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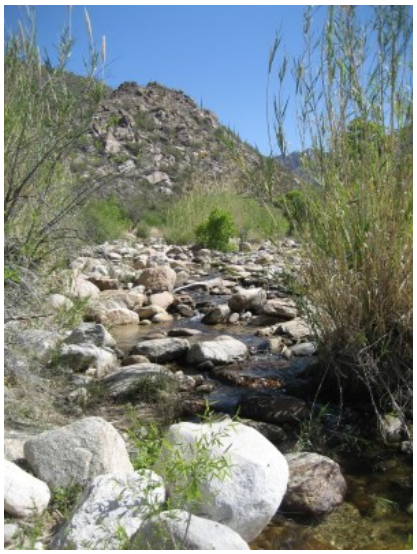
Hospitalist Update



The Daily Progress Note

Emily Coberly MD

During my intern year, I usually arrived at the hospital early each morning to pre-round. After putting on my white coat, I would head to the nursing station where I would grab a stack of yellow progress notes. Before seeing each patient, I would review all of the morning labs and copy them into the empty fish bones that were stamped onto the bottom half of the progress note page. The next stop was the medication room, where I copied each patient's daily medication list from the red binder to my paper. I then traveled to each patient's room where I transcribed the vital signs from the bedside chart onto the progress note, being careful to leave room at the top of the note so that I could enter the subjective data later. After morning rounds, I wrote in the assessment/plan section and returned to the nursing station, where I removed a patient label from each chart, fixed them to the top of the appropriate page and inserted the note into the chart folder. Of course, a certain percentage of charts were always missing from the rack, either gone with the patient for a procedure or grabbed by a consultant team for their rounds.

Since our hospital implemented an electronic medical record and converted to electronic documentation, the process of writing a daily progress note has changed dramatically. It is now possible to obtain vital signs, lab data and medication lists for a patient from anywhere in the hospital with a click of the mouse. Progress notes are typed and can be immediately forwarded to other members of the health care team, including primary care doctors and consultants.

There are several clear advantages to electronic documentation. Electronic notes are more accessible and better organized than handwritten notes [1]. They eliminate legibility problems and are more readily available to other members of the care team; the data in these records can also be more easily obtained for billing and research purposes. Considering these advantages, electronic documentation can improve communication, efficiency and note accuracy.

Unfortunately, electronic notes also create a new set of challenges. Compared with paper notes, electronic records are longer and more redundant [1]. They can also take more time to write [2], though this fact does not consider time saved in the (continued)

(cont) manual collection of data. To increase efficiency in writing their electronic records, many physicians take advantage of features such as "copy forward" or "copy and paste." While these tools can be helpful, the failure to review and update the notes leads to the inclusion of inaccurate, outdated and extraneous information which can make the note unreliable and/or difficult to interpret [3]. This is a common problem in hospitals that use electronic documentation with up to 20% of clinical notes demonstrating use of the copy and paste option [4]. As a result, electronic notes are often long, overly detailed and redundant (compared with written notes, which were often short, illegible and difficult to locate). Despite these new problems, most physicians feel that the benefits of electronic records justify their use and would not recommend a return to handwritten, paper documentation [4].

It is easy to focus on the problems of new technologies and lose sight of the purpose of our notes. Physician documentation is important for research, billing and legal purposes but is especially vital for effective communication with other members of the healthcare team. As we look for ways to improve the quality and efficiency of progress notes, we will need to consider the development of templates and invest more time in learning how to fully utilize the system that we have; making an effort to teach residents and medical students how to construct quality notes will be equally important. While working to overcome the new challenges produced by electronic documentation, we must stay focused on the essential purpose of our daily notes.

