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Hospital Medicine

Evidence-Based Information for Hospitalists Intensivists, and Acute Care Physicians [ALERT]

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ABSTRACT & COMMENTARY

Hospital Report Cards: What Do the Grades Mean?

By Deborah J. DeWaay, MD, FACP

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Dr. DeWaay reports no financial relationships in this field of study

SYNOPSIS: Administrative databases may not give an accurate representation of hospital-acquired pressure ulcer rates and may be inappropriate for use to compare hospitals and determine financial penalties.

SOURCE: Meddings JA, Reichert H; Hofer T; McMahon L. Hospital Report Cards for Hospital-Acquired Pressure Ulcers: How Good Are The Grades? *Annals of Internal Medicine*. 2013; 159: 505-513

■ SUMMARY

Hospital-acquired pressure ulcers (HAPUs) are associated with significant morbidity and mortality for patients. The cost of a stage III or IV ulcer can range between $5,000 and $151,700. The principle motivator for hospitals to prevent HAPUs has been "value-based purchasing" programs. In 2008, Medicare began the Hospital-Acquired Conditions (HAC) Initiative, which eliminated payments for certain nosocomial complications. Beginning in October 2014, the quartile of hospitals with the highest risk-adjusted HAC rates will receive a 1% pay reduction for all of their Medicare patients. The HAPU rate will be a part of the HAC rate, which determines who is in the bottom quartile as determined by a measure known as the Agency for Healthcare Research and Quality (AHRQ) Patient Safety Index 3. As a result, the administrative data hospitals use to generate claims for payment will now be used to determine a complication rate for HAPUs and HACs in general. This study sought to assess the validity of the administrative data for this purpose. The authors compared the hospitals with the highest HAPU rates based on administrative data from the Agency for Healthcare Research and Quality (AHRQ) that is collected from insurance claims with a statewide surveillance data set of HAPU rates based on comprehensive patient skin examinations.

This study retrospectively analyzed one year's

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Hospital Medicine [ALERT]

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worth of records from the Healthcare Cost and Utilization Project State Inpatient Databases, which collect administrative patient data and are sponsored by the AHRQ, and compared them to the quarterly hospitalwide pressure ulcer surveillance data from California hospitals found in the Collaborative Alliance for Nursing Outcomes Pressure Ulcer Prevalence Study. From the administrative data set the authors excluded patients under 16, no age listed, obstetric deliveries, and prisoners. They also excluded the following: specific hospitals for children, maternity and surgery, and those with no surveillance data. In addition, the authors excluded hospitals with less than 6 months of administrative or surveillance data reported from both data sets. The surveillance data has certain clinical exclusions applied to it as it is built, such as medical instability and patients who are being palliated. It was not possible to apply these exclusions to the administrative data set.

The administrative HAPU rate for each hospital was the cumulative rate of stage II HAPUs (HAPU2+) as determined by patients with a secondary ICD-9 code for a hospital-acquired pressure ulcer stage II or greater over the entire length of stay for each hospitalization who had no pressure ulcer on admission. The surveillance HAPU rate was the percentage of patients examined in a particular hospital who had a stage II HAPU and no ulcer on admission.

Data from 196 hospitals were included in this study. The patients with HAPU2+ were older, with longer length of stays, had more diagnoses listed and were hospitalized more frequently for unscheduled surgery. The mean administrative HAPU2+ rate was .15% (CI .13% - .17%; range, 0% - .74%) as compared to the mean surveillance HAPU2+ rate of 2.0% (CI 1.8% - 2.2%; range, 0% - 7.3%). The hospitals were ranked based on their administrative HAPU2+ rate and placed into quartiles. They were then compared to the hospitals as ranked by the surveillance data. Only 35% of the bottom quartile of hospitals in the administrative data set were below average in the surveillance data set. In addition, the correlation of rates between the two data sets was only weakly positive, with a Pearson correlation coefficient of 0.2 (CI, 0.06 to 0.33).

