Perspective

Early Management of Patients With Acute Heart Failure: State of the Art and Future Directions. A Consensus Document From the Society for Academic Emergency Medicine/Heart Failure Society of America Acute Heart Failure Working Group

SEAN COLLINS, MD, MSc,[1] ALAN B. STORROW, MD,[2] NANCY M. ALBERT, PhD,[3] JAVED BUTLER, MD,[4] JUSTIN EZEKOWITZ, MBBCh, MD,[5] G. MICHAEL FELKER, MD,[6] GREGORY J. FERMANN, MD,[7] GREGG C. FONAROW, MD,[8] MICHAEL M. GIVERTZ, MD,[9] BRIAN HIESTAND, MD, MPH,[10] JUDD E. HOLLANDER, MD,[11] DAVID E. LANFEAR, MD, MS,[12] PHILLIP D. LEVY, MD, MPH,[13] PETER S. PANG, MD, MSc,[14] W. FRANK PEACOCK, MD,[15] DOUGLAS B. SAWYER, MD, PhD,[2] JOHN R. TEERLINK, MD,[16] AND DANIEL J. LENIHAN, MD,[2] FOR THE SAEM/HFSA ACUTE HEART FAILURE WORKING GROUP

Nashville, Tennessee; Cleveland and Cincinnati, Ohio; Atlanta, Georgia; Alberta, Canada; Durham and Winston-Salem, North Carolina; Los Angeles, California; Boston, Massachusetts; Philadelphia, Pennsylvania; Indianapolis, Indiana; Detroit, Michigan; Houston, Texas; and San Francisco, California

ABSTRACT

Heart failure (HF) afflicts nearly 6 million Americans, resulting in one million emergency department (ED) visits and over one million annual hospital discharges. An aging population and improved survival from cardiovascular diseases is expected to further increase HF prevalence. Emergency providers play a significant role in the management of patients with acute heart failure (AHF). It is crucial that emergency physicians and other providers involved in early management understand the latest developments in diagnostic testing, therapeutics and alternatives to hospitalization. Further, clinical trials must be conducted in the ED in order to improve the evidence base and drive optimal initial therapy for AHF. Should ongoing and future studies suggest early phenotype-driven therapy improves in-hospital and post-discharge outcomes, ED treatment decisions will need to evolve accordingly. The potential impact of future studies which incorporate risk-stratification into ED disposition decisions cannot be underestimated. Predictive instruments that identify a cohort of patients safe for ED discharge, while simultaneously addressing barriers to successful outpatient management, have the potential to significantly impact quality of life and resource expenditures. (*J Cardiac Fail 2015;21:27–43*)

Key Words: Acute heart failure, emergency medicine, early management.

Heart failure (HF) afflicts nearly 6 million Americans, resulting in 1 million emergency department (ED) visits and more than 1 million annual hospital discharges.[1,2] An aging population and improved survival from cardiovascular diseases is expected to further increase HF prevalence.[3] By 2030 an estimated 25% increase in HF prevalence will result

From the [1]Nashville Veterans Affairs Medical Center and Vanderbilt University, Nashville, Tennessee; [2]Vanderbilt University, Nashville, Tennessee; [3]Cleveland Clinic, Cleveland, Ohio; [4]Emory University, Atlanta, Georgia; [5]University of Alberta, Edmonton, Alberta, Canada; [6]Duke University, Durham, North Carolina; [7]University of Cincinnati, Cincinnati, Ohio; [8]Ronald Reagan-UCLA Medical Center, Los Angeles, California; [9]Harvard Medical School, Boston, Massachusetts; [10]Wake Forest University, Winston-Salem, North Carolina; [11]Thomas Jefferson University, Philadelphia, Pennsylvania; [12]Henry Ford Hospital, Detroit, Michigan; [13]Wayne State University, Detroit, Michigan; [14]Indiana University School of Medicine, Indianapolis, Indiana; [15]Baylor College of Medicine, Houston, Texas and [16]San Francisco Veterans Affairs Medical Center, University of California, San Francisco, California.

Manuscript received April 28, 2014; revised manuscript received June 28, 2014; revised manuscript accepted July 10, 2014.

Reprints requests: Sean Collins, MD, MSc, Vanderbilt University, Department of Emergency Medicine, 1313 21st Ave South Nashville, TN 37232. Tel: 615-936-0087; Fax: 615-936-3754. E-mail: sean.collins@vanderbilt.edu

This paper is being simultaneously copublished in *Academic Emergency Medicine*.

See page 38 for disclosure information.

1071-9164/$ - see front matter

http://dx.doi.org/10.1016/j.cardfail.2014.07.003

in an additional 3 million affected individuals.[1,4] Of the $39.2 billion spent on HF care in the United States in 2010, inpatient admissions accounted for the single largest proportion. By 2030, the amount spent on hospital care for HF will be even greater as annual total costs are expected to be close to $70 billion.