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2. Poissant, L et al., *The impact of electronic health records on time efficiency of physicians and nurses: a systemic review*. J Am Med Inform Assoc, 2005; 12(5): 505-516

3. Hirschtick, RE, *A piece of my mind: Copy and paste.* JAMA, 2006; 295(20): 2335-2336

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HOSPITALIST CONFERENCE & LUNCHEON

MISSOURI ACP MEETING

<u>THIS SATURDAY</u>, SEPTEMBER 26, 12:15 PM

TAN-TAR-A RESORT, LAKE OF THE OZARKS

TOPIC: HOSPITAL ACQUIRED INFECTIONS

http://www.acponline.org/meetings/chapter/mo-2009.pdf

CASE OF THE MONTH

Timothy Enders DO, Molly Lewandowski MD, Annette Quick MD, Sindhu Koshy MD, Harshal Patil MD UMKC

Apical Ballooning Syndrome and Multivessel Coronary Vasospasm in a Postmenopausal Woman

Abstract:

Objective: To assess a patient who developed Apical Ballooning Syndrome (ABS)

Aim: To evaluate the potential factors leading to ABS

Background: Apical Ballooning Syndrome is a cause of reversible cardiomyopathy that usually occurs in postmenopausal women. To date, no single cause of ABS has been identified; rather, ABS appears to be a consequence of multiple factors.

Methods: This report evaluates a patient with ABS by comparing her case to the factors believed to contribute to the development of this syndrome.

Results: This case involves a postmenopausal woman who developed multivessel coronary vasospasm and ABS while receiving estrogen replacement therapy (type and strength unknown).

Conclusion: Further studies are indicated to evaluate the possible relationship between estrogen replacement therapy and the development of ABS in postmenopausal women.

Case Report:

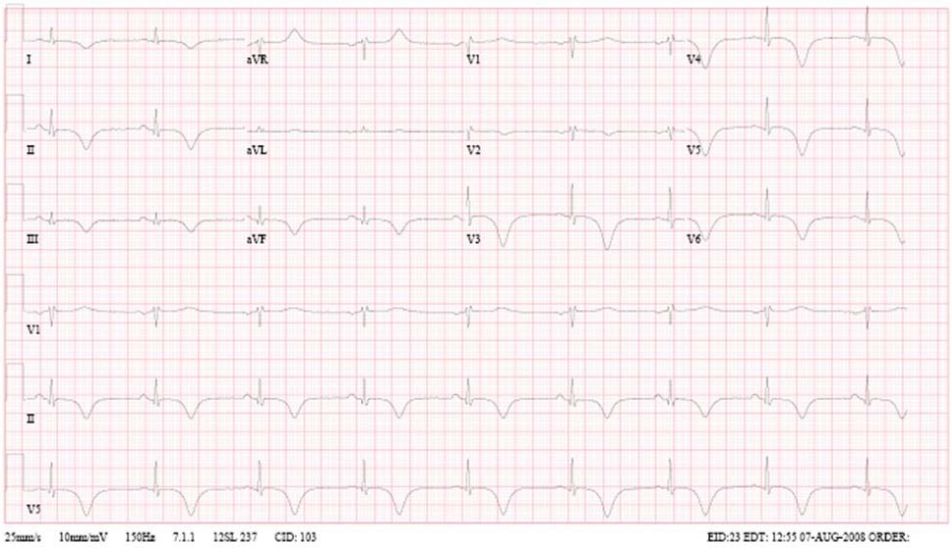
A 59 year old Caucasian female presented to the emergency department with left sided, substernal chest pain which had been increasing in intensity and frequency over the past four months and was not occurring three to four times per day. She described her chest pain as squeezing in nature, lasting 2-3 minutes, and reported that it occurred with exertion and at rest. On the evening prior to presentation, she experienced an episode that lasted 20 minutes which was 10/10 in intensity; she reported that the pain did not radiate and she could not identify any precipitating or alleviating factors. She denied nausea, dizziness or palpitations; however, she did describe diaphoresis and dyspnea with her most recent episode. The patient denied any history of orthopnea or PND. She lives alone and reported increased anxiety due to the loss of her job; she had used much of her savings to pay her bills.

