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Hospitalist Update

Low Mobility of Older Adults During Hospitalization: a Problem to Address

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Most hospitalists recognize that the hospital environment can be dangerous for older adults. Older patients are at increased risk for many potentially preventable complications of hospitalization, including injurious falls, use of restraints, adverse drug events and pressure ulcers. Of equal concern is the threat to their functional independence along with the looming possibility that they will leave the hospital less independent and may require nursing home care.

The functional decline related to an acute illness and hospitalization is well described for older adults. Older patients frequently lose the ability to perform one or more basic Activities of Daily Living (ADL) during an acute illness. Decline in ADL ability is a potent predictor of subsequent acute illness and the need for nursing home placement. The decline in function can be the result of acute and chronic medical conditions but can also stem, in part, from the care provided in the hospital. Prognosticating which patients will experience functional recovery or decline following an acute illness can be challenging. Of note, one study found that physicians tend to underestimate the prognosis for functional recovery in seriously ill older adults while the patients, themselves, are more optimistic and more accurate than the physicians (Wu et al).

Many older adults experience low levels of mobility in the hospital, often limited to the bed or a chair; one study showed that only 27% of paients walk in the halls during their hospitalization. The consequences of being essentially bedridden also influence the outcome of the hospitalization in numerous ways, including low plasma volume, orthostatic intolerance, loss of muscle mass, loss of strength, constipation and pressure sores; of course, all of this should be of concern to hospitalists.

Brown et al. recently provided new insights into the scope of the problem. Utilizing wireless accelerometers attached to patients, they continuously recorded the activity level of 45 hospitalized patients over the age of 65; patients with delirium, dementia or who had been nonambulatory for two weeks prior to admission were excluded.

The results of their study are disappointing. On average, 83% of their hospital stay was spent in bed (approximately 20 hours per day); the average percent of time that any one individual was standing or walking ranged from 0 to 21%, with a mean of 3% (43 minutes per day).

What are the barriers to mobilizing hospitalized older adults? Brown et al. investigated these factors through structured interviews with patients, nurses and physicians. The most common barriers were symptoms (especially weakness, pain and fatigue) and tethers (IV lines and urinary catheters). Other perceived barriers were concerns about fall risk and the lack of staff to assist patients. Interestingly, health care providers often cited lack of patient motivation as a limitation to mobility while patients never mentioned this as a contributing cause.

So, what can hospitalist do to improve the mobility of their hospitalized older adults? First, we address the underlying condition leading to the hospitalization while managing the symptoms that may be limiting their mobility (such as pain). We should order the least restrictive activity level possible and reserve bedrest for the few patients for whom it is medically indicated. Early consultation with physical and occupational therapists can help to maximize mobility for patients who are already impaired or are quickly developing disability, especially if they have had recent falls or may need assist devices. We can also address the tethers by reviewing the need for IV lines, catheters, oxygen and other tubing on a daily basis. Physicians should also avoid prescribing medications that may trigger orthostatic symptoms or lead to altered mental status. Finally, it may be helpful to establish a better method for communicating and documenting the mobility status of older patients; one method recently described is a quick and simple bedside evaluation called I-MOVE, which yields a score of 1-12, from an inability to reposition in bed to walking independently in the hallway (Manning et al.).

REFERENCES:

Wu, AW et al., *Estimates of Future Physical Functioning by Seriously Ill Hospitalized Patients, their Families and their Physicians*, J Am Geri Soc 2002; 50:230-237

Brown, CJ et al., *The Underrecognized Epidemic of Low Mobility During the Hospitalization of Older Adults*, J Am Geri Soc 2009; 57:1660-1665

Brown, CJ et al., *Barriers to Mobility During Hospitalization from the Perspective of older Patients and their Nurses and Physicians*, J Hospital Medicine 2007; 2:305-313

Manning, DM et al., *Home Alone: Assessing Mobility Independence before Discharge*, J Hospital Medicine 2009; 4:252-254

HOSPITAL MEDICINE VIRTUAL JOURNAL CLUB

WASHINGTON UNIVERSITY SCHOOL OF MEDICINE

Abstracts and Full-text Links from recent journals of interest to Hospitalists:

