PUTTING THE PIECES TOGETHER:

Can We Get Better with Hearts?

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Objectives:
1) Explain the interrelation of the early diagnosis and treatment of acute coronary syndrome.
2) Participants will explain how physicians can improve their hospital’s response to acute coronary syndrome and heart failure through improving relationships with Cardiology, Administration and the Laboratory.

The answer to the question posed by the title of this lecture is absolutely we can. Through a deliberate approach to building current thinking on acute coronary syndrome (ACS), including the impact of expert panel guideline implementation, the foundation has been developed for emergency physicians to become expert in this disease process. As noted in the two presentations on heart failure (HF), suboptimal understanding of the diagnosis and treatment of ACS, and failure to adhere to evidence-based approaches to care, can only increase the number of patients with HF. It is our hope that we are successful in providing a clear approach to the diagnosis and treatment of ACS and HF in the emergency department (ED).

An understanding of the pathophysiology of ACS has evolved substantially over the last decade. The role of inflammation and the acutely ruptured atheromatous coronary artery plaque has identified multiple new diagnostic and therapeutic approaches to the care of these patients. Markers of inflammation such as C-reactive protein can identify patients at greater risk for developing ACS and its complications. Efforts at reducing inflammation and the causes of atherosclerosis including elevated lipids (low density lipoproteins) with agents such as statins, now requires emergency physicians to be comfortable with this discussion. An acutely ruptured atheromatous plaque also requires rapid diagnosis and treatment to minimize complications including myocardial infarction and death.

Diagnostic evaluations for possible ACS have evolved dramatically. The development of the chest pain center concept now emphasizes the coherent approach to the diagnosis of ACS which includes an evaluation for myocardial necrosis, rest ischemia, and exercise-induced ischemia. The 12-lead ECG and troponins have been demonstrated as the best tools for risk stratification in patients with ACS. Through these two modalities, most patients with non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA) at risk for MI and death can be identified. Radionuclide imaging has also proved to be enormously successful in evaluating patients with possible ACS in the ED, particularly for identifying the low risk patient that can be sent home. Provocative testing using exercise diagnostics such as graded exercise testing, dobutamine echocardiography, or exercise sestamibi testing can provide useful information as to patients with fixed, hemodynamically significant atheromatous lesions requiring treatment. Perhaps the most exciting new additions to the diagnostics for ACS are the various biomarkers of acute ischemia. In the coming years, ischemia modified albumin, brain natriuretic peptide, and fatty acid binding protein promise to provide further information on patients with rest ischemia in the emergency setting.
While diagnostic advances have been substantial during the last decade, the evolution of therapy for ACS has been truly amazing. The elucidation of the role of platelets in the very early pathophysiology of ACS has allowed enormous gains in therapy for this disease process. Glycoprotein IIb/IIIa receptor inhibitors such as eptifibatide and ADP receptor inhibitors such as clopidogrel have revolutionized therapy for patients with ACS through the inhibition of platelet aggregation. These agents improve both short term and long term outcome for these patients and in most cases can be appropriately administered in the ED. In addition, antithrombotic treatment with both unfractionated and fractionated low molecular weight heparin is better understood and has certainly improved care.

Chest pain centers provide a frame work for EDs to develop a cohesive approach to the diagnosis and treatment of ACS. In collaboration with Cardiology, emergency physicians can develop a consistent approach to the early diagnosis and treatment of ACS. Involving the multiple constituents in this process of care, including Cardiology, Emergency Medicine, Internal Medicine, Family Medicine, Laboratory Medicine, and Administration, a coordinated diagnostic and therapeutic approach to ACS can be provided to every patient with chest discomfort presenting to the ED. Meeting with leaders from these various groups off-line and establishing diagnostic and treatment algorithms specific for a particular hospital, prevents a variable approach to each new patient which reflects individual emergency and cardiology clinician preference rather than an evidence-based approach.

The development of the CRUSADE Quality Improvement Initiative reflects this desire to incorporate evidence-based practice into the diagnosis and treatment of ACS. Based on the 2002 ACC/AHA guidelines for NSTEMI and UA, the CRUSADE initiative seeks to provide both the function of documenting care provided at over 500 hospitals across the United States and providing comparative data back to participating centers. A recent report from the RAND center estimates that physicians in a variety of disciplines across the United States follow expert specialty-based guidelines only 50% of the time. Data from the CRUSADE initiative demonstrate that centers which follow the 2002 ACC/AHA guidelines have fewer adverse outcomes, including death, from ACS than hospitals that do not have the same high level of adherence. Through providing specific tools to member hospitals such as algorithm cards, pre-printed nurse orders, and other aids, care for all patients with ACS at these institutions is expected to improve.

In a similar fashion to the diagnosis and treatment of ACS, HF has had a significant increase in new knowledge over the last decade. In the past, the diagnosis and treatment of HF was typically based on rather imprecise determination of a patient’s past medical history, symptoms, and presenting physical examination. The objective measurement of brain natriuretic peptide (BNP) has substantially improved the emergency physician’s diagnostic approach to these patients and also provides prognostic information as well. For HF, BNP serves as a diagnostic test as well as a therapy. In addition to the typical emergency treatment for HF which can include agents such as diuretics, ACE inhibitors, inotropic agents, morphine, and nitrates, BNP can provide an additional therapeutic modality for these severely ill patients. Emergency physicians must be adept at diagnosing HF on presentation and providing rapid treatment to prevent respiratory and cardiac complications. Better diagnostic and treatment regimens for ACS should presumably decrease the number of patients with HF presenting to emergency departments across the United States in the coming years.
In conclusion, an improved understanding of the pathophysiology of ACS has lead to significant advances in diagnostic testing and therapeutic agents. Diagnostic testing in the emergency setting must address not only myocardial necrosis, but also rest ischemia and exercise-induced ischemia. A thorough understanding of the 12-lead ECG, markers of necrosis such as troponin, and radionuclide testing can help provide better care for these ACS patients. Research into platelet biology has elucidated therapeutic mainstays such as glycoprotein (GP) IIb/IIIa receptor inhibitors and ADP receptor antagonists which can prevent platelet activated aggregation. Chest pain centers, using ACC/AHA guidelines for NSTEMI and UA, can provide a consistent, cohesive approach to the care of these patients with ACS. The CRUSADE Quality Improvement Initiative not only tracks the care of these patients at the participating centers, but also provides feedback to these hospitals to improve their processes. Hopefully improving the care of ACS, up front in the emergency department, can decrease the number of patients having HF as a complication of their disease.