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The Role of the Emergency Department in the Patient with Acute Heart Failure

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Abstract The diagnosis and management of the patient with acute decompensated heart failure (ADHF) presents a unique challenge to the emergency medicine (EM) physician. ADHF is one of the most common cardiac emergencies managed in the emergency department (ED). ED presentations for ADHF will grow as survival rates after myocardial infarction continue to increase and thus, the incidence and prevalence of heart failure (HF) increases. There are very little data to aid EM physicians when trying to identify low-risk patients who are safe for ED discharge and observation units are not yet universally utilized. This results in 80 % of patients with ADHF getting admitted to the hospital. The aim of this review is to evaluate current strategies for diagnosis, treatment, and disposition of the ADHF patient in the ED while highlighting new approaches for treatment and disposition, and areas in need of additional research.

Keywords Heart failure · Emergency department · Observation unit

Introduction

Overall care for heart failure (HF) presents a significant burden on the US healthcare system. The prevalence and incidence are estimated at 5,700,000 and 670,000, respectively [1]. HF patients require chronic management but the natural course of HF inevitably leads to the need for urgent management for acute decompensation. While the management of chronic HF in the outpatient setting is well studied and its associated morbidity and mortality well documented, the emergency department (ED) management of acute decompensated heart failure (ADHF) presents specific challenges. The need for further research and standardization of management in patients with ADHF is important. ED presentations for ADHF will grow as survival rates after myocardial infarction increase and thus, the incidence and prevalence of HF increases.

HF patients live on a continuum between a stable, chronic state and acute decompensation. ADHF has multiple precipitants and etiologies, making ED management of the patient with ADHF challenging. Given the current model for healthcare in the United States, the ED is one of the few locations equipped to assess and treat the ADHF patient. ADHF is one of the most common cardiac emergencies managed in the ED. There are over 670,000 visits to the ED annually; this entails 21 % of all HF related visits according to the National Ambulatory Medical Care Survey [2]. Of those patients seen in the ED for ADHF, over 80 % are admitted to the hospital for further management [3].

Such a high admission rate raises the question of whether there may be a safe alternative for the ED patient with ADHF which avoids admission. Less than 10 % of ED patients with ADHF are severely ill [4–6]. Though the majority of patients are clinically stable, the high post-discharge event rate and lack of guidance regarding safe ED discharge results in high hospital admission rates. Thus, the decision to admit an ED patient with ADHF is often not based on acute severity of disease. Instead, it is largely a function of medical comorbidities and the uncertainty regarding near-term events. Previous work suggests over 50 % of such patients would be appropriate for a brief period of observation and treatment aimed at avoiding inpatient admission. However, the limited data in those discharged directly from the ED suggests they have a high rate of adverse events [7, 8]. Thus, a safe alternative to hospital admission is a critical unmet need.

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The diagnosis and management of the patient with ADHF presents a unique challenge to the emergency medicine (EM) physician. The management of this patient population requires a balance between achieving hemodynamic stability, improving symptomatology, and decision making to minimize morbidity and mortality. While it is fairly easy for the EM physician to identify those patients who are hemodynamically unstable or critically ill, it is sometimes difficult to identify those patients with ADHF who require hospitalization versus those who can be discharged after a brief period of observation. There are very little data to aid EM physicians when trying to identify low-risk patients who are safe for ED discharge.