The patient's PMH included hypertension, migraine headaches, fibromyalgia, breast cancer (at age 32) and degenerative disc disease. Past surgical history was limited to a right mastectomy. Home medications included Inderal LA, Fentanyl patch, oxycodone, prn colace and an estrogen supplement of unknown type and dosage. She reported no known drug allergies.

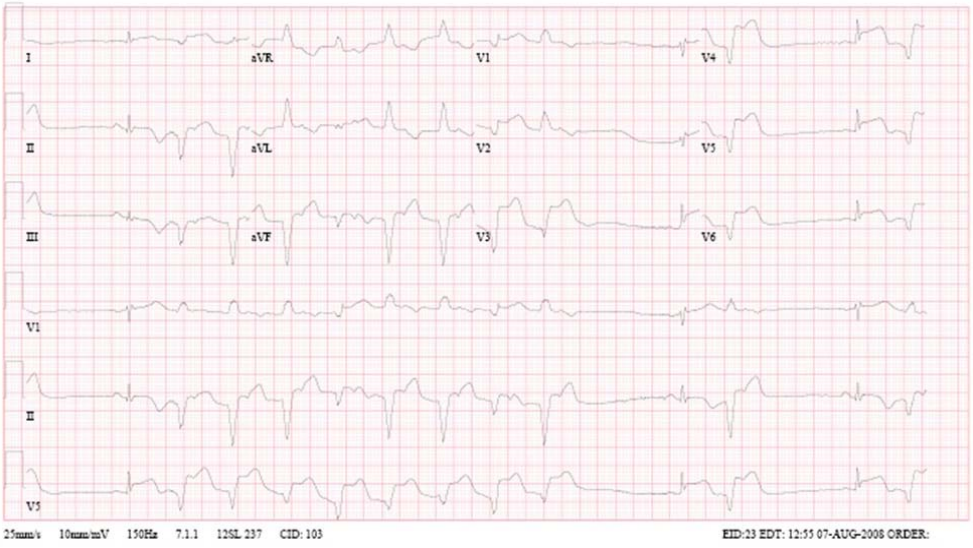
Exam revealed T 97.4F, P 74, BP 139/82, R 16, O2 sat 98% on RA. No heart or lung abnormalities were found on exam; there were no clinical signs of right or left heart failure (no JVD, rales, peripheral edema).

Her initial lab data revealed a serum troponin-I 0.80 ng/ml, CK-MB mass 6.9 ng/ml, total CK 102 IU/L, serum K 5.5, Ca 9.1, Mg 2.1, Phos 4.3, triglycerides 155, HDL 38, LDL 128, Albumin 2.2; LFTs were otherwise normal. Her WBC was 9900 and hemoglobin was 16.8 g/dl. A urine drug screen was negative. Urinalysis was negative for nitrites and leukocyte esterase.

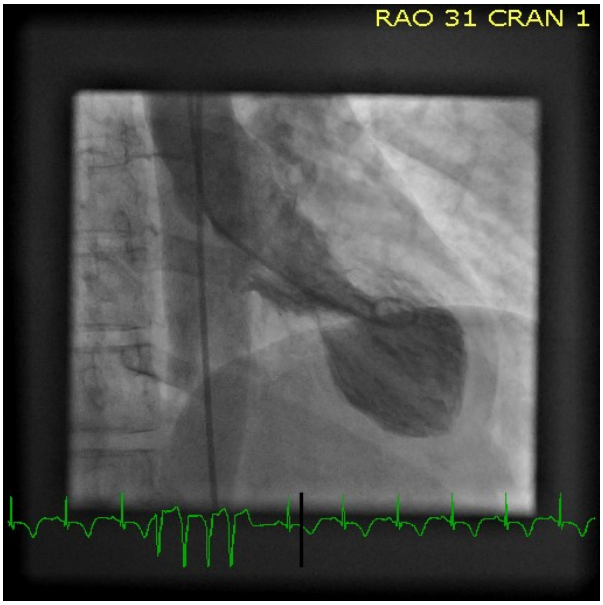
Her initial EKG (below) revealed sinus bradycardia, prolonged QTC and deep, diffuse T wave inversions, suggesting myocardial injury:



The patient was treated with morphine and nitroglycerin which alleviated her chest pain. She was placed on 2L of oxygen and was given ASA 325 mg, which was continued daily. A heparin drip was initiated and she was started on metoprolol, 25 mg every 12 hours, atorvastatin 80 mg qd, lisinopril 10 mg daily and clopidogrel 600 mg (as a one time dose). Initial CXR was negative for any acute cardiopulmonary process. On the following morning, the patient's chest pain redeveloped and her EKG revealed sinus tachycardia, non-sustained V tach and diffuse ST elevation:



The patient was taken to the cardiac cath lab emergently, where the angiogram revealed 60-70% stenosis in both the proximal LAD and ostial left circumflex arteries, which improved with intracoronary nitroglycerin; the left main and right coronary arteries were angiographically normal. A left ventriculogram revealed hypokinesis and paradoxical movement of the apex (figure, below); contractility at the base was reported to be normal.



The day following the cardiac catheterization, the patient had a 2D echocardiogram which revealed normal LV size and wall thickness. No segmental wall motion abnormalities were seen and the ejection fraction was estimated to be 45%. The patient was also reported to have grade II LV diastolic dysfunction. No significant valvular abnormalities were noted. The was continued hypokinesis of the apex but the findings were much improved.

The patient was started on amlodipine and the metoprolol was discontinued; ASA, lisinopril and prn NTG were continued; her atorvastatin dose was cut to 40 mg per day and she was prescribed a nicotine patch (21 mg per day) to help with cigarette cessation. The patient was discharged to home after 3 days of hospitalization.

Discussion: Apical Ballooning Syndrome (ABS), also known as Takotsubo cardiomyopathy, was first described in Japanese patients by Sato et al., in 1990 [1] and the topic has received increasing attention over the past two decades. One systemic review of 14 studies with 266 patients concluded that nearly 90% of reported patients were women and that the median age range was 58-77 [2]. ABS presents with chest pain and/or dyspnea and can imitate an acute ST-elevation myocardial infarction by clinical and EKG presentation; however, the elevation of serum myocardial markers is usually minimal. The onset of the syndrome is thought to be associated with a physiologic or emotional event and patients may have no angiographic evidence of coronary artery disease. The echocardiogram demonstrates normal basal wall motion with apical akinesis/dyskinesis that cannot be correlated with the distribution of a single coronary vessel. The condition is reversible and has a favorable prognosis, with full recovery often occurring within days to weeks of the acute event [2,3].

An early proposed mechanism for ABS was multivessel epicardial vasospasm; however, this was found in only 3 of 212 patients in the above systemic review [2]; more recent studies have demonstrated that this cause is unlikely though vasospasm may be a component (as in our case). Early, liberal use of nitroglycerin in these patients, who

(cont) are presumed to be having an MI, may mask the actual number of individuals who experience multivessel vasospasm [4].

An abnormality in the coronary microcirculation has also been evaluated as a possible cause for ABS [2,3,4]. Mohri et al. found that 29 of 117 patients (25%) had microvascular spasm; of these 29 individuals, most of whom were women, few had coronary risk factors [5]. One study of 16 women with LV wall motion abnormalities used Thrombolysis In Myocardial Infarction (TIMI) frame counts to evaluate these patients; in all 16 women, the TIMI frame counts were abnormal and usually involved all three major coronary arteries, indicating the possibility of diffuse coronary microcirculatory function impairment [6].

In one systemic review of 542 individuals with ABS, nearly 80% had experienced intense emotional or physical stress, presumably leading to stimulation of the sympathetic nervous system [7,8]. Regional wall motion abnormalities and decreased LV ejection fraction were found in a clinical study where mental stress led to a rise in serum catecholamines [9]; the physical stress of subarachnoid hemorrhage and pheochromocytoma, associated with high catecholamine levels, have been proposed as factors leading to some cases of ABS [10,11]. Apical Ballooning Syndrome has also been associated with high dopamine,, norepinephrine and epinephrine levels [12].