■ COMMENTARY

Although the authors anticipated a discrepancy between the surveillance and administrative data, a 10-fold increase in the HAPU2+ rate from the surveillance data as compared to the administrative data was quite surprising. The authors attribute the low rate in the administrative data set to its basis on what the coders use in their claims. The coders can only use diagnoses that are supported by providers (MDs, PAs or nurse practitioners). Wound care nursing notes can only be used if they are supported in the primary provider's documentation. In addition, the surveillance data is generated by a team that does quarterly hospitalwide skin examinations and reviews the entire medical record, including nursing notes, in order to generate data. Given the financial impact of the new rules to hospitals, this discrepancy between the two data sets in terms of HAPU2+ rate in general and the lack of consistency in determining the "poor performers" is disturbing for several reasons. First, there appear to be several ways hospitals can manipulate the system to lower their administrative HAPU2+ rates, such as not listing the ulcer in the discharge diagnoses. Although I agree with the authors that "gaming the system" was probably not occurring in this data set because there was no financial incentive to do so in 2009, there will be financial incentive in 2014. Second, if all hospitals are not making the same effort to really minimize their HAPU rate, which first requires identifying the "real" rate via excellent data collection, then the hospitals that are making the most effort to record correctly will be penalized. There is serious concern that the race to stay out of the bottom quartile and avoid potential financial penalty will not be a race of truly preventing HAPUs but a race to have the best administrative data set. ■

ABSTRACT & COMMENTARY

Oral Apixaban for the Treatment of Acute Venous Thromboembolism

By Harold L. Karpman, MD, FACC, FACP
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Dr. Karpman reports no financial relationships relevant to this field of study.
This article originally appeared in the November 15, 2013, issue of Internal Medicine Alert. *It was edited by Stephen Brunton, MD, and peer reviewed by Gerald Roberts, MD. Dr. Brunton is Adjunct Clinical Professor, University of North Carolina, Chapel Hill, and Dr. Roberts is Senior Attending Physician, Long Island Jewish Medical Center, NS/LIJ Health Care System, New Hyde Park, NY. Dr. Brunton serves on the advisory board for Abbott, Amarin, Boehringer Ingelheim, Duchesnay, Janssen, Lilly, Novo Nordisk, Sunovion, and Teva; he serves on the speakers bureau of Boehringer Ingelheim, Janssen, Lilly, Novo Nordisk, and Teva. Dr. Roberts reports no financial relationship to this field of study.*

Synopsis: A fixed dose of oral apixaban alone was noninferior to conventional therapy for the treatment of acute venous thromboembolism and was associated with significantly less bleeding.
Source: Agnelli G, et al. Oral apixaban for the treatment of acute venous thromboembolism. *N Engl J Med* 2013;369:799-808.

Venous thromboembolism occurs only one to two times per 1000 persons in the general population of the United States. However, it is the third most common cause of vascular death after myocardial infarction and stroke.[1] Conventional therapy consists of a parenteral anticoagulant such as enoxaparin for at least 5 days and warfarin, which should also be started upon diagnosis and continued for at least 3 months.[2] This classic therapeutic regimen is moderately inconvenient, especially in remote geographic regions, both because enoxaparin requires daily subcutaneous injections and because warfarin therapy requires coagulation monitoring and dose adjustment. The oral factor Xa inhibitor apixaban has a rapid onset of action and predictable pharmacokinetics that permits a fixed-dose regimen that significantly simplifies the treatment of venous thromboembolism by eliminating the need for both the initial parenteral anticoagulant therapy and subsequent laboratory monitoring of warfarin therapy.[3,4]

Although apixaban had been demonstrated to be effective for the prevention of recurrent venous thromboembolism with rates of major bleeding that are similar to those for placebo,[5] Agnelli and his colleagues decided to compare apixaban with conventional anticoagulant therapy in patients with acute symptomatic venous thromboembolism.[6] In this randomized, double-blind study, apixaban therapy (at a dose of 10 mg twice daily for 7 days, followed by 5 mg twice daily for 6 months) was compared with conventional therapy of subcutaneous enoxaparin followed by warfarin therapy in 5395 patients with acute venous thromboembolism. The primary efficacy outcomes were the avoidance of recurrent symptomatic venous thromboembolism or death related to venous thromboembolism. The principal safety outcomes were the occurrence of major bleeding alone or clinically relevant nonmajor bleeding. The results revealed that the primary efficacy outcome occurred in 2.3% of the apixaban group compared with 2.7% in the conventional therapy group. Therefore, apixaban proved to be not inferior to conventional therapy. Major bleeding occurred in 0.6% of patients receiving apixaban and in 1.8% of patients who received conventional therapy. Major bleeding and clinically relevant nonmajor bleeding occurred in 4.3% of the patients in the apixaban group and in 9.7% of the conventional therapy group.

