important factor in the diagnostic failure in this case was the multiplicity of the patient's diseases. In addition to her severe coronary artery disease, she had severe pulmonary emphysema, residual lung cancer, steatohepatitis and diverticulosis. She had heart failure and the most likely explanation for her nausea, vomiting and diarrhea may be ischemic malfunction of the gastrointestinal tract due to heart failure. Every additional disease a patient has makes it more difficult to correctly diagnose the first one. Perhaps the most important factor of all in this case, however, may have been the patient's own misdiagnosis of her shoulder pain. It was the patient who brought her shoulder pain up as an incidental complaint and provided her own interpretation of the etiology, accepted by the ED physician and all the subsequent diagnosticians. One can easily imagine that a former emergency 9-1-1 telephone operator spoke with authority and told her clinicians what to think in a way similar to what she told her 9-1-1 callers in years past.

The main error in this case appears to be the diagnostician accepting the patient's misdiagnosis at the outset in a way similar to accepting an emergency department triage nurse's misdiagnosis. This could be called 'patient cueing', akin to 'triage cueing' when the triage nurse in the emergency department anchors on a salient feature and the initial label sticks, especially if it has coherence and makes intuitive sense. Of course, we tend to look for meaning and when we find it, then 'search satisficing' can take over and further search is called off. The diagnosis gathers momentum without gathering evidence. Part of it, too, has to do with the 'cognitive miser function' where the brain defaults into the intuitive mode to conserve further effort.[2] Finding a ready and plausible explanation is a disincentive to expending further cognitive effort on searching for other possibilities.

A patient's presentation style can dramatically alter the physician's diagnostic approach. This was demonstrated in a study of 44 internists who were randomized to view videotapes of an actress performing the role of a 40-year-old patient with chest pain in a scripted physician-patient interview, in a businesslike style in one version and in a histrionic style in the other. A cardiac cause was suspected by 50% of the physicians viewing the businesslike portrayal, 93% of whom would have pursued a cardiac workup, while a cardiac cause was suspected by only 13% of those viewing the histrionic portrayal, 53% of whom would have pursued a cardiac workup.[3] The results of this study suggest that patients' presentation style can be a source of diagnostic bias and error.

Increasing age is associated with increasing incidence of missed diagnosis of myocardial infarction.[4] A further source of error in the diagnosis of myocardial infarction in women is that they have fewer classic symptoms and more nonspecific symptoms of it. In an observational study of 481,581 women and 661,932 men with myocardial infarction, 42% of the women presented without chest pain, compared to 30.7% of the men.[5] This poses yet another psychological challenge, specifically for the accurate diagnosis of myocardial infarction.

Diagnostic failures have been shown to contribute to approximately 10% of patient deaths.[6] While system errors are contributory, the majority appear to be

due to various cognitive and affective failures in the diagnostician.[7] The desire to settle on a diagnosis, so as to start therapy and move on to the next patient, can lead to premature closure, accepting a diagnosis as final with insufficient evidence. The aversion to a diagnosis associated with bad outcomes may lead a clinician to favor a misdiagnosis associated with better outcomes. The need to focus one's thinking when a patient has numerous signs and symptoms creates a tendency to lock on to one or two salient features of a patient's presentation and neglect the others, sometimes causing a diagnostician to make a diagnosis too soon, dropping anchor before reaching the safe harbor of the correct diagnosis. The way the diagnostic problem is framed may facilitate making the right diagnosis or it may have the opposite effect. The diagnosis made by the first clinician to see the patient may bias those who follow. Once a diagnosis has been made, with each subsequent clinician following the lead and making the same diagnosis, it gathers momentum without gathering evidence.

The greatest value in recognizing cognitive factors in causing diagnostic errors comes with ways to avoid them. So, how can a diagnostician avoid the psychological pitfall of patient cueing? Essentially, this is most likely to impact intuitive thinking, and Hogarth has advanced a variety of strategies to 'educate intuition', one of which involves imposing a 'circuit-breaker' that routinely establishes screening and censoring processes that interrupt automatic, tacit system processes.[8] The circuit breaker is, therefore, a forcing function that obliges the decision maker to reflect on their thinking and, perhaps, pursue a different course.

A useful first forcing function for avoiding patient cueing to the wrong diagnosis is to quickly and politely interrupt any patient who starts to give their own or another's diagnosis. Instead, the patient can be asked to focus on the specific symptoms that brought them to seek medical care, and the clinician can offer to return later to what the patient or others might believe is the diagnosis. Patients may be exasperated by this 'circuit breaker' but it serves the important purpose of allowing the physician to gather uncontaminated data, and promotes independent thinking. To avoid patient cueing and also remain patient-centered is difficult.[9] It requires physicians to walk such an extremely fine line that avoiding all missteps from this line cannot be reasonably expected; it can only be an ideal to strive for.

A second forcing function is to delay reading other clinician's notes until the patient's presenting complaint and history have been obtained. This will, at least partly, avoid cueing. Colleagues and other members of the team should also be discouraged from offering opinions and 'drive-by' diagnoses.

A third forcing function is aimed at reducing any undue influence of contextual cues by re-framing the information that has been gathered. This enables another of Hogarth's strategies – the re-configuration of the problem in different ways that may allow other options to be generated.

A fourth forcing function is to always ask the question 'What else could this be?' This reflective step allows the clinician to detach from the immediate pull of the situation and consider other possibilities on the differential diagnosis.

Cognitive forcing strategies should help reduce diagnostic errors. A recent study of medical student training, however, failed to show any reduction in diagnostic error in correctly diagnosing computer-based cases after a 90-minute seminar providing instruction about two types of bias leading to diagnostic error.[10] This result could be interpreted as simply demonstrating that the first footstep in a thousand-mile walk does not show statistically significant progress in reaching the destination. The destination of reducing diagnostic error merits a long hard journey.

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