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An Unresponsive Patient in Postanesthesia Care Unit: A Case Report of an Unusual Diagnosis for a Common Problem

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An unresponsive patient in the postoperative period is a serious complication that can be caused by anesthetics. However, nonanesthetic causes should also be considered. In this case report, we present an unresponsive postoperative patient diagnosed with possible psychosomatic catatonia. We further describe a systematic approach to the unresponsive patient in the postanesthesia care unit (PACU). While not an uncommon occurrence, catatonia is a complex psychomotor syndrome that can be difficult to diagnose; however, catatonia should be considered in unresponsive postoperative patients. (A&A Practice. 2020;14:e01293.)

GLOSSARY

BMI = body mass index; **BMP** = basic metabolic panel; **CAS** = central anticholinergic syndrome; **CT** = computed tomography; **EEG** = electroencephalogram; **MRI** = magnetic resonance imaging; **NMS** = neuroleptic malignant syndrome; **PACU** = postanesthesia care unit; **POC** = point-of-care; **TSH** = thyroid-stimulating hormone

Unresponsiveness in a patient in the immediate postoperative period can be a life-threatening condition that must be urgently addressed. Anesthetic-related causes (eg, residual anesthetics, opioid overdose, and residual neuromuscular blockade) are the most common causes and should be considered first, while maintaining airway patency, breathing, and circulation. Thereafter, the unresponsive patient should be assessed via differential diagnoses delineated by organ systems.[1–4] In this case report, we present an unresponsive patient diagnosed with possible psychosomatic catatonia. We further describe a systematic approach to the unresponsive patient in the postanesthesia care unit (PACU). We hope that this case report can serve as an educational guide in assessing an unresponsive patient in the PACU. A Health Insurance Portability and Accountability Act authorization has been obtained from the patient.

CASE DESCRIPTION

This case report presents a 76-kg 43-year-old female patient with a history of left breast high-grade ductal carcinoma in situ, who was scheduled for tumor mass resection and sentinel lymph node biopsy. She was physically active, had a body mass index (BMI) of 28 kg/m^2, did not smoke or consume recreational drugs or alcohol, took 25 mg of hydroxyzine twice daily for seasonal allergies, and 600 mg of gabapentin 3 times daily for unilateral carpal tunnel syndrome. The patient denied other allergies. A chart review showed that multiple providers had noted that the patient was under significant stress due to her diagnosis of breast cancer.

Preoperatively, the patient was notably anxious and was given 2 mg of midazolam intravenously. Intraoperatively, she received 50 μg of fentanyl immediately before intubation and 0.6 mg of hydromorphone before incision. Surgery lasted 3 hours. Before extubation, while spontaneously breathing at 18 breaths per minute, the patient was administered 0.4 mg of hydromorphone and 4 mg of ondansetron. Immediately after extubation, the patient was able to open her eyes on command and make purposeful movements. However, on arrival to the PACU, the patient digressed to a nonresponsive state, was nonverbal, unable to follow commands, and unresponsive to noxious stimuli in all extremities, with a Glasgow Coma Scale of 3. Vital signs remained stable, with a temperature of 36.9°C, normal sinus rhythm, heart rate of 70 beats per minute, blood pressure of 112/65 mm Hg, respiratory rate of 18 breaths per minute, and oxygen (O_2) saturation at 99% on room air. A point-of-care (POC) glucose was 95 mg/dL. A bispectral index monitor was placed on the patient's forehead, which read 93–97, with a 97 signal quality index.

A neurology consultation was requested while preparing the patient for transport for an urgent noncontrast computed tomography (CT) scan of the head. On examination, pupils were equally round at 3 mm and reactive to light with a conjugate gaze. The patient's face appeared symmetric, with no facial droop or jaw stiffness. Normal muscle

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bulk and tone, with no rigidity or tremor, were noted. Deep tendon reflexes were 2+ throughout with absent Babinski reflex and clonus. A noncontrast CT of the head was negative for cerebral hemorrhage. A CT-perfusion scan was also performed and was negative for any large-vessel occlusions.

The patient's condition was deemed inconsistent with a cerebrovascular event or a seizure episode. Additional workup in the PACU included the following laboratory results: arterial pH 7.38, O_2 partial pressure 100 mm Hg on room air, carbon dioxide (CO_2) partial pressure 39 mm Hg, troponin .001 ng/mL, white blood count of 9.6 K/cumm, hemoglobin 13.4 g/dL, and platelet 291 K/cumm. Basic metabolic panel (BMP) was unremarkable, with sodium of 137 mmol/L, potassium 3.9 mmol/L, chloride 103 mmol/L, CO_2 29 mmol/L, blood urea nitrogen 7 mg/dL, creatinine 0.67 mg/dL, and calcium of 9.1 mg/dL. Thyroid-stimulating hormone (TSH) was 0.925 (normal 0.5–5). A serum toxicology panel was negative for amphetamines, cannabis, cocaine, opiates, ethanol, and phencyclidine. Urinalysis was negative for leukocytes, nitrites, ketones, glucose, and blood. Screenings for human immunodeficiency virus antibody, *Chlamydia trachomatis*, *Neisseria gonorrheae*, and rapid plasma reagin for syphilis were also negative.

