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Misconceptions in acute heart failure diagnosis and Management in the Emergency Department

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ABSTRACT

Introduction: Acute heart failure (AHF) accounts for a significant number of emergency department (ED) visits, and the disease may present along a spectrum with a variety of syndromes.

Objective: This review evaluates several misconceptions concerning heart failure evaluation and management in the ED, followed by several pearls.

Discussion: AHF is a heterogeneous syndrome with a variety of presentations. Physicians often rely on natriuretic peptides, but the evidence behind their use is controversial, and these should not be used in isolation. Chest radiograph is often considered the most reliable imaging test, but bedside ultrasound (US) provides a more sensitive and specific evaluation for AHF. Diuretics are a foundation of AHF management, but in pulmonary edema, these medications should only be provided after vasodilator administration, such as nitroglycerin. Nitroglycerin administered in high doses for pulmonary edema is safe and effective in reducing the need for intensive cardiogenic shock, norepinephrine is associated with improved outcomes and lower mortality. Disposition is complex in patients with AHF, and risk stratification tools in conjunction with other assessments allow physicians to discharge patients safely with follow up.

Conclusion: A variety of misconceptions surround the evaluation and management of heart failure including clinical assessment, natriuretic peptide use, chest radiograph and US use, nitroglycerin and diuretics, vasopressor choice, and disposition. This review evaluates these misconceptions while providing physicians with updates in evaluation and management of AHF.

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1. Introduction

Acute heart failure is a heterogeneous syndrome and one of the most common reasons for hospitalization in the U.S. for those older than 65 years [1-4]. It is commonly associated with coronary disease, renal disease, atrial fibrillation, diabetes, and hypertension [1,2,5]. This disease accounts for over 650,000 ED visits annually in the U.S., and close to 80% of patients with AHF are first evaluated in the ED, with the majority admitted [5-7]. Patients may present in a variety of ways, including gradual decline with worsening symptoms over several weeks, hypertensive pulmonary edema, or cardiogenic shock.

Emergency medicine evaluation and management typically focuses on initial resuscitation based on patient hemodynamics and degree of illness. Testing typically includes electrocardiogram, imaging, and laboratory assessment, with management including airway support, vasodilators, and/or diuretics [8-12]. Disposition is typically inpatient

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Though emergency physicians are well versed in the evaluation and management of this condition, with multiple sets of guidelines available [8-12], there are several components that remain misunderstood. This review seeks to provide emergency physicians with an improved understanding of evaluation and management in heart failure by addressing several common misconceptions.

2. Discussion

This review will focus on several components of evaluation, management, and disposition in the ED by investigating misconceptions in AHF.

2.1. Misconception: natriuretic peptide testing is routinely helpful in diagnosing or excluding AHF

Natriuretic peptides include B-type natriuretic peptide (BNP) and NT-proBNP. These molecules are cardiac neurohormones functioning

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in volume and sodium homeostasis produced in the cardiac musculature due to myocyte stretch, which may occur in AHF [8,10,13]. A precursor molecule, proBNP, is released with myocyte stretch, which is enzymatically cleaved to NT-proBNP and BNP [13-17]. The half-life of BNP is close to 20 min, while NT-proBNP's half-life is 3–6 times that of BNP's [13-17]. These molecules increase sodium and water excretion, increase peripheral vasodilation, and decrease activity of the renin angiotensin aldosterone system (RAAS) [8,10].

Natriuretic peptides are often used in AHF. An American College of Emergency Physicians (ACEP) clinical policy provides level B recommendations that with BNP < 100 pg/mL or NT-proBNP < 300 pg/mL, AHF is unlikely, while for BNP > 500 pg/mL or NT-proBNP > 1000 pg/mL, AHF is likely [8]. The 2017 American College of Cardiology (ACC)/American Heart Association (AHA)/Heart Failure Society of America (HFSA) guideline updates provide a Level IA recommendation that natriuretic peptides are useful to support the diagnosis or exclusion of AHF, similar to the 2013 guidelines [9,10]. However, the literature behind their use in the ED is controversial.

Pearl: Natriuretic peptides should only be used in conjunction with clinical evaluation, rather than using the test in isolation. Acknowledgement of other causes of elevated levels is essential.

Approximately one-quarter of patients with dyspnea will fail to demonstrate definitive levels of the biomarker, creating difficulty in interpretation of the test [13-20]. One of the first observational studies included 1586 patients with dyspnea, finding sensitivity of 90% for BNP of 100 pg/mL, with specificity 76% [18]. Authors stated BNP levels were more accurate than any history or examination finding. However, emergency physicians were correct in their diagnosis of AHF in over 95% of cases if they were sure of the diagnosis, and they were correct 92% of the time if they were sure AHF was not the cause of symptoms [18]. Another analysis suggested that emergency physicians had a sensitivity of 49% and specificity of 96% for diagnosis, while BNP 100 pg/mL had a sensitivity of 90% and specificity of 73% [19]. Though authors state BNP may have corrected physician diagnosis, there is no discussion for patients in whom BNP was incorrect. Cardiology diagnosis, the gold standard, was 90% accurate for diagnosis of AHF, and cardiologists disagreed on diagnosis in 11% of cases [19]. The RED-HOT trial suggested BNP level was correlated with 90-day mortality and need for readmission in AHF [20]. However, close evaluation of the area under the curve for 90-day outcomes was 0.67 for BNP, which is poor, suggesting no additional benefit [20]. A systematic review and meta-analysis suggested a pooled sensitivity of 95% and pooled specificity of 63% if a cutoff of 100 ng/L was utilized, while NT-proBNP cutoff of 300 ng/L demonstrated pooled sensitivity and specificity 99% and 43%, respectively [21]. Another metaanalysis found sensitivity and specificity of 93.5% and 52.9%, respectively, for BNP, and 90.4% and 38.2%, for NT-proBNP, respectively [22]. This meta-analysis suggested BNP levels are better than isolated history and examination findings, but included studies demonstrated several weaknesses including poor gold standard (typically cardiologist opinion), and few looked at emergency physician judgment [22]. In summary, observational data suggest BNP and NT-proBNP possess high sensitivities for AHF, but moderate to poor specificity. When emergency physicians are less certain of the diagnosis, natriuretic peptides demonstrate less accuracy, and it is not clear that BNP can outperform clinical iudgment.

