Case Analysis

The Mystery Diagnosis

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Case Presentation: The following scenario is an actual case that, initially, was a mystery to the staff, nurse practitioner, and oncologist. Mrs. Smith, a 72-year-old woman, was finishing her first treatment of cyclophosphamide, doxorubicin, vincristine, and prednisolone plus rituximab for non-Hodgkin lymphoma. She had been dozing during the four hours required for the infusion. Toward the end of the last bag of fluid, her daughter noticed that Mrs. Smith was breathing more slowly than normal. The chemotherapy nurse assessed Mrs. Smith. Her respirations were 10 breaths per minute and a little deeper than usual. Mrs. Smith was awakened, and her respirations increased to 16 breaths per minute. Her oxygen saturation was 88%. Although the saturation was not evaluated immediately prior to treatment, the level was previously documented at 94%. Mrs. Smith denied dyspnea or any other symptoms. Her other vital signs were a blood pressure of 110/70 mmHg and a pulse of 72. The nurse practitioner was summoned to evaluate the patient.

The nurse practitioner ordered oxygen at 2 liters per minute. The oxygen saturation increased to 92%. A review of Mrs. Smith’s history revealed porcine aortic valve replacement at age 55 with moderate restenosis and corresponding aortic valve murmur. As a result, the only medication she was on was warfarin for lifelong anticoagulation. The review of systems and complete physical examination were unchanged. The only positive finding on physical examination was the aortic valve murmur.

The Suspects

Although the primary diagnosis was hypoxia, the practitioner had to identify the cause and proceed with determining the underlying problem. The differential diagnoses included pulmonary embolus (PE), congestive heart failure (CHF), silent myocardial infarction (MI), anemia, sedation- or hypopnea-induced hypoxia, and cardiac arrhythmia.

The following section will examine each of the diagnostic suspects along with the associated symptoms (alibis).

A PE may present with a sudden onset of pain, with or without dyspnea, that may result in hypoxia if a large portion of the alveolar surface is affected. It may be accompanied by dullness on percussion. A PE is associated with deep vein thrombosis (DVT). Electrocardiogram (EKG) changes also may be present (Henke, 2000). In this case, the suspect had an alibi: Mrs. Smith did not have pain or a sudden onset of dyspnea. The physical examination was negative for dullness to percussion over the lung fields. She had no calf tenderness, erythema, or edema to suggest DVT, and the use of warfarin for anticoagulation made it unlikely. An EKG would assist in the differentiation process. However, one was not done at the initial symptom presentation.

The second differential diagnosis and primary suspect, CHF, is characterized by dyspnea with fine rales on auscultation of lungs. CHF also may be accompanied by tachycardia and hypotension, although generally, these are later signs. EKG changes may be seen, but usually after CHF is well established. Clues that suggest reduced cardiac output include narrow pulse pressure, Cheyne-Stokes respirations, resting tachycardia, and/or cool extremities (Hunt et al., 2004; Winkour, 2000). Although Mrs. Smith did not exhibit any of these symptoms, CHF remained the primary suspect.

Hypoxia may be an expression of circulatory compromise caused by a silent MI. Generally, a silent MI may reveal itself with changes in blood pressure, heart rate, and EKG, in addition to symptoms of dyspnea and fatigue (Shelton, 2000). Mrs. Smith experienced none of these changes.

Anemia often presents with diminished oxygen-carrying capacity and may...
be demonstrated with dyspnea on exertion along with hypoxia. Anemia is defined as a hemoglobin less than 12 mg/dl and a hematocrit less than 35 mg/dl (Lynch, 2000; Winkour, 2000). Mrs. Smith’s hemoglobin and hematocrit were borderline low at 11.5 mg/dl and 33 mg/dl. However, the anemia was not believed to be sufficient to cause Mrs. Smith’s level of hypoxia.

Sedation may result in hypopnea because of suppression of the respiratory center. Generally, increasing the respiratory rate and/or reversing the sedation will correct the hypoxia (McDermott, 2000). Even though she had received lorazepam 0.5 mg via IV for pretreatment anxiety, being awakened and having an increased respiratory rate did not result in elevation of Mrs. Smith’s oxygen saturation. Therefore, this suspect also was eliminated.

The last suspect, cardiac arrhythmia, could result in diminished cardiac output and be demonstrated through a variety of symptoms. Hypoxia might result from an untreated arrhythmia; however, it rarely presents as a single symptom (Shelton, 2000). Which of these suspects is the real culprit?