Diagnosis of ADHF in the ED

History and Physical Exam

In general, patients with ADHF present with complaints that are a direct result of systemic congestion. One of the cardinal chief complaints of a patient presenting with ADHF is dyspnea. It is important to clarify acute precipitants, as well as the quality and extent of the dyspnea. Dyspnea on exertion (DOE), paroxysmal nocturnal dyspnea (PND) and orthopnea are all common symptoms. However, a large meta-analysis suggests PND and orthopnea are not very helpful symptoms, as they have intermediate likelihood ratios (LRs) of 2.6 and 2.2, respectively, for a diagnosis of ADHF [9]. Similarly, complaints of edema and fatigue have poor LRs and are not helpful when evaluating ED patients for possible ADHF. Prior episodes of HF, with a LR of 5.8, may be a helpful historical finding when present. The time course of symptom onset may help the clinician delineate the precipitant which has led to the acute presentation. Patients who present with a more insidious onset of symptoms (>48 hours) may be more likely to show signs of volume overload and peripheral edema [10••], suggestive of either gradually worsening underlying heart failure, or medication or dietary indiscretion. Abrupt onset of symptoms often suggests poorly controlled blood pressure, an arrhythmia or an acute coronary syndrome (ACS). Overall, historical features are of limited utility in the ED evaluation of patients with possible ADHF. The physical exam findings most suggestive of ADHF include a third heart sound (S3) and jugular venous distention (JVD), which have LRs of 11 and 5.1 respectively for ADHF [9]. While an S3 and JVD are classic signs of ADHF, their low sensitivity limits their utility as a screening test, and the inability to detect these physical exam findings does not rule out ADHF. This is especially true in a patient with a classic presentation of ADHF where physical examination is limited due to obesity or background environmental noise from the ED. While bibasilar rales and peripheral edema on physical exam are helpful, they are less specific for ADHF, and have intermediate LR's of 2.8 and 2.3, respectively [9].

Vital signs can also be helpful in determining the etiology of ADHF as well as guiding initial management and therapy. Diastolic dysfunction is often associated with hypertension and preserved ejection fraction [11]. However, both those with systolic and diastolic dysfunction can present with elevated blood pressure [12]. Tachycardia can also be seen as the body's way of trying to improve cardiac output. If bradycardia is encountered, the EM physician should consider complicating precipitants that may have led to the ADHF presentation, including heart block, drug toxicity (specifically digoxin), and electrolyte abnormalities including hyper-/hypokalemia.

Ancillary Diagnostic Tools

In addition to physical exam and history, standard work-up for suspected ADHF includes a series of diagnostic tests such as chest x-ray, 12-lead EKG, cardiac enzymes (specifically cardiac Troponin), and b-type natriuretic peptide (BNP) or N-terminal (NT)-proBNP. Other laboratories that are typically ordered include a complete blood count, creatinine, blood urea nitrogen (BUN), and electrolytes. The chest x-ray is a cornerstone in the evaluation of ADHF. Pulmonary and interstitial edema are highly suggestive of ADHF, both with LRs of 12.0 [9]. Cardiomegaly, with a LR of 3.3, may be a finding that is more synonymous with chronic HF rather than AHDF, and for that reason it is less helpful when making a diagnosis. While chest radiograph is helpful, there may be an absence of any findings of congestion in 15-18 % of ED patients with ADHF [13].

BNP and NT-proBNP have utility in certain clinic scenarios where there may still be uncertainty about the diagnosis [14]. These biomarkers are released from myocytes secondary to myocardial stretch and increasing end diastolic pressures [15, 16]. Elevation of these biomarkers and the degree of elevation are both indicators of the presence and severity of illness. The Breathing Not Properly trial suggests that BNP levels have diagnostic utility especially when there is intermediate probability or uncertain clinical suspicion for ADHF. In cases of intermediate probability for ADHF, BNP identified 74 % of patients correctly. BNP had a sensitivity of 90 % and specificity of 76 % for ADHF diagnosis [14]. NT-proBNP has also demonstrated a similar diagnostic utility [17]. The dyspneic patient with a plasma BNP level of less than 100 pg/dl or NT-proBNP of less than 300 pg/dl is unlikely to have ADHF. The caveat to using both BNP and NT-proBNP is that in certain patient populations these biomarkers can be falsely high or low. Both are falsely elevated in renal insufficiency, NT-proBNP more than BNP [18]. On the other hand, obesity and insulin insufficiency appear to result in falsely low BNP

levels. The mechanisms are not well understood but proposed hypotheses include metabolism of BNP by adipose tissue, reduced synthesis or inhibition of the peptide [19, 20].

A 12-lead EKG is important to identify treatable conditions such as cardiac ischemia and arrhythmias. Atrial fibrillation in the setting of dyspnea is the most common arrhythmia in patients with ADHF [9]. However, new T-wave changes or any abnormal EKG can also be associated with ADHF. A normal EKG decreases the likelihood of ADHF [9].

