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Hospitalist Update

Hospital Readmissions

Kristin Hahn-Cover, MD, FACP

Hospital readmissions are under scrutiny in the context of health care delivery and payment reform. Hospital readmission rates for Medicare (CMS) patients with principal diagnoses of heart attack, heart failure and pneumonia are published to the consumer-focused Hospital Compare site as an "outcome of care" for hospitals participating in the Inpatient Quality Reporting program [1]. The CMS Hospital Readmissions Reduction Program will first affect Medicare payment in federal fiscal year 2013. In year one, the focus is 30-day readmissions of patients with heart attack, heart failure and pneumonia. Hospitals with higher-than-predicted readmission rates between July 1, 2008, and June 30, 2011, will be subject to as much as a 1% payment reduction (1% reduction in base operating DRG payment amount for Medicare Fee for Service patients aged 65 or older) in October, 2012. In subsequent years, more diagnoses will be added and the payment reductions increase (up to 2% in 2014 and 3% in 2015). Hospital readmissions also appear as a quality performance standard in the Medicare Shared Savings Program final rule on Accountable Care Organizations. Additionally, in some bundled payment models outlined by the Center for Medicare & Medicaid Innovation, participating hospitals would not receive payment beyond the predetermined "bundled payment" for patients with related readmissions within 30 days.

What is the evidence that hospital readmissions are preventable or that readmission rates reflect the quality of a hospital's care? In an analysis of 2005 Medicare data, 76% of 30-day readmissions were deemed to be potentially preventable. The greatest variation amongst hospitals in readmission rates was for patients with heart failure, COPD and pneumonia [2]. Some of this variability is accounted for by patient characteristics used to calculate the hospital's "risk standardized readmissions measure" but the assumption is that variability outside of patient characteristics reflects the quality of care provided by that hospital. In a systemic review by van Walraven et al., the median proportion of potentially avoidable hospital readmissions was 27% [3].

Precisely what the hospital must do to optimize quality and thereby influence readmission rates is unclear, however. In a review by Hansen et al., no single intervention during hospitalization clearly reduced 30-day readmissions [4]. All elective "bundles" of interventions included patient-centered discharge instructions and post-discharge telephone calls. Interestingly, a recent article by Epstein et al., demonstrated regional variations in hospital readmission rates as having the single largest impact on hospital readmission rates (larger than case mix or discharge planning). [5]

Variability in readmission rates may reflect how well the hospital manages the continuum of the patient's care rather than the quality of care provided in the inpatient setting. In a study by Misky et al., patients with the same medical condition who had follow-up with a primary care provider within 4 weeks of discharge had a 30 -day readmission rate of 3.1% compared to 21.3% for those who did not receive such follow-up. [6] A similar relationship was demonstrated by Hernandez et al. in patients with heart failure; this study evaluated the impact of a follow-up within 7 days on all-cause readmissions within 30 days. [7].

Patient characteristics do influence readmission rates. A number of studies have examined patient characteristics which correlate with readmission rates, demonstrating positive relationships between readmission and low socioeconomic status, increasing age, prior hospitalization and a higher burden of comorbidities, including depression [8]. Another approach to reducing readmission rates would be to use these characteristics to stratify interventions at the individual patient level.

What is a hospitalist to do with this information? At the patient level, we must recognize those who are at an increased risk for readmission. We must then look for ways to mitigate this risk by ensuring timely post-hospital appointments and by providing patient-centered discharge information (especially regarding their discharge medications). Hospitalists engaged in performance improvement for their organization should be aware that hospital readmissions are an area of focus in a number of health care reform programs. Finally, we must stay abreast of literature defining key patient characteristics and key hospital interventions that influence potentially preventable readmission risk.

