MORNING REPORT: PART II

A FALL AND A CULTURE OF CONFUSION

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A 71-year-old male with a past history that included throat cancer, end stage renal disease (ESRD), coronary artery disease (CAD) with congestive heart failure (CHF), and rheumatoid arthritis (RA) was transferred from the intensive care unit (ICU) to our general medical service overnight. While in the ICU, he was treated for acute respiratory failure secondary to pneumonia without requiring ventilator support. On the night of transfer, the patient had suffered an unwitnessed fall. On our initial evaluation the patient was responsive to painful stimuli and unable to establish orientation, a new finding since admission. Given the alteration in sensorium, CT brain without contrast was performed revealing a right frontal subdural hematoma measuring 3mm with no midline shift.

In caring for a complex inpatient, several lines of query often need to be addressed simultaneously. In the foreground, currently we need to consider the etiology of the hematoma, and whether it needs surgical evacuation. Factors that may contribute to intracerebral bleeding in this patient include platelet dysfunction secondary to renal disease and blood thinning medications given his history of CAD. Urgent or emergent surgical evacuation of a SDH is recommended if the clot thickness exceeds 10 mm or the midline shift is greater than 5 mm, regardless of the neurologic condition.[1]

Labs revealed a thrombocytopenia with platelet count of 117 K/µL. His antiplatelet medications were discontinued. His platelet count dropped to 70 K/µL requiring multiple transfusions to maintain counts above 100 K/µL. His other labs were as follows: Na 144, K 5.4, Cl 97, CO_2 28, BUN 24, Cr 4.85, Ca 9.5, and Glucose 111. The SDH was monitored daily and managed conservatively with platelet transfusions.

The background question now is the etiology of the delirium. Incidence of delirium in medical inpatients is reported between 3%-29% and may be missed in up to 66% of patients.[2] *Elderly patients are prone to both falls and delirium. In this patient, correctable factors such as hypoglycemia and electrolyte abnormalities were ruled out. Although SDH would be easy to anchor on, it is important to thoroughly investigate other causes. A recent ICU stay can add to an acute confusional state, as can concurrent infection. A review of the history of present illness would lead to the next decision point.*

A review of the chart revealed that the patient presented to the emergency department with 2 days of shortness of breath and a cough productive of brown sputum. He was admitted at another hospital 3 weeks prior for pneumonia and was treated with a 10 day course of an unknown antibiotic. He also reported 2 weeks of watery diarrhea. He denied any abdominal pain, chest pain, hemoptysis, or leg swelling. His medications included prednisone and plaquenil and oral vancomycin for presumed C difficile infection. He was an ex-smoker with no history of alcohol or recreational drug use. He lived in a senior building with his wife and was retired from a career in the steel mill.

In the ED, the patient had a blood pressure of 149/90, pulse of 109, respiratory rate of 28, saturation of 89% on room air, and was afebrile. His respiratory exam exhibited no increased work of breathing but did reveal oral thrush, reduced air exchange, and coarse rhonchi in the upper lung fields. His initial labs were notable for a mildly elevated troponin (0.19) and potassium (5.5) with acidosis and lactate of 3.3. Records obtained from his prior admission revealed chronically elevated troponin (0.18-0.28). Electrocardiogram revealed left ventricular hypertrophy with no evidence of acute ischemia. A chest X-ray showing patchy ground-glass opacities involving both lung bases with small pleural effusions. He was admitted to intensive care and started on vancomycin, metronidazole, and cefepime as empiric therapy.

Defined as two episodes of pneumonia in one year or 3 episodes over any time frame, etiologies of recurrent pneumonia can include anatomical abnormalities, immunodeficiencies, aspiration syndromes, other infections, and abnormal mucous clearance. Our patient was noted to be on immunosuppressants. Next steps to elucidate etiology would include imaging to evaluate for anatomical abnormalities such as abscess formation, and

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cultures to evaluate for secondary or untreated infections. In the meantime, guidelines[3] recommend using clinical criteria to start empiric therapy for the management of hospital acquired pneumonia, based on local antibiogram. The additional use of procalcitonin or C-reactive protein is not recommended. ESRD portends an elevated cardiac risk, and although his troponin levels have not been alarming, further cardiac imaging may help to lower cardiac causes on the differential.

A CT scan demonstrated multilobulated nodular mass-like consolidative opacities in both lower lobes, trace bilateral pleural effusions, and mildly enlarged right prevascular and subcarinal lymph nodes. A transthoracic echocardiogram revealed normal cavity size, wall thickness, systolic function, and ejection fraction. The mitral valve annulus and leaflets were mildly calcified with no vegetation. There was moderate aortic valve thickening, consistent with sclerosis.

His history of throat cancer brings up the concern for recurrent, or a de novo, malignancy. Given immunosuppression, an atypical or opportunistic pneumonia should also be explored with both noninvasive and invasive testing to establish an accurate diagnosis. Bronchoscopy with endoscopic sampling would be the natural next step. However in patients with traumatic brain injury, caution is advised due to risk of raising intracranial pressure, albeit transiently. Finally, a careful review of his medications may reveal other causative agents for delirium.

Sputum cultures and galactomannan antigen were sent. Bronchoscopy was delayed due to continuing alteration in sensorium and poorly controlled blood pressure. A review of medications raised concern for possible cefepime neurotoxicity, and this was switched to piperacillin-tazobactam. An MRI of the brain was pursued to rule out recurrence of malignancy causing altered mental status and results showed no mass or midline shift, mild-to-moderate white matter ischemic disease, and no enhancing lesions. The small cerebral convexity subdural hemorrhage had not changed.

Cefepime-induced neurotoxicity[4] most commonly presents with altered mental status, with reduced consciousness (47%), myoclonus (42%), and confusion (42%). Electroencephalography abnormalities are nonspecific, including non-convulsive status epilepticus (25%), myoclonic status epilepticus (7%), triphasic waves (40%), and focal sharp waves (39%). This adverse reaction can occur despite appropriate dosing, usually resolves with drug interruption, but may require additional interventions such as antiepileptic drug administration or dialysis.

The patient's mentation improved by day 3 and he underwent endobronchial ultrasound (EBUS), revealing thick secretions throughout the entire bronchial tree. Preliminary studies were positive for parainfluenza virus. There was no evidence of malignancy. Microbiology reported acid fast branching filamentous rods with beaded appearance on modified Ziehl-Neelsen staining, suggesting nocardia infection. The antibiotic regimen was narrowed to renally-dosed intravenous trimethoprim-sulfamethoxazole (TMX) and amikacin. The patient's mentation improved significantly and he was transferred to acute rehabilitation with an anticipated 6-month antibiotic regimen.

Conclusion

Nocardia is a pathogen in immunocompromised patients. Diagnostic accuracy is paramount since resistance to common antibiotics is high, and duration of treatment is prolonged.[5] Our patient's immunocompromised state put him at risk for opportunistic infections and his complex history put us on a tortuous path to his final diagnosis—Nocardia cyriacigeorgica pneumonia. Although there was no evidence for disseminated nocardiosis, he required long term antibiotics with close follow-up.

When faced with a patient with acute delirium, a thorough review of the history, and detailed evaluation of abnormalities found, is necessary in accomplishing an accurate diagnosis.

References

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