The Plot Thickens

At that point, the intent was to complete a more in-depth examination of Mrs. Smith to help narrow down the suspect list. However, Mrs. Smith proved to be an unamenable victim and halted the investigation by refusing tests that would include an EKG, spiral computed tomography scan, and chest x-ray. Mrs. Smith was tired, lived two hours away from the office, and wanted to go home. By then, the time was almost 5 pm and she was the last patient in the chemotherapy suite. She realized that a workup would require hours of outpatient time or an overnight admission to the hospital. She was not willing to submit to the clinicians’ requests and justified it by saying “I feel fine; I just don’t have much wind when I walk.” The clinicians very reluctantly consented and allowed her to go home with her daughter, provided that she agree to go immediately to the local hospital if she had any symptoms. She acquiesced and was sent home with portable oxygen. A home health referral was arranged. An oxygen concentrator was delivered to her home that evening.

The Culprit Is Caught

Most criminals ultimately are discovered because they reveal themselves in some way. The primary suspect, CHF, proved to be the culprit when Mrs. Smith’s symptoms advanced enough to overpower her reluctance to have a workup. After two days, Mrs. Smith sought care at the local emergency room for progressive dyspnea with rest, tachycardia, and anxiety. On examination, she had presence of fine basilar rales. Diagnostic tests, including a chest x-ray and a B-natriuretic peptide level, confirmed the diagnosis of CHF. Mrs. Smith was treated with IV furosemide with rapid recovery. However, she remained on oxygen because her saturations never returned to baseline.

Reviewing the Method of Operation

CHF, a common culprit, affects nearly five million people, of which 80% are aged 65 or older (Mathew, Gottdiener, Kitzman, Aurigemma, & Gardin, 2004). The clinical features include orthopnea, dyspnea on exertion, paroxysmal nocturnal dyspnea, altered mental status, lower-extremity edema, decreased exercise tolerance, and distended abdomen. Physical findings that reveal compromise include elevated jugular venous distention, presence of an S1, rales that do not clear with cough, peripheral edema, weight gain of three to four pounds in one to three days, hepatojugular reflux, and displaced left ventricular apical impulse. Laboratory tests used to confirm the diagnosis of CHF include complete blood count, metabolic profile, and B-natriuretic peptide. Additional studies in the investigation include a chest x-ray, EKG, echocardiogram, and, possibly, a cardiac catheterization (Vesley, 2004; Winkour, 2000).

Keeping the Culprit Under Control

After conviction, many offenders are monitored by a probation officer. The probation officer’s duty is to keep tabs on the status and whereabouts of the offender. Likewise, CHF control requires monitoring treatment and the potential return of symptoms, both by the healthcare provider (probation officer) and the patient (victim). The mainstay of treatment for CHF is aimed at control of causative factors. Medications such as diuretics reduce volume overload. Angiotensin-converting enzyme inhibitors reduce systemic resistance and blood pressure that results in increased cardiac output and improved exercise tolerance (Winkour, 2000). One angiotensin receptor blocker, valsartan, has an indication for CHF treatment (Jacobs & Ramsey, 2005). Carvedilol is a mixed alpha 1 and beta antagonist used to improve functional status and increase long-term survival. Studies have shown that the use of beta blockers may decrease mortality rates associated with CHF. Eplerenone, a selective aldosterone antagonist, was approved in 2002 for the treatment of CHF following MI (Oestreng, Pitcock, & Pitcock, 2005; Riggs, 2004). Other medications that may play a role in supervising the CHF criminal are arterial or venous vasodilators (e.g., nitrates) and inotropic agents (e.g., digoxin). Surgical intervention, such as revascularization or valve replacement, also may be indicated to treat the cause of CHF. Early intervention can do much to improve long-term survival (Vesley, 2004).

Analysis of the Clues

In Mrs. Smith’s instance, the normal alibis (symptoms) were absent, and little evidence was available on which to base the case. Plenty of suspicion did not get the clinicians very far. One clue that the clinicians did recognize was Mrs. Smith’s history of aortic valve replacement with moderate restenosis, which put her at risk for CHF. The reason this suspect was released (CHF was dismissed as the cause) was the lack of evidence on physical examination, such as a lack of rales or other associated findings. Because Mrs. Smith would not allow a workup, a B-natriuretic peptide test, which is indicative of CHF, was not completed. The clinicians simply had to rely on the inconclusive evidence at hand. Given her aortic valve disease, they underestimated the narrow circulating fluid level that Mrs. Smith could tolerate. The American College of Cardiology reported that the majority of patients with clinical evidence of volume overload do not exhibit hypoperfusion (Hunt et al., 2004). Analysis of Mrs. Smith’s chemotherapy treatment
revealed that she had been given approximately 900 cc of fluid, an amount most patients should tolerate. In subsequent treatments, the dose was concentrated in half of the amount of fluid, administered over the same amount of time, and was tolerated without further episodes of CHF. Mrs. Smith completed a total of six courses of chemotherapy with no evidence of disease. She has improved with the addition of medication for chronic CHF. She only requires oxygen at night. The mystery case was solved.

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References