Evolving Diagnostic Strategies

Echocardiography has traditionally been a test that was reserved for inpatients undergoing further evaluation. However, with the advent of portable ultrasound devices with high resolution, as well as advanced training for EM physicians, the utility of non-invasive imaging in the ED continues to evolve. While a traditional echocardiography exam is not feasible in many ED patients, there is evidence suggesting focused bedside cardiac ultrasonography performed by emergency physicians may add useful diagnostic information during the initial evaluation of the ADHF patient. After initial management and stabilization of the patient with suspected ADHF, limited bedside echocardiography can give the EM physician insight into the patient's cardiac mechanics. The use of mitral valve E point septal separation (EPSS) in M-mode ultrasonography has been established as a valid way to assess for reduced ejection fraction; EPSS has a strong negative correlation with ejection fraction. It is 87 % sensitive for detecting an abnormal ejection fraction at a cutoff of greater than 7 mm [21]. A recent study showed that even junior EM physicians (PGY3/4) are able to obtain EPSS measurements that closely correlate with the visual estimates of ejection fraction made by clinicians, both EM and cardiologists, with extensive echocardiography and clinical experience [22].

Assessing for B-lines, also known as ultrasound comet tail artifacts, is an additional diagnostic tool readily and easily accessible to the EM physician. B-lines arise from water-thickened interlobular septa at the pleural line. Assessing for B-lines can help differentiate between cardiogenic and noncardiogenic causes of dyspnea. The technique is simple, reproducible and fast; it involves assessment of the anterior and lateral chest wall from the second to fifth intercostal spaces [23]. Three or more B-lines in one viewing field (Fig. 1) are considered a positive finding for pulmonary edema [24]. One study suggests that B-lines have similar diagnostic utility to biomarkers. NT-proBNP and ultrasound were used to evaluate 149 patients who presented with acute dyspnea. B-lines were found in 93 of the 122 patients who were found to have a cardiogenic etiology of dyspnea. The negative predictive value was higher for NT-proBNP (100 %) than B-lines (45 %) but the positive predictive value was higher for B-lines (97 %) than NT- proBNP (92 %).

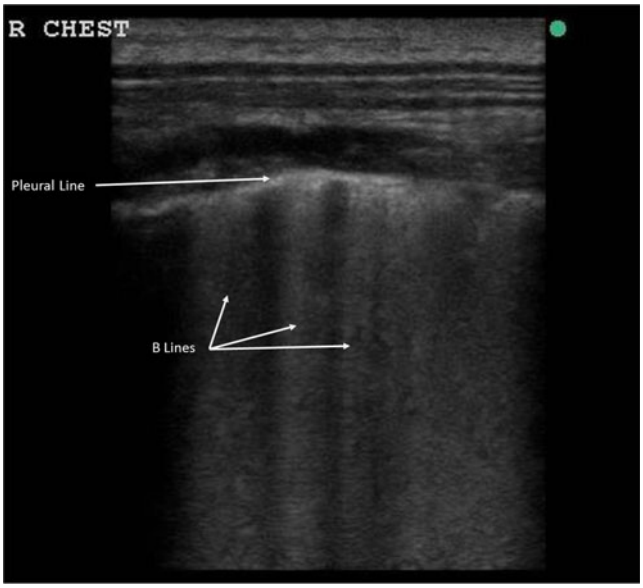

Fig. 1 Ultrasound B-lines (courtesy of Robinson Ferre, MD)

Preliminary studies of noninvasive monitoring of left ventricular end diastolic pressure (LVEDP) suggest it may have utility in the hospitalized ADHF patient. In a small prospective trial of patients admitted with ADHF, patients were randomized to treatment based upon LVEDP monitoring or clinical assessment. The patients in the LVEDP group had targeted management to achieve LVEDP <20 using the VeriCor monitor. The VeriCor monitor is a noninvasive hemodynamic monitor that measures LVEDP. Using left heart catheterization as the gold standard, the predictive accuracy of the VeriCor monitor for LVEDP was superior to pulmonary capillary wedge pressure measured by right heart catheter [25]. Endpoints of this analysis were LVEDP at discharge and rehospitalization rate; both of which were significantly reduced in the group with management guided by LVEDP [26]. The utility of LVEDP monitoring in the ED has not been extensively studied; however, given that there are certain patient populations that may be able to be discharged from the ED after a brief period of observation, a noninvasive measure of LVEDP may be a useful tool to guide ED management [26].