■ COMMENTARY

For the treatment of acute venous thromboembolism, this well-conducted, multicenter study clearly demonstrated that a fixed-dose regimen of oral apixaban alone was as effective as conventional treatment and, equally important, it was associated with a clinically relevant reduction of 69% in major bleeding episodes.[6] The reduction in major bleeding was paralleled by a decrease in clinically relevant nonmajor bleeding, which thereby provided further evidence for the safety of the apixaban regimen. These results added to the previously published evidence[3,4] that simple therapy with one of the newer oral anticoagulants proved to be an excellent alternative to conventional therapy for patients with acute venous thromboembolic conditions. The

efficacy of apixaban in patients with pulmonary embolism was similar to that in patients with deep venous thrombosis. The efficacy and safety of apixaban also proved to be consistent across a broad range of subgroups, including patients who were > 75 years or who were obese. On the basis of the results obtained in this current study as well as in other studies such as the Apixaban for the Extended Treatment of Deep Vein Thrombosis and Pulmonary Embolism trial,[5] oral apixaban appears to be a simple, effective, and safe drug for the initial and long-term treatment of venous thromboembolism. ■

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ABSTRACT & COMMENTARY

Colchicine in Acute Pericarditis

By Andrew J. Boyle, MBBS, PhD

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Dr. Boyle reports no financial relationships relevant to this field of study.

This article originally appeared in the December 2013 issue of Clinical Cardiology Alert. *It was edited by Michael H. Crawford, MD, Professor of Medicine, Chief of Clinical Cardiology, University of California, San Francisco, and peer reviewed by Ethan Weiss, MD, Assistant Professor of Medicine, Division of Cardiology and CVRI, University of California, San Francisco. Dr. Crawford reports no financial relationships relevant to this field of study, and Dr. Weiss is a scientific advisory board member for Bionovo.*

Source: Imazio M, et al. A randomized trial of colchicine for acute pericarditis. *N Engl J Med* 2013;369:1522-1528.

Colchicine is indicated for treatment of recurrent pericarditis. However, its role in acute pericarditis is less clear. Previous observational series and single-center, open-label studies have suggested a benefit in acute pericarditis, but this remains to be proven. Accordingly, Imazio and colleagues performed a randomized, placebo-controlled, multicenter study of usual care (aspirin, ibuprofen, or glucocorticoids) plus either colchicine or placebo in patients with acute pericarditis.

Patients ≥ 18 years of age presenting with their first episode of acute pericarditis were enrolled. They were considered to have acute pericarditis if they had at least two of the following four clinical features: typical chest pain (pleuritic and relieved by sitting forward), typical ECG changes, pericardial friction rub, or pericardial effusion on echocardiography. Patients with idiopathic, viral, post-cardiac injury or connective tissue disease-associated pericarditis were included. Exclusion criteria included the presence of myopericarditis, tuberculous, neoplastic or purulent pericarditis, significant liver or kidney disease (creatinine > 2.5 mg/dL), blood dyscrasias, myopathy, pregnancy, lactation, and inflammatory bowel disease. Colchicine or placebo were administered for at least 3 months. The dose of colchicine was 0.5 mg twice daily, or 0.5 mg once daily for those under 70 kg and those with side effects on 0.5 mg twice daily. Usual care was either aspirin 800 mg or ibuprofen 600 mg every 8 hours. Glucocorticoids were reserved for patients intolerant of aspirin or ibuprofen, and were tapered over 2 weeks. All patients also received proton pump inhibitors. All patients were followed for at least 18 months, and the primary endpoint was recurrent or incessant pericarditis. Recurrent pericarditis was defined as resolution of acute pericarditis for at least 6 weeks followed by another episode. Incessant pericarditis was defined as ongoing symptoms or a repeat episode with less than 6 weeks symptoms-free. Secondary endpoints were symptom persistence at 72 hours, remission within 1 week, number of recurrences, time to first recurrence, pericarditis-related hospitalization, cardiac tamponade, and constrictive pericarditis.

