CLINICAL COMMENTARY

"Captain of the Ship"

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An 81-year-old man presented to the emergency room after sudden onset of severe generalized weakness while shopping. He was hypotensive in the field at 72/48 mm Hg. In the emergency room, his blood pressure improved to 113/45 mm Hg. A few days prior, he had seen his primary care physician for low blood pressure and was advised to stop his antihypertensive medicines, however, continued to take them. His son noted his father's eyes were increasingly yellow. Labs included a white blood cell count [WBC] of 13,500 per microliter, hemoglobin 10.9 g/dL, blood urea nitrogen 32 mg/dL, creatinine 1.76 mg/dL, total bilirubin [T. bili.] 17.5 g/dL, aspartate aminotransferase [AST] 203 units/L, alanine aminotransferase [ALT] 294 units/L, alkaline phosphatase [ALP] 835 units/L and albumin 3.7 gm/dL. Computed tomogram (CT) scan of the abdomen and pelvis with intravenous (IV) contrast was limited due to motion artifact. Focal posterior atelectasis/infiltrate in left lung base was noted and a 1.4 cm subpleural nodule in the right lower lobe. Records were reviewed from an admission five weeks prior for generalized weakness and bradycardia. Second degree, Mobitz type 2 heart block was diagnosed, and a permanent pacemaker implanted. During admission, elevated transaminases were noted including: T. bili, 1.5 g/dL, AST 186 units/L, ALT 305 units/L, and ALP 520 units/L. Abdominal ultrasound revealed non-specific liver heterogeneity and gallbladder sludge without stone or focal tenderness. Gastroenterology felt the liver abnormality was due to congestive hepatopathy from his cardiac disease and advised outpatient follow up. Liver ultrasound revealed gallbladder sludge with possible gallbladder wall thickening. Nuclear medicine hepatobiliary scan demonstrated non-visualization of the gallbladder and delayed biliary to bowel transit up to 24 hours post tracer administration. This was thought to be related to markedly impaired liver function and cholestasis. Gastroenterology attributed his presentation to possible shock liver from hypotension, versus drug toxicity from the cefazolin he received during pacemaker insertion five weeks earlier or possible congestive hepatopathy. They recommended discharge given hemodynamic stability, without obvious biliary obstruction, with follow up including lab evaluation in two to four weeks. Magnetic resonance cholangiopancreatography [MRCP] was not felt to be essential on this admission. Our clinical suspicion prompted consideration of scanning. After discussion with the patient and his son, MRCP was scheduled. The procedure was delayed four days due to the need for pacemaker deactivation. MRCP revealed a poorly displayed common biliary duct [CBD] to pancreatic duct junction with a 3 mm hypodensity in the distal common bile duct with proximally adjacent intermediate intensity material. This was thought to represent choledocholithiasis and proximal sludge. The adjacent pancreatic duct was patent and appeared normal. Endoscopic retrograde cholangiopancreatography [ERCP] cleared blood clots from the distal CBD. The "irregular" mid and proximal CBD was biopsied and brushed and a biliary stent placed. Surgical consultation was obtained due to haemobilia. In absence of iatrogenic trauma, malignancy was the primary concern. CT liver pancreatic protocol did not provide additional information. Pathology of the lesion revealed moderately differentiated adenocarcinoma. CT chest with IV contrast was significant for segmental pulmonary emboli in his right upper, middle, and lower lobes. Venous doppler ultrasound revealed bilateral peroneal deep venous thrombosis. Two-dimensional echocardiogram did not show right ventricular strain and he was started on apixaban. Due to persistently elevated bilirubin, interventional radiology was consulted. His common hepatic artery was completely occluded by tumor and the internal-external drain could not be placed. An external drain was placed in his left hepatic biliary duct and follow up ERCP revealed biliary tract obstruction from a mass completely obstructing the left hepatic duct. The prior stent was removed, and a new stent was placed in the deep right intrahepatic duct with distal drainage in the duodenum. Hepatobiliary surgery and medical oncology were consulted and recommended transfer for higher level of care. Unfortunately, his condition progressed with recurrent hyperbilirubinemia and a second sub-massive pulmonary embolus despite anticoagulation. A second biliary stent was placed but quickly obstructed. Oncology felt that radiation was not an option as his tumor was poorly visualized on multiple scans. Oncology also felt chemotherapy would provide less than twenty percent likelihood of success. Palliative care consulted, consistent with patient and family wishes, he entered hospice and died two weeks later.