Treatment

General Approach to Therapy

In the ED it is paramount to address clinical stability at the beginning of the patient encounter. Patients with ADHF who present in extremis require stabilization, and hemodynamic abnormalities or respiratory distress should be immediately addressed. Airway maneuvers may be required, such as endotracheal intubation or non-invasive positive pressure

ventilation (NIPPV). The greatest benefit of NIPPV is improving dyspnea, reducing preload, and avoiding intubation [27]. A large randomized study suggests that while it improves work of breathing, it may not have an impact on mortality [28]. Both continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP) have been found to be useful [29]. These modalities require patient cooperation and have limited use in the obtunded patient.

Diuretics remain a cornerstone of ADHF therapy. While they have been guideline-recommended therapy, until recently this was largely based on consensus recommendations, as there had been no randomized trials. However, a recently completed small randomized trial gives us some guidance for initial diuretic dosing. In a prospective double-blind, randomized trial, 308 patients with ADHF received IV furosemide by means of a bolus every 12 hours or as a continuous infusion, further stratified by either high- or low-dose [30]. The primary endpoints were patients' global assessment of symptoms and change in creatinine from baseline. Results showed no significant difference in patients' global assessment of symptoms or mean creatinine with bolus versus continuous infusion. There was a trend to slightly increased improvement in dyspnea and greater diuresis in the high-dose diuretic group, with a small increased risk of worsening renal function [30]. This trial suggests that while higher diuretic doses may have a slightly increased risk of worsening renal function, physicians can choose a wide range of diuretic doses to aggressively treat their patients. Based on this data, a reasonable starting dose for ED patients with ADHF is 1-2 times their total daily dose. For example, if the patient takes furosemide 80 mg two times daily, the initial ED dose would be 80-160 mg intravenously. In those patients who are diuretic-naïve, 40-80 mg intravenously would be a good initial dose.

Vasodilators, including nitrates and IV angiotensin-converting enzyme (ACE) inhibitors, are highly utilized in the HF patient. Nitrates are recommended as initial therapy for ADHF, especially in the setting of hypertension or pulmonary edema. Prior studies suggest high-dose nitroglycerin is associated with less need for intubation, bipap, and ICU admission [31–33]. Patients with ADHF and significant hypertension (systolic blood pressure [SBP] $\geq$160 mmHg) have benefitted from aggressive intravenous vasodilation. While commonly used in the IV form, ED physicians also frequently use sublingual or topical nitroglycerin. Nesiritide, a recombinant B-type natriuretic peptide (BNP) with vasodilatory properties has also been approved for use in the ADHF patient. Early trials suggested it was efficacious and produced hemodynamically favorable results. However, subsequent secondary analyses suggest it may worsen renal function and adversely impact mortality. For those reasons, the ASCEND-HF trial was designed to definitively test nesiritide's impact on mortality. This was a randomized, double-blind placebo-controlled study of 7141 hospitalized patients who either received nesiritide or placebo for 24 to 168 hours in addition to standard therapy. Standard therapy was administered at the discretion of the investigators and included diuretics, morphine and vasoactive medications [34]. Nesiritide was found to have no significant effect on the rate of death, rehospitalization, or renal function. It had a small effect on dyspnea but was associated with an increased risk of hypotension. This trial disproved previous studies that suggested it caused worsening renal function but also brings into question the efficacy of nesiritide in the treatment of the ADHF patient [35]. Given the results of this study, nesiritide should be considered a second line agent when treating the ED patient with ADHF. Relaxin, a naturally occurring human peptide with vasodilatory effects, was evaluated in a randomized study in ADHF patients who were enrolled within 16 hours of ED presentation, had SBP$\geq$125 mmHg and moderate renal dysfunction. The results of this study suggest Relaxin may safely improve dyspnea, minimize ongoing myocardial and renal injury, and perhaps improve long-term events. This study enrolled patients much earlier than prior ADHF studies, which may be an explanation for the promising results. Importantly, early enrollment suggests this therapy may have a role in ED patients with ADHF, but further study is necessary [36, 37].