Two hours after arrival to the PACU, with her eyes and mouth open, the patient started making incomprehensible sounds, while withdrawing from noxious stimuli. Thirty minutes later, some ability for the patient to follow commands returned, including squeezing her hands and moving her toes. However, she would not close her eyes or verbalize on command. The patient was admitted to the neurology service for further workup. She gradually returned to baseline within 6 hours after extubation. A complete physical examination was normal, with no signs of nuchal rigidity and normal neurologic examination. Further evaluation during admission included a 21-channel electroencephalogram (EEG) recording, which showed no epileptiform discharges. CT angiograms of the head and neck were negative for any vascular abnormalities. Noncontrast and intravenous contrast magnetic resonance imaging (MRI) of the head were also negative.

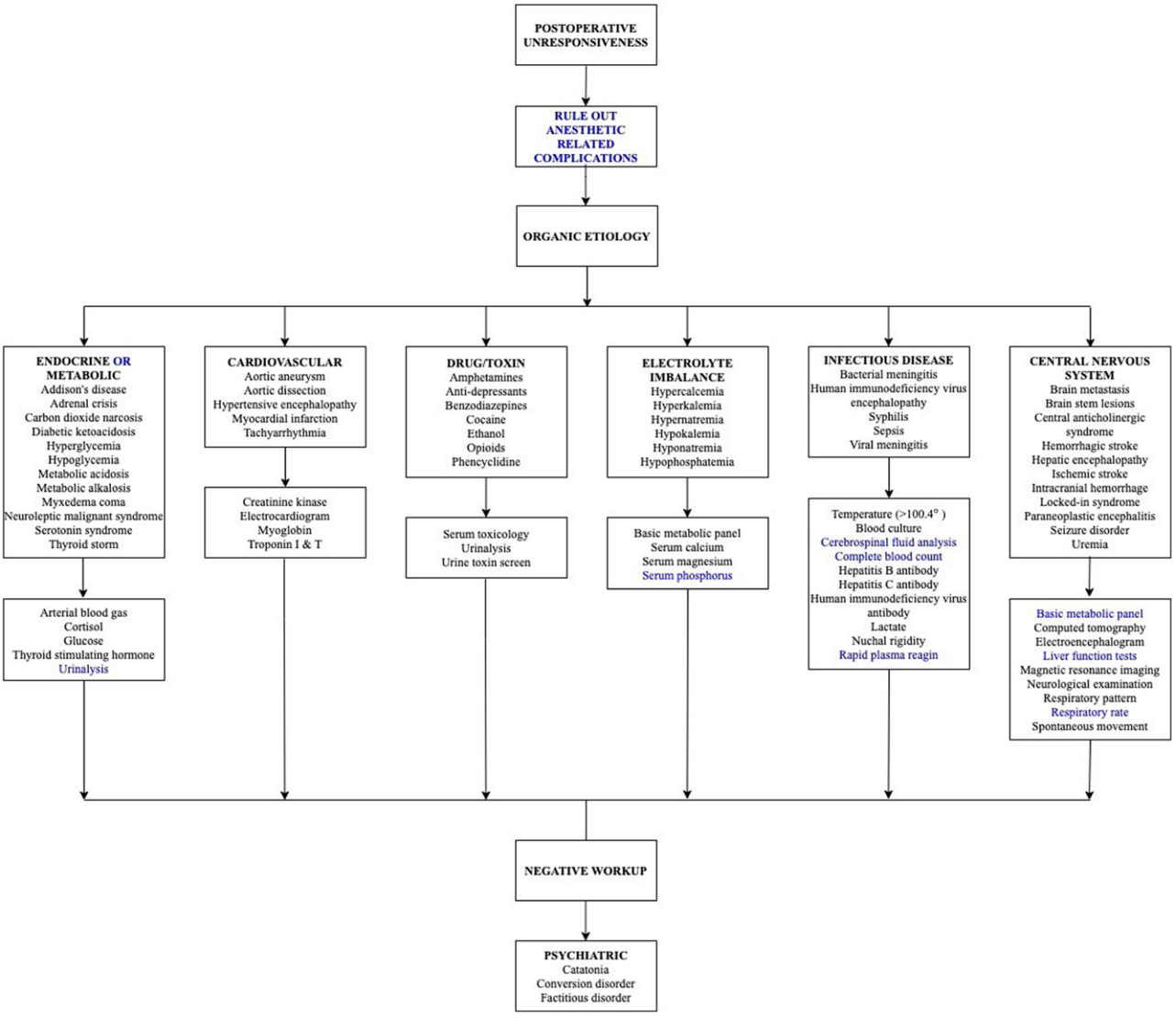

Figure. A systematic approach to an unresponsive patient in the postoperative period.