Randomized controlled trial (RCT) data also differ in outcomes. One study from 2004 suggested fewer in-hospital admissions and ICU admissions, as well as lower cost, when utilizing natriuretic peptides [23]. No differences in mortality or readmission were found; however, physicians in this study were not blinded, and objective outcomes were not different when utilizing BNP. The IMPROVE-CHF study evaluated NT-proBNP, with decrease in length of stay by 0.7 h and decreased total costs [24]. However, no differences in hospitalizations, readmission rate, hospital LOS, or ICU admissions were found, with a nonsignificant increase in mortality when using NT-proBNP. A marginal improvement in diagnostic accuracy was found when BNP was added to clinical assessment, while BNP alone was not better than clinical gestalt alone [24]. Another study found decreased hospital LOS (two days), but no difference in hospitalization rate or ED LOS [25]. Several more recent RCTs suggest no difference in clinical outcomes such as mortality, readmission, or hospital LOS [26-30].

Other causes of elevated natriuretic peptides include coronary syndromes, valvular heart disease, pericardial disease, atrial fibrillation, cardiac surgery, cardioversion, older age, anemia, renal failure, pulmonary hypertension, critical illness, sepsis, and burns [21-23]. Age, gender, and body weight/body mass index can affect BNP levels. Due to less myocardial stress, obese patients may demonstrate lower BNP and NT-proBNP [31,32].

In isolation, BNP may outperform other history and examination features for AHF diagnosis, though it may not outperform overall clinical impression. For patient-oriented outcomes, studies are not definitive, as several suggest a decrease in admission, cost, and LOS, while others suggest no difference in these outcomes or patient-centered outcomes such as mortality [26-30]. Cutoffs vary, and there is significant lack of blinding and spectrum bias present in studies evaluating BNP.

2.2. Misconception: chest radiograph is the go-to imaging test in AHF

Patients presenting with suspected AHF undergo a variety of tests, including chest X-ray. This test is an important component of the overall assessment of patients with suspected AHF, with a variety of findings [8-12,22]. However, chest X-ray findings are not definitive [22]. Kerley B-lines demonstrate a sensitivity of 9.2% and specificity 98.8%, interstitial edema sensitivity 31.1% and specificity 95.1%, cephalization sensitivity 44.7% and specificity 94.6%, alveolar edema sensitivity 5.7% and specificity 89.2%, pleural effusion sensitivity 16.3% and specificity 92.8%, and cardiomegaly sensitivity 74.7% and specificity 61.7% for AHF [22]. Though the test may be specific, it is not sensitive, as close to 20% of chest X-rays demonstrate no findings of AHF [9-12,22]. Chest X-ray may suggest an alternative diagnosis such as chronic obstructive pulmonary disease, pneumonia, or pneumothorax.

Pearl: A more valuable means of diagnosis for pulmonary edema associated with AHF is ultrasound.

Point-of-care ultrasound (POCUS) is a vital tool in the diagnosis and management of several critical conditions, including AHF. Reliance on chest X-ray and laboratory assessment may result in delays in diagnosis and treatment, and POCUS can provide clinicians with a means of more reliable and rapid diagnosis, while also considering potential etiologies and mimics of AHF. POCUS may include evaluation of several components, including the lungs, heart, and inferior vena cava (IVC), with several protocols available [22,33,34]. Lung US alone with the presence of \geq 3 B lines in \geq 2 bilateral thoracic lung zones possesses a positive likelihood ratio (+LR) of 7.4, sensitivity approaching over 90%, and specificity 92.7% for pulmonary edema, while the absence of B lines possesses a negative likelihood ratio (-LR) of 0.16 [22,35-37]. Figs. 1 and 2 demonstrate B lines suggestive of pulmonary edema. The number of B lines correlates with AHF severity [38,39]. Measurement of intravascular volume is completed through assessment of the IVC diameter and percentage change in diameter while breathing [22,33]. However, specific numbers vary for IVC collapsibility index, including 20%-50%. IVC collapsibility <33% is associated with sensitivity approaching 80% for volume overload, with specificity 81%-87% [39-42]. IVC assessment is complicated by other conditions such as tricuspid regurgitation, pulmonary hypertension (pulmonary embolism), and right ventricular myocardial infarction [22,39-42]. Assessment of cardiac function can assist, measuring the inward movement of the interventricular septum and inferior wall of the LV in systole and degree of excursion of the anterior mitral valve leaflet in diastole [22,33,34]. A reduction in LV function on POCUS by emergency clinicians demonstrates a sensitivity for AHF 77-83% and specificity 74-90% [22,39,40]. A quantitative measure includes *E*-point septal separation (EPSS), which is the distance between

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