http://beckerinfo.net/JClub

Hospitalists are invited to peruse the articles and to post comments

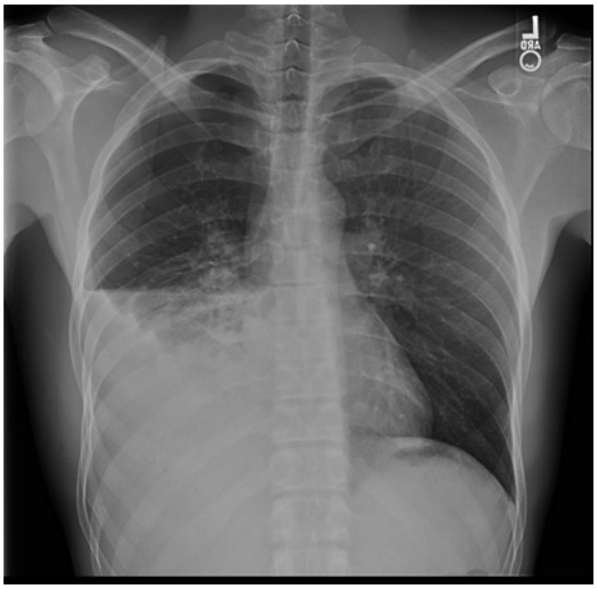
CASE OF THE MONTH

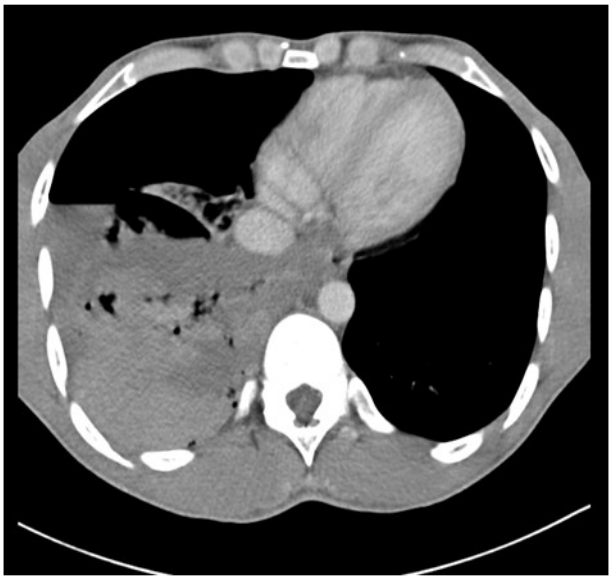
Kelsey Flynt M4 & Emily Coberly MD

A previously healthy 26 year old African American male presented to the ED with the acute onset of right-sided posterior thoracic pain and shortness of breath over the previous four hours. He described the pain as severe, sharp and exacerbated by deep inspiration and reported that it radiated to the RUQ and anterior chest wall. He denied fever, chills, cough, trauma to the chest wall or a history of similar episodes in the past. He denied the use of IV drugs and had no known exposure to TB; however, he did admit to a history of tobacco use (1/2 ppd for two years), a history of inhaling cocaine (most recently two days ago) and a history of incarceration several years prior to this presentation.

Physical exam on admission demonstrated that he was alert and fully oriented. Vitals were WNL except for tachypnea with shallow respirations; his O2 Sat was 97%. He was lying very still for his exam and grimaced with any movement. The ENT exam was unremarkable and his trachea was midline though asymmetric chest motion was noted, with diminished chest expansion on the right. In the supine position, his right chest was tender to palpation, hyperresonant to percussion anteriorly and dull to percussion laterally. He was mildly tender to palpation in the RUQ. The remainder of his exam was normal.

Admission labs revealed a WBC of 6.5, Hgb 10.5, Hct 31.7, MCV 92.3 and Platelet count of 143. Coagulation studies were normal. BMP was normal except for BUN 26 and Cr 1.4; LFTs, serum amylase and serum lipase were all normal. Cardiac enzymes were normal and a UA was unremarkable. ABGs on supplemental oxygen via nasal cannula revealed pH 7.3, pCO2 45, pO2 111, HCO3 24, BE –0.6 and O2 sat of 98.7%. A rapid HIV was negative and a PPD later proved to be negative. A CXR and CT Chest revealed a right hydropneumothorax, possibly representing a large empyema (see images below).





Empiric IV antibiotics were ordered and a chest tube was placed with the immediate return of 1 liter of bloody fluid, free of clots. Pleural fluid studies were not consistent with empyema but, rather, suggested a hemothorax: the fluid HCT was 30.1%, the gram stain was negative and cultures, including mycobacteria, were all negative. Cytology of the pleural fluid revealed RBCs but was negative for malignant cells. Following placement of the chest tube and initiation of IV hydration, his Hgb remained stable, renal function normalized and the lung (cont)

(cont) re-expanded without evidence of underlying mass or infiltrate. The patient remained afebrile and had no leukocytosis throughout his hospital stay; the chest tube was removed without complication and he was discharged to home.

DISCUSSION:

The recreational use of cocaine can be traced back to early civilizations. Originally documented by the ancient Moche tribe of Peru over 2500 years ago, the practice of liberating cocaine from coca leaves (using lime produced from roasted sea shells) was still occurring in Venezuela in 1499, at the time of Amerigo Vespucci. Cocaine's ability to alter physiologic activity was clear to even the early users of the drug. Cieza de Leon wrote "When I inquired of certain Indians why they keep their mouths ever filled with that herb…..they say that they have little sense of hunger and feel great vigor and strength." [1] The ability of cocaine to induce such feelings of euphoria is likely responsible for its popularity in modern-day culture.