REFERENCES:

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3. Van Walvaren, C et al., *Proportion of hospital readmissions deemed avoidable: a systemic review*. CMAJ 2011; 183(7): E391-402
4. Hansen, LO et al., *Interventions to reduce 30-day rehospitalization; a systemic review.* Ann Intern Med 2011; 155: 520-528
5. Epstein, AM et al., *The relationship between hospital admission rates and rehospitalizations.* NEJM 2011; 365: 2287-2295
6. Misky, GJ et al., *Post-hospitalization transitions: examining the effects of timing of primary care provider follow-up.* Journal of Hospital Medicine 2010; 5: 392-397
7. Hernandez, AF et al., *Relationship between early physician follow-up and 30-day readmission among Medicare beneficiaries hospitalized for heart failure*. JAMA 2010; 303(17): 1716-1722
8. Mitchell, SE et al., *Post-discharge hospital utilization among adult medical inpatients with depressive symptoms.* Journal of Hospital Medicine 2010; 5: 378-384

CASE REPORT Robert Folzenlogen MD

SHEEHAN'S SYNDROME

CASE: A 61 year old female was referred to University Hospital for evaluation due to altered mental status. The patient's symptoms, which had increased over the preceding 12 hours, were characterized as lethargy and decreasing responsiveness. There was no report of fever, chills, recent URI, UTI symptoms, recent nausea or vomiting, diarrhea or headache. Neither were there any reports or evidence of tick or insect bites. Some melena had been noted at the referring facility but this was not corroborated with an FOBT.

Her PMH was remarkable for a history of urinary incontinence and an episode of post-partum hemorrhage; she had since undergone a hysterectomy. The patient was a nonsmoker and did not use alcohol or illicit drugs. Of note, approximately 1 year ago, she was treated for a similar episode of altered mental status, associated with a headache; though hyponatremia was noted, the remainder of her workup, including an LP, had been unremarkable. Current medications were limited to oxybutynin and estrogen supplementation. Her family history was significant for breast cancer.

On initial exam, she was noted to be very lethargic and was unable to provide any reliable history (the above data was provided by her husband). Her T was 35 C, P 65, R 14, BP 128/71, O2 sat 98% on 4L of oxygen. She was in no apparent distress. Her head and neck exam was normal; no thyromegaly or adenopathy was found. Chest was clear and cardiovascular exam was WNL. Abdomen was soft and nontender with no organomegaly. Her neurologic exam did not reveal any focal neurologic deficits; diminished DTRs were noted.

An ABG revealed a pH of 7.4, pCO2 46, PO2 67, HCO3 28 and O2 sat of 94.5% on 4L of oxygen. Other initial labs included WBC 5.3, Hgb 10.9, MCV 94.6, Platelet Count 159, Na 123, K 3.2, Gluc 71, BUN 22, Creat 0.97, Alb 3.8, AST 71, ALT 49, TB 0.1, INR 0.9, PTT 61.6, CK 4135, CKMB 57.8, Trop 0.08, TSH 1.1, spot cortisol 18.4

An EKG revealed sinus bradycardia with first degree AVB and low voltage QRS. A CT of her head was reported to be normal from the outside facility but an MRI revealed partial empty sella. An echocardiogram demonstrated a small pericardial effusion. An LP returned a CSF protein of 102, glucose 59, negative gram stain, WBC 30 (92N, 4M, 4L) RBC 4. A sample was sent for ehrlichia via PCR.

The patient was admitted to the ICU, pancultured and placed on IV fluids. Infectious Disease and Endocrinology were consulted. Her free T4 was nondetectable and there was no response to an ACTH stimulation test. A diagnosis of myxedema coma with panhypopituitarism was made, presumably secondary to Sheehan's syndrome, especially in light of her history and her partially empty sella. Noting that myxedema coma carries a mortality rate of 40%, Endocrinology recommended the administration of 150 mcg of levothyroxine IV; this was followed by 200 micrograms IV on day 2 and thereafter changed to 125 mcg PO daily. She was also treated with hydrocortisone 100 mg IV q8h, tapered to 50 mg q8h and then switched to oral hydrocortisone 20 mg q AM and 10 mg q PM . Since myxedema can blunt a patient's ability to respond to infection with leukocytosis, empiric antibiotic coverage was considered but, based on the lack of other clinical findings, was not initiated. Her hyponatremia responded to conservative measures and to the above therapeutic regimen and the patient's overall clinical condition gradually improved. She was discharged to home within a week of her admission and will be followed by her PCP and the Endocrinology Clinic.