A total of 240 patients were randomized to colchicine (n = 120) or placebo (n = 120). Baseline demographics were similar between groups. The average age was 52 years, 61% were male, and the vast majority were idiopathic/viral in

etiology (77%). Pericardial effusion was present in two-thirds. Usual care consisted of aspirin in approximately 80%, ibuprofen in 15%, and glucocorticoids in 5%. Adherence to the study drug was closely monitored and exceeded 95% in both groups. The primary outcome of recurrent or incessant pericarditis occurred in 17% of the colchicine group and 38% of the placebo group (relative risk 0.56; $P < 0.001$). The number needed to treat (NNT) to prevent one case of recurrent or incessant pericarditis was 4. The results were similar whether patients were treated with aspirin or ibuprofen. Patients treated with colchicine, compared to placebo, had lower rates of symptom persistence at 72 hours (19% vs 40%; $P = 0.001$), pericarditis-related hospitalization (5% vs 14%; $P = 0.02$), and number of recurrences per patient (0.21 vs 0.52; $P = 0.001$). Colchicine improved the rate of remission at 1 week (85% vs 58%; $P < 0.001$) and time to first recurrence (25 weeks vs 18 weeks; $P < 0.001$). Adverse event rates and drug discontinuation rates did not differ between groups (12% vs 10% and 12% vs 8%, respectively). The authors conclude that in patients with acute pericarditis, colchicine added to conventional anti-inflammatory therapy significantly reduced the rate of recurrent or incessant pericarditis.

■ COMMENTARY

Colchicine is an old drug that has an established role in recurrent pericarditis. Recently, it has been shown to prevent pericarditis after cardiac surgery and now in the current study, it has gained a place as treatment for the first attack of acute pericarditis. The rigorous trial design strengthens the conclusions made from this dataset. The study was an investigator-initiated, randomized, placebo-controlled, multicenter trial with all events adjudicated by a blinded clinical events committee. Interestingly, the majority of patients took aspirin as the anti-inflammatory of choice, which may reflect regional practice variations. The study did not address which anti-inflammatory is the best, allowing ibuprofen or aspirin at the treating physician's discretion. The major limitations to the use of colchicine in clinical practice are nausea and diarrhea. Interestingly, in this study, there were no differences in the rates of gastrointestinal upset or drug discontinuation compared to placebo. The dose reduction in patients who develop side effects (rather than cessation) and the lower dose in smaller patients are important in maintaining patients on their therapeutic regimen. This is a low-cost, well-tolerated treatment that can improve symptoms and reduce hospitalizations for pericarditis. This will be a welcome addition to standard anti-inflammatories in patients with their first attack of acute pericarditis. ■

ABSTRACT & COMMENTARY

Esmolol in Septic Shock

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Dr. Boyle reports no financial relationships relevant to this field of study.

This article originally appeared in the December 2013 issue of Clinical Cardiology Alert. *It was edited by Michael H. Crawford, MD, Professor of Medicine, Chief of Clinical Cardiology, University of California, San Francisco, and peer reviewed by Ethan Weiss, MD, Assistant Professor of Medicine, Division of Cardiology and CVRI, University of California, San Francisco. Dr. Crawford reports no financial relationships relevant to this field of study, and Dr. Weiss is a scientific advisory board member for Bionovo.*

Source: Morelli A, et al. Effect of heart rate control with esmolol on hemodynamic and clinical outcomes in patients with septic shock: A randomized clinical trial. *JAMA* 2013;310:1683-1691.

Cardiac patients are prone to develop sepsis in the intensive care unit (ICU) setting. The physiological changes associated with septic shock, such as tachycardia and hypotension, may be particularly deleterious to cardiac patients. Therefore, knowledge of ways to ameliorate these physiological changes may benefit cardiologists. Morelli and colleagues addressed the issue of whether beta-adrenoceptor blockade would be beneficial by reducing tachycardia or deleterious by reducing inotropy in patients with septic shock. They performed a single-center, randomized, open-label study of patients in ICU with septic shock, a heart rate > 95/min, and requiring high-dose norepinephrine to maintain mean arterial blood pressure (MAP) > 65 mmHg. They excluded minors, those on beta-blocker therapy prior to randomization, patients with significant cardiac

impairment (cardiac index < 2.2 L/min/m^2 and pulmonary capillary wedge pressure > 18 mmHg) or valve disease, and pregnant women. All patients had a pulmonary artery catheter and arterial line, and received fluid resuscitation to achieve a right atrial pressure ≥ 8 mmHg. Patients were randomized to a continuous infusion of esmolol, titrated to heart rate 80-94/min for the duration of ICU stay, vs usual care. The primary endpoint was attaining heart rate 80-94/min. Secondary endpoints included hemodynamic parameters and 28-day survival.