Cocaine can be introduced to the body in a multitude of ways. Popular methods include inhalation, intravenous injection or mucosal absorption (intranasal, sublingual, intravaginal or rectal). Inhalation results in a more rapid onset of action and a shorter time to peak effects than other methods [2]. The mechanism by which the drug is introduced is an important consideration when evaluating a patient for pathologic effects.

Pulmonary complications are common in patients who inhale cocaine. Cough is present in 26-61% of subjects and hemoptysis develops in up to 26% [4]. Bleeding may result from rupture of submucosal vessels or directly from the alveolar-capillary membrane. Autopsy series have revealed that 85% of patients who died from cocaine intoxication had evidence of pulmonary hemorrhage [5]. Pneumothorax, pneumomediastinum and pneumopericardium may occur less frequently but are also well-documented complications of cocaine inhalation. In one study of 71 crack smokers who presented to an ER with chest pain, two were found to have pneumomediastinum, one had a pneumothorax and one had a hemopneumothorax, directly attributed to the cocaine inhalation [4].

The manner in which the cocaine is inhaled and the techniques used to increase absorption across the pulmonary -capillary barrier can predispose the patient to barotrauma. Users attempt to introduce cocaine into the pulmonary system by maximal inhalation; once the peak inspiratory volume is achieved, users resort to one of several methods to enhance absorption of the drug into the bloodstream. These techniques include the Valsalva maneuver and the forceful exhalation by a smoking partner into the mouth of the user [3]. Additionally, severe coughing may be induced by irritation from the cocaine powder. All three of these factors trigger a sudden increase in intra-alveolar pressure that may exceed the elastic potential of the parenchyma, resulting in alveolar rupture [3].

Treatment of pneumothorax or hemothorax due to cocaine inhalation is similar to that for spontaneous pneumothorax: chest tube placement, supplemental oxygen and supportive care, including pain management. Of course, cessation of cocaine use is an important part of the therapy and a referral to a drug abuse treatment program should be considered. [3]

REFERENCES:

1. Karch, Steven, A Brief History of Cocaine: from Inca Monarchs to Cali Cartels: 500 years. CRC Press, 2006
2. Lange, R & D. Hillis, *Cardiovascular Complications of Cocaine Use*, NEJM August 2, 2001; 345:351-358
3. Mechem, C Crawford, *Pulmonary Complications of Cocaine Abuse*,, UpToDate, September 24, 2009
4. Haim, D et al., *The Pulmonary Complications of Crack Cocaine: A Comprehensive Review*, Chest, January, 1995; 107 (1):233-240
5. Restrepo, C et al., *Pulmonary Complications from Cocaine and Cocaine-based Substances: Imaging Manifestations*. Radiographics 2007; 27:941-956

FROM THE JOURNALS — Carla Dyer MD

These two recent articles on Pulmonary Embolism should be of interest to hospitalists:

Early Anticoagulation is Associated with Reduced Mortality for Acute Pulmonary Embolism

Smith, Sean B et al.

Chest, June, 2010; 137:1382-1390

Acute Pulmonary Embolism

Giancarlo Angelli MD and Cecilia Becattini MD, PhD

New England J Medicine 2010; 363:266-274

ID CORNER — William Salzer MD

TREATING ACINETOBACTER

Hospital acquired Acinetobacter infections are often resistant to multiple antibiotics and difficult to treat. Here is a nice short review on antibiotic therapy for Acinetobacter infections:

Fishbain, J and AY Peleg, Treatment of Acinetobacter Infections

Clin Infect Dis 2010; 51:79-84

http://www.journals.uchicago.edu/doi/pdf/10.1086/653120

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MISSOURI HOSPITALIST CALENDAR

20th Annual Conference: Caring for the Frail Elderly, August 20-21, Holiday Inn Select Executive Center, Columbia, MO: call 573-882-5661 or register at som.missouri.edu/CME **LOCAL**

12th Annual Critical Care Update, Saturday, September 11, Drury Hotel, Chesterfield, MO; Washington University Medical Center; register online at: http://cme-online.wustl.edu **LOCAL**

Missouri Chapter ACP Scientific Meeting, Updates in Internal Medicine, Tan Tar A Resort, Osage Beach, September 23-26; Hospitalist Luncheon at 12:15, September 25; contact Patrick Mills 573-636-3366, pmills@msma.org **LOCAL**

Update on Current Management of Aortic Valve Disease, Saturday, October 2, Ritz-Carlton, St. Louis; Washington University Medical Center; register online at: http://cme-online.wustl.edu **LOCAL**

Brain Attack! 2010, Comprehensive Stroke Care Door-to-Door, Saturday, October 9, Eric P. Newman Education Center, Washington University Medical Center, St. Louis, register at http://cme-online.wustl.edu **LOCAL**

Chest 2010, October 30-November 4, Vancouver, BC, register online via: www.chestnet.org/accp/chest/chest-annual-meeting

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Please direct all comments, ideas and newsletter contributions to the Editor:

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Please forward this newsletter to Hospitalists that you might know!