

administration appears to be a major factor in the development of NSF in patients with severe renal failure, the fear of this complication should not eliminate the use of enhanced MRI/MRA when truly indicated.

#### References:

Cowper, SE, Nephrogenic systemic fibrosis: an overview, J. Am College Radiology 2008 Jan; 5(1):23-28

Perazella, MA, Current status of gadolinium toxicity in patients with kidney disease, Clin J Am Soc Nephrol 2009 Feb; 4(2): 461-469

U.S. Food & Drug Administration: Gadolinium-based Contrast Agents for MRA, 2007:

[http://www.fda.gov/cder/drug/InfoSheets/HCP/gcca\\_200705.htm](http://www.fda.gov/cder/drug/InfoSheets/HCP/gcca_200705.htm)

**CARE OF THE HOSPITALIZED PATIENT 2009**  
WASHINGTON UNIVERSITY, SATURDAY, MAY 2  
SEE CALENDAR FOR REGISTRATION DETAILS

## CASE OF THE MONTH

### Divya Gupta MD, Ankit Mehra MD, Emily Coberly MD, James Koller MD

A 72 year old male presented to the MU Internal Medicine inpatient service with a two month history of fatigue and malaise. He reported URI symptoms and low grade fevers during this period for which he had been seen at several outpatient facilities. Treatment had included courses of penicillin and Azithromycin with no improvement in his symptoms; a strep screen had been negative. He finally saw his PCP who ordered blood cultures, a urine culture and a CXR; when 4 of 4 blood cultures turned positive for gram + coccobacilli, he was admitted for further workup.

His past medical history was remarkable for mitral prolapse, mitral regurgitation, prolactinoma chronic renal insufficiency (baseline Cr 1.5) and hypertension. In 2004, he was treated for mitral valve Strep viridians endocarditis with a 4-week course of Penicillin G, followed by ceftriaxone. Regular meds at the time of his current admission included lisinopril, allegra, pravastatin, atenolol and cabergoline; he denied drug allergies. His family history was limited to CHF in his mother. The patient denied use of alcohol, tobacco or illicit drugs. He was a retired chemical engineer and lived with his wife.

On admission, he denied chills, rigors, cough, chest pain, dyspnea, headache, visual change, rashes, joint pain, back pain, orofacial pain, dysuria or hematuria. He denied preceding travel abroad or insect bites. His most recent dental care was 6 months prior to the onset of his current illness and he took prophylactic penicillin before that appointment.

Admission exam revealed T 37C, HR 73, BP 138/65 and O2 sat 96% on RA. He was alert and oriented with no signs of acute distress. A grade 3/6 pansystolic murmur was best heard at the apex with no radiation. Chest was clear to auscultation and abdominal exam was normal without organomegaly. The remainder of his exam was normal, with no peripheral stigmata of endocarditis. Admission labs returned Hgb 12.5, WBC 15.5 (70%N), Platelets 321. CMP was normal except BUN 27 and Cr 1.5; urinalysis was clear; CXR showed no infiltrate or effusion. Blood cultures were repeated from two sites and, due to the suspicion of endocarditis, he was treated empirically with ceftriaxone and vancomycin.

A TTE on the following day revealed a mobile vegetation on the anterior leaflet of the mitral valve, measuring 10mm x 7mm; his ejection fraction was normal but moderate-severe mitral regurgitation was noted. A Panorex revealed an abscess in an upper molar, confirmed by dental exam, and the tooth was extracted. Three separate blood cultures grew gram + coccobacilli which were difficult to culture on blood agar but grew well on chocolate agar. With the help of the State lab, the organism was identified as *Abiotrophia defectiva*. The ID consult team recommended changing his antibiotic regimen to ampicillin and gentamicin at synergy dosing and the patient was discharged, with home health arrangements to complete a six week course of IV antibiotics. He was educated regarding the high risk of recurrence and on the use of prophylactic antibiotics.

**Discussion:** With a past history of mitral regurgitation and endocarditis, our diagnosis of recurrent endocarditis was straight forward; however, the case highlights the importance of maintaining a high index of suspicion for recurrence in these patients and of stressing good dental care. There is some controversy regarding the risk imposed by dental procedures; current evidence suggests that low grade bacteremia occurs with daily activities such as brushing, flossing and food chewing and that the cumulative exposure over one year is 5.6 million times the bacteremia induced by a single tooth extraction. Furthermore, various studies have shown that antibiotic therapy is not effective in reducing the frequency of bacteremia. Nevertheless, current AHA guidelines recommend prophylaxis for cardiac conditions associated with a high risk of endocarditis, including a prosthetic heart valve, a valve repaired with prosthetic material, previous endocarditis and congenital heart disease (unrepaired cyanotic, repaired with prosthetic material or repaired but with residual defect).