A total of 154 patients were randomized to receive esmolol infusion (n = 77) vs usual care (n = 77). Baseline demographics were similar between groups; the mean age was 68 years and 54% were male. Pneumonia was the most common source of sepsis. There was a wide spectrum of bacteria isolated as the causative organisms, with *Klebsiella* being the most common and *Staphylococcus aureus* being isolated in 8%. The target heart rate was achieved in 100% of patients receiving esmolol with a mean reduction of 18 beats/min ($P < 0.001$ vs usual care). Patients receiving esmolol showed improved hemodynamic parameters compared to usual care, including significant improvements in stroke volume, systemic vascular resistance, and left ventricular stroke work index. Fluid requirements were lower in the esmolol group (3.98 L/d vs 4.43 L/d; $P < 0.001$) and norepinephrine requirements, which were equivalent at baseline, were also reduced. Renal function improved in the esmolol group, but not in the usual care group. Unadjusted mortality at 28 days was lower in the esmolol group (49.4% vs 80.5%; $P < 0.001$). Multivariate analysis showed that the Simplified Acute Physiology (SAPS) II score and esmolol were the only independent predictors of survival. The authors conclude that for patients in septic shock, open-label use of esmolol compared to usual care was associated with reductions in heart rates to target levels, without increased adverse events, and that the observed improvements in mortality and other secondary endpoints warrant further investigation.

■ COMMENTARY

Heart rate control in septic shock is controversial. Studying patients in the ICU is inherently difficult because of the heterogeneous nature of the diseases that caused them to be there, and also because of the myriad comorbidities that may confound their clinical course and treatment. Thus, randomizing patients to a particular treatment regimen is mandatory in such studies to exclude biases. In addition, the high acuity of such patients makes blinding of treatment difficult, and titrating to a heart rate goal makes a placebo problematic to deliver. The randomized, open-label design of this trial is therefore appropriate and the authors should be commended on completing such a study. However, the open-label design does allow for some confounding of the results.

Several aspects of this study should be highlighted. First, they excluded patients with significant cardiac impairment (cardiac index < 2.2 + wedge pressure > 18 mmHg) and patients on beta-blockers prior to randomization. Therefore, the results may not be generalizable to all cardiac patients. Second, the doses of esmolol used were modest, with the mean being 100 mg/hr, and the dose used did not predict mortality at 28 days. Third, the heart rate goal was arbitrarily chosen, and the optimal heart rate in patients with septic shock remains unknown.

How should one incorporate these data into clinical practice? It is reasonable in patients with septic shock, in whom tachycardia may be producing deleterious cardiac effects, to try low-dose esmolol to reduce the heart rate. There were no safety concerns raised by this study, and this may allow reduction in vasopressor doses. However, routine use of esmolol to reduce mortality based on the secondary endpoints of this study would be premature. ■

ABSTRACT & COMMENTARY

Embolic Risk in Endocarditis

By Michael H. Crawford, MD

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This article originally appeared in the December 2013 issue of Clinical Cardiology Alert. *It was peer reviewed by Ethan Weiss, MD, Assistant Professor of Medicine, Division of Cardiology and CVRI, University of California, San Francisco. Dr. Crawford reports no financial relationships relevant to this field of study, and Dr. Weiss is a scientific advisory board member for Bionovo.*

Source: Hubert S, et al. Prediction of symptomatic embolism in infective endocarditis: Construction and validation of a risk calculator in a multicenter cohort. *J Am Coll Cardiol* 2013;62:1384-1392.

At the time infective endocarditis (IE) is diagnosed, an accurate predictor of the risk of embolic events would help guide the risk/benefit ratio of surgical intervention. Thus, these investigators from two regional centers in France reviewed cases of definite IE by the modified Duke criteria that did not have isolated cardiac device electrophysiologic lead endocarditis or recurrent IE. Embolic events that occurred before diagnosis and the initiation of antibiotic therapy were noted, as well as supporting initial imaging data. The primary endpoint was symptomatic embolic events from the initiation of antibiotic therapy to surgery or 6 months of follow-up. Over the study period, 847 patients met inclusion criteria at the two centers, mean age was 62 years, and 72% were men. Streptococci and staphylococci organisms were causative in half the patients. Prosthetic valve IE occurred in one-fourth of the patients. Head CT scans were performed on admission in 97% and 493 patients had surgery within 30 days. The subjects were randomly divided into a development sample (n = 565) and a validation sample (n = 282). In the total population, 72 had emboli within 14 days and the central nervous system received half. After 6 months, 62% had undergone surgery and 20% had died. The 6-month embolic rate was 8.5% and was highest in the first 2 weeks. In the development group, age, diabetes, atrial fibrillation, previous embolism, vegetation length, and *Staphylococcus aureus* infection were predictive of emboli. A prediction model based on these variables performed well in the validation sample and can be programmed into a handheld device. The authors concluded that at the diagnosis of IE, a simple calculation of embolic risk can be accurately made that could be useful for therapeutic decisions.