Discussion

This case illustrates important issues in medical practice. The patient was discharged despite a dramatic rise in bilirubin from 1.5 g/dL to 17.5 g/dL. The differential diagnosis at discharge included congestive hepatopathy, shock liver or drug induced liver injury from cephalosporin exposure. In hindsight, the discharge recommendation with outpatient follow-up in 2-4 weeks was not appropriate. Given his recently placed pacemaker, the MRCP would be delayed at a community hospital due to need for a company technician to temporarily deactivate the device. With the ever-increasing costs and attempts to

minimize hospital length of stay, the consultant documented "clearance" for discharge, despite clinical complexity. The primary hospitalists may be perceived as coordinators of care rather than as "the captain of the ship" and the primary patient advocates. Discharging this patient would have been a grave mistake given anticipated delays in outpatient medical care. He had a life-threatening condition. Delays in definitive diagnosis may not have changed the outcome but premature discharge was wrong. Additional hospitalization has detrimental effect on length of stay (LOS) metrics, but certainly prevented clinical deterioration at home, and emergency readmission with associated stressors and profound discomfort. As physician healers, we need to place patients and family needs first.

The progressive erosion of physician autonomy has been surreptitious, though more recently quite overt. Societal changes over decades have helped accelerate the loss of autonomy.

These include the corporatization of healthcare with expanded nonclinical bureaucracy.[1] Second, dilution of physician services by alternate medical providers like nurse practitioners and clinical pharmacists may have further undermined clinical autonomy.[2] The explosion of information technology and the electronic medical record has taken us further from the bedside with less time for developing rapport and trust.[3] Direct to consumer advertising by pharmaceutical companies create conflicts over clinical decision making that may erode the physician patient relationship, creating potential additional conflict.[4]

As hospitalists we need to place our patients' interests first, and we need to be wary of exploitation of our competitive nature. Increasing numbers of "quality metrics" favor lower lengths of stay with cost containment goals. For-profit healthcare companies may use these "savings" to provide higher returns to their investors rather than better patient care.[5]

We need to be extra vigilant in focusing on patients as individuals and resist the temptation to go against our clinical intuition when non-clinical forces push us to alter our decision making. We hope that this case provides our colleagues an understanding of conflicts in the current healthcare climate. Our goal is to create a more balanced view of clinical decision making and keeping the patients' best interests as our primary mission.

REFERENCES

1. **McKinlay JB, Marceau LD**. The end of the golden age of doctoring. *Int J Health Serv*. 2002;32(2):379-416. doi: 10.2190/JL1D-21BG-PK2N-J0KD. PMID: 12067037.
2. **Tracy C, Russell NG, DePriest K, Knestrick J, D'Aoust R, Slade E**. The impact of full practice authority on nurse practitioner compensation, collaboration, and billing. *J Am Assoc Nurse Pract*. 2023 Nov 1;35(11):691-698. doi: 10.1097/JXX.0000000000000918. PMID: 37602876.
3. **Chi J, Artandi M, Kugler J, Ozdalga E, Hosamani P, Koehler E, Osterberg L, Zaman J, Thadaney S, Elder A, Verghese A**. The Five-Minute Moment. *Am J Med*. 2016 Aug;129(8):792-5. doi: 10.1016/j.amjmed.2016.02.020. Epub 2016 Mar 10. PMID: 26972793.
4. **Findlay SD**. Direct-to-consumer promotion of prescription drugs. Economic implications for patients, payers and providers. *Pharmacoeconomics*. 2001;19(2):109-19. doi: 10.2165/00019053-200119020-00001. PMID: 11284378.
5. **Sievers MT, Neevel A, Diaz A, Rouanet E, Sheetz K, Brophy D, Dimick JB, Chhabra KR**. Private Equity Investment in Surgical Care. *Ann Surg*. 2024 Feb 19. doi: 10.1097/SLA.0000000000006238. Epub ahead of print. PMID: 38372276.