Many patients with a history of HF take beta-blockers as part of their outpatient HF regimen. But what is their role in the setting of acute decompensation? ACC/AHA guidelines for the use of beta-blockers in the acute setting include (1) use in patients with reduced ejection fraction who are hemodynamically stable but have acute decompensation and are on beta-blockers as an outpatient, and (2) initiation of beta-blockade after stabilization and volume optimization of the ADHF patient who is not otherwise on beta-blockers as an outpatient [38]. Beta-blockers are not routinely recommended in hypotensive, bradycardic or hemodynamically unstable patients and should be held in these patients; however, they do have a role in HF and should be resumed or initiated after euvolemia and stability are achieved [39]. In the current model for management of acute decompensation in the ED, beta blockers are not routinely initiated, but ideally are resumed or initiated at the time of clinical stability prior to hospital discharge.

Tailored Therapy

The EM physician must identity and treat the patient with ADHF, focusing on the acute symptoms and possible treatable precipitants. ADHF presentations are heterogeneous and may require individualized therapies. Patients typically fall into one of three categories based on initial hemodynamics: hypertensive, normotensive or hypotensive.

More than 50 % of patients will present with SBP greater than 140 [13, 40]. These patients typically present with more severe symptoms, often within 24-48 hours of symptom onset,

and may not have evidence of peripheral edema but rather signs of pulmonary edema. While they often appear acutely ill, if treated appropriately they can have a brisk symptomatic response to therapy [33]. The goal of therapy in these patients is to reduce preload and afterload, which decreases systemic and pulmonary congestion. Vasodilators are the mainstay of treatment in hypertensive patients with ADHF. Sublingual or topical nitroglycerin, ACE-inhibitors, and IV hydralazine are all useful in reducing blood pressure in these patients [41–43]. While diuretics may also be used, great care should be taken to not over-diurese these patients as their symptoms are often secondary to intravascular shifts in volume leading to pulmonary edema, not necessarily whole body volume overload [44, 45]. Patients with ADHF who present with hypertension have a lower 60- to 90- day mortality than those patients who present normotensive [10••, 40].

The normotensive patient with ADHF often benefits from diuresis and total body fluid reduction. These patients typically present more insidiously with symptoms which are a result of gradual fluid overload. They often have systolic dysfunction and benefit from more aggressive diuresis. However, it is important to consider that these patients often run out of blood pressure "reserve", limiting diuresis. The hypotensive patient is rare, as less than 10 % of patients present with hypotension or cardiogenic shock [5, 6]. Patients who present with hypotension or in extremis have poor outcomes, including increased in-hospital mortality [40]. These patients should be triaged rapidly to an inpatient bed as they may require aggressive time-sensitive therapies including intravenous fluids, inotropes and possibly mechanical cardiovascular support.

Overall, the management of the ADHF patient in the ED is multifactorial and based on several factors including their blood pressure, comorbidities, precipitants, and clinical severity. Table 1 summarizes the multifaceted approach to therapy based on these factors.

Risk-Stratification and Disposition

The ADHF patient is challenging for EM physicians as there is limited data and few clinical tools for disposition decision making. The default disposition strategy is inpatient admission for the majority of ED patients with ADHF. Worsening symptoms of chronic HF account for the majority of patients who present to the ED and are admitted to the hospital. While these patients do not appear critically ill, they are still at high risk for short-term adverse events [10••]. There is no standard model or algorithm for patient disposition. This is likely both a result of the complexity of the ADHF patient population but also the variability in both the clinical approach and resource availability between institutions. Inpatient admission allows time for observation, appropriate patient education, close follow-up (when it cannot otherwise be obtained), and therapeutic interventions.