■ COMMENTARY

Systemic emboli are common in IE and portend a poor prognosis. Prior studies have shown an association between emboli and vegetation size, *staphylococcus* infection, and prior embolization, which are characteristics of the infections. These factors highlight the importance of the early deployment of blood cultures and echocardiography in suspected IE. Prior emboli refers to the period before the diagnosis is made and antibiotics are started. The diagnosis of emboli could be made by clinical presentation in symptomatic patients (e.g., stroke) or by imaging in asymptomatic patients. The best imaging approach is not determined by this study, but the importance of prior emboli suggests that brain imaging should be done early to detect silent lesions. Several studies have used MRI for this purpose, but PET is promising and doesn't require contrast. This study adds three patient characteristics that are also strongly associated with emboli: diabetes, atrial fibrillation, and age. These features are combined with the three infection features to provide a risk calculator that can be downloaded on a handheld device. The calculator can be accessed through the electronic version of the paper, in the appendix.

This study and others have shown that after antibiotic therapy is started, the risk of emboli decreases rapidly in the first 2 weeks and is unusual after 4 weeks. Surgical therapy has also been shown to reduce emboli and mortality in selected patients. If surgical therapy is to have an impact on emboli, it needs to be done early. Delays to sterilize the vegetation or other such unproven tactics put the high-risk patient at considerable risk of emboli. Delays that last longer than 2 weeks diminish the potential benefits of surgery for emboli prevention. Surgery beyond 2 weeks is usually done for hemodynamic reasons. In considering early surgery in IE, the potential benefit of surgery has to be balanced against the risk. Estimation of the former has been augmented by this study, because it better identifies the patient at high risk for emboli. Some of these factors used to predict emboli risk also predict surgical risk such as age, so the calculation of surgical risk is complicated in IE patients. If a patient is high risk for emboli and seems low risk for surgery, then early surgery makes sense.

This is a retrospective database study done at two tertiary care centers. Thus, there are referral and selection biases. The study only addresses the prediction of embolic risk by factors discernible early in the course of IE. It does not address the value of surgery, but this has been studied before and there are guidelines for deploying surgical therapy early to prevent emboli. The international guidelines specify prior emboli and vegetation length as factors favoring early surgery in IE patients. This paper adds four more factors to consider and recommends a combined risk score to help with the decision. ■

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CME QUESTIONS

1. Beginning in October of 2014, how will the hospitals with the highest risk-adjusted HAC rates be penalized?

a. 3% pay reduction from insurance companies for all of their patients

b. 1% pay reduction for all of their Medicare patients

c. 1% pay reduction from insurance companies for all of their patients

d. 3% pay reduction for all of their Medicare patients

2. In the prospective randomized controlled trial of oral apixiban for acute venous thromboembolism, which of the following outcomes was observed:

a. The group receiving oral apixiban had slightly more major and minor bleeding.

b. The group receiving oral apixiban had a substantially higher rate of recurrence of venous thromboembolism.

c. Oral apixiban was noninferior to conventional therapy in that the principle efficacy was similar in both groups.

d. All of the above.

3. The use of colchicine for acute pericarditis was found by Imazio and colleagues to:

a. Decrease the risk of persistent or recurrent pericarditis by around half.

b. Increase the risk of hemorrhagic pericarditis.

c. Increase the risk of GI bleeding.

d. Decrease the risk of mortality by half.

CME OBJECTIVES

Upon completion of this educational activity, participants should be able to:

- discuss pertinent safety, infection control and quality improvement practices;
- explain diagnosis and treatment of acute illness in the hospital setting; and;
- discuss current data on diagnostic and therapeutic modalities for common inpatient problems.

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