(continued)

DISCUSSION:

Hypopituitarism is caused by pituitary adenomas or their therapy (radiation, surgery) in about 75% of cases. Another 13% are caused by extrapituitary tumors and almost 10% of cases are idiopathic. Rare causes include tuberculosis, Wegener's granulomatosis, hemochromatosis and sarcoiodosis. Sheehan's Syndrome (postpartum pituitary necrosis) is responsible for about 0.5% of cases in developed countries (though significantly higher in developing countries with limited resources for post-partum care). However, as discussed below, the incidence of this syndrome may be more common than realized since symptoms may be mild and the diagnosis is often delayed for many years.

Enlargement of the anterior pituitary is a normal physiologic development during pregnancy, compressing the superior hypophyseal artery and placing the organ at risk for infarction should hypotension or vasospasm develop in the face of postpartum hemorrhage. Rapid response with fluid resuscitation and transfusion mitigates this complication though focal ischemia and infarction may occur even with aggressive therapy. In rare cases, acute decompensation may result, but, in the majority of cases, the endocrinologic effects are mild and, though failure of lactation and postpartum amenorrhea are common, the patient may otherwise remain asymptomatic for years or even decades. Indeed, in a study by Ozkan and Colak [2], 20 cases of Sheehan's syndrome were reviewed; the age at diagnosis ranged from 40 to 65 years, with a mean age of 51 years. All 20 patients were found to have GH, Prolactin, FSH, TSH and ACTH insufficiency; 11 had empty sella and the other 9 had partial empty sella.

Empty sella on CT or MRI may be primary or secondary. Primary empty sella is due to a defect in the diaphragm sella, allowing CSF pressure to enlarge the sella space; these patients usually have normal pituitary function. Secondary empty sella is due to destructive lesions (or surgical resection) of the pituitary; the sella itself is normal in size but the pituitary mass is small and hormonal deficiencies develop. Patients with Sheehan's syndrome may have one or more pituitary hormone deficiencies, related to the degree of infarction and atrophy that occurs. Though some have postulated that an abnormally small sella may predispose a woman to Sheehan's syndrome, the great majority of cases have been associated with a normal sized sella on imaging.

The role of autoimmunity in the development of Sheehan's syndrome remains controversial. This condition has not be clearly associated with any other autoimmune diseases and, while some studies have demonstrated an increase in anti-pituitary antibody in these patients, this may be secondary to antigen shedding following the initial tissue infarction. Lymphocytic hypophysitis, which occurs in both men and women, may mimic Sheehan's syndrome, especially since its development is postpartum in almost 60% of the female patients; however, this condition has no association with postpartum hemorrhage.

REFERENCES:

1. Kelestimur, F., Sheehan's Syndrome, Pituitary 2003; 6: 181-188
2. Ozkan, Y and R. Colak, Sheehan Syndrome: clinical and laboratory evaluation of 20 cases, Neuro Endocrinol Lett 2005; 26(3): 257-260
3. Zagar, AH et al., Epidemiologic aspects of postpartum pituitary hypofunction (Sheehan's Syndrome), Fertil Steril 2005; 84: 523
4. Barkan, AL, Pituitary atrophy in patients with Sheehan's Syndrome, Am J Med Sci 1989; 298: 38
5. Up to Date, 2012