Patients with relapsing or recurrent endocarditis are at greater risk of CHF and the need for valve replacement; our patient had moderate-severe mitral regurgitation but demonstrated no signs of CHF or systemic embolization. The risk of CHF with mitral valve endocarditis is 20% and the risk of embolization is greatest with vegetations > 10mm during the first 2 weeks of therapy. The highest incidence of embolic complications have been reported with *Abiotrophia*, *S. aureus*, *Candida* and HACEK organism. Early diagnosis is essential to initiate appropriate therapy and prevent complications.

*Abiotrophia defectiva*, a nutritionally variant streptococcus (NVS), is part of the normal oral flora. NVS accounts for 5-6% of cases of endocarditis and is an important component of culture-negative endocarditis. This infection carries a higher mortality rate than enterococci or strep viridians, has a relapse rate of 17% and has a treatment failure rate of 41% (despite antibiotic efficacy in vitro); it has been suggested that, in light of their slow growth rate, a longer course of antibiotics may be beneficial. Furthermore, while NVS is sensitive to many antibiotics in vitro, this does not always predict a good in vivo response. Current AHA guidelines recommend treating NVS like enterococcal endocarditis with Ampicillin 12 g/24h or Penicillin G 18-30 million units/24h in combination with Gentamicin for synergy at 3mg/kg/24hr, adjusted to a peak serum concentration of 3-4 mcg/ml and a trough concentration of < 1 mcg/ml. Repeat TTE is recommended to establish a new baseline prior to completing therapy and close clinical followup is crucial to monitor for signs of relapse or CHF. The patient must be educated to watch for signs of relapse, to adhere to good dental hygiene and to receive regular dental checkups. During therapy, it is also important to monitor for antibiotic toxicity (especially ototoxicity and nephrotoxicity with aminoglycosides).

(continued on page 4)

**Microbiology:** As *Abiotrophia defectiva* does not routinely grown on blood agar, many cases likely remain unidentified (and its incidence in endocarditis may be underestimated). NVS was first identified by Frenkel and Hirsch, in 1961; in 1989, Bouvet et al. showed that NVS occurs as two strains: *Streptococcus defectiva* and *Streptococcus adiacens* (confirmed by RNA sequencing in 1995). Both Vitamin B6 and cysteine are required for growth and the bacteria cannot be cultured on media without these nutrients; NVS can grow on chocolate agar and brucella agar. As discussed above, it grows more slowly than other streptococci, has a high affinity for endocardium and may be less sensitive in vivo than is evident in vitro, leading to persistent infection. As always, prompt identification and treatment of the pathogen are critical.

*Infective Endocarditis: Diagnosis, Antimicrobial Therapy and Management of Complications*, Circulation 2005; 111:e394-e434

*Blood stream and endovascular infections due to Abiotrophia defectiva and Granulicatella species*, BMC Infectious Diseases 2006; 6:9

---

## FROM THE JOURNALS

William Steinmann MD

### **Asthma in Seniors: Part 1. Evidence for Underdiagnosis, Undertreatment and Increasing Morbidity and Mortality**

Stupka, E. and R. deShazo, American Journal Medicine, January 2009, Volume 122, No. 1, 6-11

This is a comprehensive survey of the clinical literature supporting the epidemiology, diagnosis and natural history of asthma in individuals over 65 years of age. In addition, expert consensus publications by the National Heart, Lung & Blood Institute and National Institute of Health were reviewed. The authors found that:

- The population of seniors with asthma is increasing rapidly in the U.S.
- These patients have a high level of morbidity and mortality with their asthma
- Seniors with asthma have often been excluded from clinical trials of asthma management due to their age or comorbid conditions
- Evidence is presented that a new approach to this chronic disease is warranted

### **Hand-carried Ultrasound performed by Hospitalists: Does it improve the Cardiac Physical Examination?**

Martin, L. David et al., American Journal of Medicine, January 2009, Volume 122, No. 1, 35-41

Observer variability of any clinical observation may be considerable and previous studies have demonstrated disagreement of cardiac auscultation findings, even among cardiologists. In this study, the hospitalist's performance with and without a hand-carried ultrasound was compared with a cardiologist's interpretation of the patient's hospital echocardiogram.

The hospitalist's assessment of right ventricular function, cardiomegaly and pericardial effusion were improved clinically and statistically by using the ultrasound; assessments of aortic stenosis, aortic regurgitation and mitral regurgitation were not improved. While the clinical benefit achieved by the immediacy of this device was not determined, the promise of its potential is highlighted in an accompanying editorial. We await further study as hospitalists are looking to expand their diagnostic capabilities.