Since post menopausal women make up the vast majority of patients with ABS, the possible role of estrogen is beginning to receive greater attention. Clinical studies have demonstrated that long term estrogen replacement therapy attenuates vasoconstrictive response to catecholamines in postmenopausal women [4,13] and to mental stress in perimenopausal women [4,14]. In premenopausal women who experience Prinzmetal's variant angina, incidence of anginal attacks and endothelial dysfunction had an inverse correlation with the serum estradiol level [15,16]. Experimental studies have demonstrated that exogenous estrogen protects from the adverse cardiac effects of mental stress in oopherectomized rats [4,17,18].

Conclusion: The cause for Apical Ballooning Syndrome remains uncertain. Multivessel coronary vasospasm, as found in our patient, was once thought to be the cause but has since been found in a minority of cases. It is likely that ABS is a multifactorial condition that includes intense emotional or physical stress leading to a catecholamine surge which, in turn, stuns the myocardium via diffuse microvascular vasospasm; a lack of estrogen protection in postmenopausal women may increase their risk for ABS. Further studies are indicated to evaluate the potential association between ABS and estrogen replacement therapy.

References:

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FROM THE JOURNALS — Robert Folzenlogen MD

The following articles should be of interest to hospitalists:

Risk Factors for Preoperative and Postoperative Delirium in Elderly Patients with Hip Fracture

Vibeke Juliebo et al., *J American Geriatric Society* 2009; 57: 1354-1361

Does my patient have Clostridium difficile Infection?

Peterson, L.R. and A. Robicsek, *Annals Internal Medicine* 2009; 151: 176-179

Addition of Propranolol and Isosorbide Mononitrate to Endoscopic Variceal Ligation does not reduce Variceal Re-bleeding Incidence

Kumar, A. et al., *Gastroenterology* 2009; 137: 892-901

Efficacy of Procalcitonin in the early diagnosis of Bacterial Infections in a Critical Care Unit

Nakamura, A. et al., *Shock* 2009; Vol 31, 6: 586-591

ID CORNER — William Salzer MD

If you provide care for surgical inpatients, you may encounter infections in prosthetic joints. This recent article in the NEJM is a very good review of the management of those patients:

Infection associated with Prosthetic Joints

DelPozo, J.L. and R. Patel, New England Journal of Medicine 2009; 361:787-794

http://content.nejm.org/cgi/reprint/361/8/787.pdf

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MISSOURI HOSPITALIST CALENDAR

Missouri ACP Meeting, September 24-27, Tan-Tar-A Resort, Lake of the Ozarks; Hospitalist Conference Luncheon on Saturday, September 26, 12:15 pm; topic: Hospital Acquired Infections; see ad on page 2 of this newsletter. **LOCAL**

7th Annual Cardiology Update, October 3, 7:30-1PM, Memorial Union, University of Missouri, Columbia, to register: call Kathleen Yates, 573-882-2296 or visit www.mucardiovascular.org/update 2009 **LOCAL**

Health Care Access and Allocation of Resources, 5th Annual Health Ethics Conference 2009, University of Missouri School of Medicine, October 8-10, Reynolds Alumni Center and Hilton Garden Inn, Columbia; contact Kara at 573-882-5661, **LOCAL**

Brain Attack! 2009: Networks and New Therapies, October 10, Newman Education Center, Washington University; call 800-325-9862 or visit http://cme.wustl.edu; **LOCAL**

Hypertension & the Cardiometabolic Syndrome, October 15, 2009, Hampton Inn & Suites, Columbia, MO, University of Missouri Department of Medicine, call 573-882-0366 or visit www.som.missouri.edu/CME; **LOCAL**

CHEST 2009, October 31-November 5, San Diego; information and registration online at www.chestnet.org

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

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