The negative workup, the patient's anxious affect, and her significant distress with respect to her breast cancer diagnosis rendered our diagnosis for her postoperative unresponsiveness as possibly psychosomatic. Once the patient was fully awake, the last thing she remembered was the immediate preoperative period. She also stated that another similar episode had occurred postoperatively when she had undergone a breast biopsy 6 months earlier. This previous incident had not been reported during the preoperative evaluation.

DISCUSSION

Catatonia is a multifaceted, complex psychomotor syndrome that can present as either a depressed or excited state. Depressed catatonia is more common, with immobility and mutism the most common presenting symptoms, which can lead to significant complications, from pressure ulcers to deep venous thromboses. Excited catatonia presents with immense psychomotor agitation that can lead to autonomic dysfunction.[5] Although an uncommon occurrence, anesthesiologists should consider catatonia as part of the differential diagnosis for unresponsive patients in the PACU or the intensive care unit.

During evaluation of the postoperative unresponsive patient, anesthetic-related complications, such as narcotic overdose, residual anesthetic, or residual neuromuscular blockade, must first be ruled out. In this case, normal ventilation, nonpinpoint reactive pupils, and bispectral index monitoring were consistent with a fully awake patient. Central anticholinergic syndrome (CAS) was also considered because the patient was on hydroxyzine, and histamine receptor type 1 and 2 blocking agents have been implicated in CAS. The CAS can present with altered mental status, stupor, and even coma.[6] However, because our patient did not receive any additional anticholinergic medications, CAS was less likely.

In addition, in the unresponsive patient, cerebrovascular accident, cardiac and pulmonary etiologies, metabolic or endocrine imbalance, seizures, urinary tract infection, toxin ingestion, and infection should also be considered (Figure). In this case, cerebrovascular etiologies, including hemorrhagic and ischemic strokes, had been ruled out with CT and MRI. Arterial blood gases revealed no evidence of hypoxemic or hypercarbic respiratory failure.

Not uncommonly, metabolic derangements, including myxedema coma, neuroleptic malignant syndrome (NMS), and serotonin syndrome, can also lead to acute changes in mental status, especially considering the common use of psychiatric medications implicated in serotonin syndrome.[7] The NMS typically presents with tachycardia, hyperthermia, rigidity, and hypoactive bowel sounds. However, serotonin syndrome can have a similar presentation, with hyperactive reflexes and hyperactive bowel sounds.[7] Myxedema coma is perhaps the most common of the above and was ruled out with a normal TSH.[8] Other common metabolic or electrolyte abnormalities within our differential included hyponatremia and hypoglycemia, which were excluded by obtaining a POC glucose and BMP.[9] Urinary tract infection, which commonly causes altered mental status in the elderly, and toxin ingestion were ruled out with normal urinalysis and a negative urine toxicology panel.[10]

Given the patient's normal EEG, a seizure episode with a prolonged postictal state was also ruled out. Although an early EEG may have ruled out a nonconvulsive epileptic state, it was not immediately available at our institution. Considering the patient's history of breast cancer, other neurologic etiologies of her catatonia, such as paraneoplastic encephalitis and brain metastasis, were also included in the differential. Although these conditions are unlikely to present acutely, they were nonetheless ruled out with a normal MRI.[11,12]

Psychogenic seizures, conversion disorder, or stress-induced catatonia may result in postoperative unresponsiveness and are similar in presentation. Conversion disorder displays symptoms affecting motor and sensory functions for which patients are not purposefully producing their symptoms. In contrast, with factitious disorder, the patients are cognizant of their actions.[2,13–15] Although rare, psychogenic seizures resemble an epileptic event. However, these lack the abnormal electrical impulses found on EEG.

The many areas of overlap among the above differential diagnoses for the unresponsive postoperative patient can make a diagnosis challenging. One could argue that the patient's episode of unresponsiveness was somatization. Nonetheless, this unusual case exemplifies a common problem concerning unresponsiveness in the immediate postoperative period, when life-threatening conditions had to be quickly ruled out. It is useful for anesthesiologists to remember that unresponsiveness is not always related to anesthesia. The patient was discharged from the neurology service without psychological evaluation and treatment. However, we recommend that cancer patients who exhibit such postoperative unresponsiveness be referred to cancer care psychological services to help them cope with the stress of their diagnosis. We further advise judicious use of anxiolytics in the perioperative period as prophylaxis against such episodes.

DISCLOSURES

Name: Sassan Rafizadeh, MD, PhD.
Contribution: This author participated in the patient's care and literature review, and drafted and edited the article.
Name: Ariel R. Kerry-Gnazzo, DO.
Contribution: This author helped review the literature and draft the article.
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Contribution: This author oversaw the patient's care and revised the article.
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