HOSPITAL MEDICINE VIRTUAL JOURNAL CLUB

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Abstracts & Full Links from recent journals of interest to Hospitalists

http://beckerinfo.net/JClub

ACP HOSPITALIST CONFERENCE

AT THE MISSOURI ACP SCIENTIFIC MEETING

SATURDAY, SEPTEMBER 15, 2012

8 AM TO 4:30 PM

LUNCHEON INCLUDED

8AM	DNR—Time to Resuscitate the Code Blue Discussion Richard Butin MD, FACP
8:50 AM	Stress Cardiomyopathy—an update on Broken Heart Syndrome Kevin Bybee MD
9:40 AM	Break and Exhibits
10 AM	Management of Pressure Ulcers David Thomas MD, FACP, AGSF, GSAF
11 AM	Vascular Embolization by I.R. Clinical applications: present & future Nasir Siddiqi MD
12:15 PM	Hospitalist Luncheon
	Transitions of Care: from the Office to the Hospital and Back
	James Duff MD, Marc Merbaum MD, James Rogers MD, FACP, Stephen Wen MD
1:30 PM	Open forum on Hospitalist Issues (Non-CME Program) Andrew Evans MD, FACP
2:30 PM	ABIM SEP Module—Hospital Based 2012 Update (83-M)
	Kyle Moylan MD, FACP and Kevin Clary MD

Visit **www.missouriacp.org** for more details!

FROM THE JOURNALS

CARLA DYER, MD

The following articles should be of interest to Hospitalists:

Prediction of Heart Failure Mortality in Emergent Care

Lee, et al., Annals of Internal Medicine 2012; 156: 767-775

http://annals.org/content/156/11/767.full.pdf+html

Infectious Diseases Society of America Clinical Practice Guideline for Diagnosis and Treatment of Diabetic Foot Infections

Lipsky, et al., Clin Infect Dis 2012; 54 (15 June): e132-173

http://www.idsociety.org/uploadedFiles/IDSA/Guidelines-Patient_Care/PDF_Library/2012%20Diabetic%20Foot%20Infections%20Guideline.pdf

Effect of the Medicare Part D Coverage Gap on Medication Use Among Patients with Hypertension and Hyperlipidemia

Pengxiang, et al., Annals of Internal Medicine 2012; 156: 776-784

http://www.annals.org/content/156/11/776.full.pdf+html

ID CORNER

WILLIAM SALZER, MD

MANAGEMENT OF ACUTE SINUSITIS

The IDSA has just released its practice guidelines for managing acute sinusitis:

IDSA clinical practice guidelines for acute bacterial sinusitis in children and adults

Chow, AW et al., Clin Infect Dis 2012; 54: 1041-1045

http://www.idsociety.org/uploadedFiles/IDSA/Guidelines-Patient_Care/PDF_Library/IDSA%20Clinical%20Practice%20Guideline%20for%20Acute%20Bacterial%20Rhinosinusitis%20in%20Children%20and%20Adults.pdf

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MISSOURI HOSPITALIST CALENDAR

Inflammatory Bowel Disease, A Combined Medical & Surgical Symposium, June 16, Eric P. Newman Education Center, Washington University Medical Center, St. Louis, register via http://cme.wustl.edu **LOCAL**

Caring for the Frail Elderly, 21st Annual Meeting, Missouri Association of Long-Term Care Physicians, August 17-18, Holiday Inn Select, Columbia, MO; you may download the conference brochure at **medicine.missouri.edu/cme**; call 573-882-9973 for information **LOCAL**

ACP Hospitalist Conference, at MO ACP Annual Scientific Meeting, Saturday, September 15, 8AM-4:30 PM (includes Luncheon); see agenda on page 5 **LOCAL**

Sepsis-related 8th Annual Midwest Hospital Medicine Meeting, Society of Hospital Medicine, October 10-13, Northwestern University, Chicago; www.hospitalmedicine.org

Unrealistic Expectations in Healthcare, 8th Annual Health Ethics Conference, October 18-20, Columbia, MO; info and registration via **medicine.missouri.edu/CME** or call 573-882-9973 **LOCAL**

Academic Hospitalist Academy, Society of Hospital Medicine, October 22-25, Atlanta; info at www.hospitalmedicine.org

Please forward this newsletter to Hospitalists that you might know!