Emergency providers play a significant role in the management of patients with acute heart failure (AHF). Therapeutic and disposition decisions made by emergency providers have direct impact on morbidity, mortality, and hospital length of stay, all of which affect health care costs.[5–9] More than 80% of ED patients with AHF are admitted to the hospital, a proportion that has remained largely unchanged over the past 5 years.[2] It is crucial that emergency physicians and other providers involved in early management understand the latest developments in diagnostic testing, therapeutics, and alternatives to hospitalization. Equally important are partnerships between emergency providers and HF specialists along with the entire interdisciplinary team caring for HF patients to streamline care from the ED to the inpatient and outpatient settings.

Current Approaches to Diagnosis

Although there is no universally accepted terminology to describe AHF, for the purpose of clarity we have chosen to use AHF defined as chronic or de novo HF with new or worsening symptoms requiring acute therapy. Patients present to the ED with signs and symptoms, not diagnoses. Although dyspnea is the most common symptom in AHF, it has a large list of differential diagnoses. Efficient diagnosis is critical, because delays in the delivery of care for AHF are associated with increases in mortality, hospital length of stay, and treatment costs.[10–14] Therefore, an understanding of the strengths and limitations of the history, physical examination, and laboratory and radiographic tests used to assist in the diagnosis of AHF is essential.

History and Physical Examination

Multiple studies suggest that there is no historical or physical examination finding that achieves sensitivity and specificity >70% for the diagnosis of AHF. Furthermore, only 1 clinical finding, the S3 gallop, achieves a likelihood ratio positive (LR+) >10 and none carries an LR− <0.1.[14] In a meta-analysis of 18 studies,[13] prior HF was the most useful historical parameter, with LR+ of 5.8 and LR− of 0.45. Dyspnea on exertion is the symptom with the lowest LR− at 0.48, but it has an LR+ of only 1.3,[13,14] whereas paroxysmal nocturnal dyspnea, orthopnea, and peripheral edema have the best LR+ (2.1−2.6), but notably poor LR− (0.64−0.70).[13,14] Emergency physician clinical judgment is only modestly useful, with LR+ 4.4 and LR− 0.45.[13] Although the S3 has the highest LR+ (11), it has far less utility as a negative predictor (LR− 0.88)[13] and suffers from poor interrater reliability.[15–18] Hepatojugular reflux (LR+ 6.4) and jugular venous distension (LR+ 5.1), are the only other examination findings with LR+ >5.

Chest Radiography

Chest radiography demonstrating pulmonary venous congestion, cardiomegaly, and interstitial edema are the most specific test findings for AHF (Table 1).[12,13] However, their absence cannot rule out AHF, because up to 20% of patients with AHF show no congestion on ED chest radiography.[19] Particularly in late-stage HF, patients may have few radiographic signs, despite AHF symptoms and elevated pulmonary capillary wedge pressure (PCWP).[12,20,21]

Electrocardiography

Electrocardiography is not useful for diagnosis, but it may suggest a specific cause or precipitant of AHF, such as myocardial ischemia, acute myocardial infarction, or arrhythmia. The presence of atrial fibrillation has a high LR+ for AHF; however, new t-wave changes are also associated with AHF (Table 1).[13] Electrocardiography may also offer clues as to the underlying cause of chronic HF (eg, Q waves in ischemic cardiomyopathy, low voltage in cardiac amyloid).

Biomarkers

The natriuretic peptides (NPs) B-type NP (BNP) and its precursor N-terminal pro-BNP are the most established AHF diagnostic biomarkers. They add value in the setting of undifferentiated dyspnea by improving diagnostic discrimination,[22,23] and they correlate with cardiac filling pressures and ventricular stretch.[24] NP testing is a class 1 (best evidence) guideline recommendation by both the Heart Failure Society of America (HFSA) and the American College of Cardiology Foundation (ACCF)/American Heart Association (AHA)[25,26] and may be particularly useful when the etiology of dyspnea is unclear; it has been shown to have greater utility than chest radiography for diagnosing AHF.[13,22] Newer markers, such as ST2 and galectin-3, have been explored for prognostic assessment and diagnosis of preclinical HF,[27,28] but their role in the ED is less clear.

NP levels can be affected by age, sex, weight, and renal function (Table 2).[29] Dyspnea not due to AHF can still be associated with NP elevation in a variety of conditions associated with myocardial stretch, (eg, right ventricular stretch from pulmonary hypertension or pulmonary embolism, acute coronary syndromes) or decreased renal clearance.[30–32] Patients with HF with preserved left ventricular ejection fraction (LVEF) have a smaller left ventricular radius and thicker walls than HF patients with reduced LVEF (HFrEF), resulting in proportionally lower NP levels for similar degrees of AHF, suggesting that different diagnostic thresholds are needed depending on whether LVEF is preserved or reduced.[33] Beyond clinical factors, additional variation in NP levels can arise from heritable[34] and specific genetic variants that have been shown to alter assay performance.[35] In general, changes of >50% from baseline represent worsening HF; however, significant variation in NP levels can