There are several studies over the last decade highlighting characteristics of high-risk groups who are in need of inpatient admission. For these patients, disposition is relatively straightforward. The high-risk patient includes those with: elevated BUN or creatinine, hyponatremia, evidence of myocardial ischemia, elevated BNP, elevated cardiac troponin or low blood pressure [40, 47, 48]. Low-risk groups are more elusive, yet may be the key to improving ADHF admission tendencies, by utilizing safe disposition alternatives to inpatient admission. One prospective cohort study demonstrated that ED triage physicians overestimate the probability of severe complications in the ADHF patient and as a result tend to over utilize critical care resources for admission [49]. This highlights one of the reasons many ADHF patients are admitted and supports the need for validated clinical prediction models to aid the EM physician with disposition. One such clinical prediction rule was retrospectively derived using administrative data to identify ED patients with ADHF who are at low-risk of death or serious medical complication. This endeavor produced a complex classification algorithm using 21 variables to describe patients at low risk for poor outcome [50]. This model was validated by applying this clinical prediction rule to a retrospective cohort of 8384 inpatients with a primary diagnosis of ADHF. Of this cohort, 1609 (19.2 %) were identified as low risk. Of those identified as low risk, 12 (0.7 %) died as inpatients, 28 (1.7 %) survived after a serious hospital complication, and 47 (2.9 %) died within 30 days of discharge [50, 51]. This study suggests that this rule may be helpful in identifying low risk groups who can be managed in an observation unit (OU) or discharged from the ED with close follow-up.

Is there a subset of patients who can be discharged safely from the ED with close follow-up? Who warrants 23-hour observation in an ED-based OU? Alternatives to admission are very dependent on the individual patient and the infrastructure of the healthcare system where care is being provided. Both hospital and outpatient resources are required for comprehensive treatment of the ED patient with ADHF. One ED disposition option is stabilization, medication adjustment and discharge home after arranging close outpatient follow-up within the next 72 hours. Previous data suggests patients with ADHF discharged from an inpatient admission in hospitals with higher early follow-up rates have a lower risk of 30-day readmission [52]. This is specific to stabilization after an inpatient stay but may suggest similar outcomes if close follow-up were established following discharge from the ED or OU. One of the crucial factors to such a process is ensuring availability of close follow-up with either a cardiologist or primary care

Table 1 ED clinical considerations in the ADHF patient

Clinical considerations			
Clinical factor	ED presentation & objective findings	Impact on management	Disposition
1) Blood pressure			
a. Hypotension (<5 %)	Shock/extremis, SBP<100	Airway support, fluids, inotropes	ICU
b. Normotension	Insidious onset (days, weeks)	Diuretics, vasodilators	ED Obs vs telemetry floor
c. Hypertension (~50 %)	Acute onset (24-72 hrs), SBP>140	Vasodilators 1st, then +/- diuretics	ED Obs vs telemetry floor
2) Comorbidities			
a. COPD	Pulmonary wheezing	Airway support, concurrent bronchodilators and steroids	Based on clinical severity
b. Obesity	Difficult physical exam, low BNP	Diuretics +/- vasodilators	
c. Renal disease	Chronically elevated BNP	Difficult diuresis, may require dialysis if in extremis	
3) Precipitants			
a. Ischemia	Chest pain	Vasodilators, anticoagulation +/- intervention	Based on clinical severity
b. Arrhythmia	May affect vital signs (HR, BP)	Treat arrhythmia (medical, electrical)	
c. Diet/Medication	Historical clue	Diuretics +/- vasodilators, reinitiating home medications when stabilized	
4) Clinical severity			
Respiratory Distress	Significant dyspnea/hypoxia	Consider NIV or intubation, aggressive vasodilation if hypertensive	ICU

Data from Collins et al. [13] and Gheorghiade et al. [46]

physician. A subset of patients in the current healthcare system do not have a primary care physician and rely on EDs for medication adjustment and acute treatment. This subset of patients would need to have an appointment scheduled with a new provider, possibly hindering their ability to be discharged directly from the ED or after OU management. Hospital resources dedicated to finding providers for these patients would be crucial for success.

Another alternative to ED discharge is the ED-based OU. ED physicians are skilled and well equipped to provide acute therapy for the first 24-48 hours for the ADHF patient, thus making ED-based observation a logical and economical means to care for this patient population [53]. Observation units were identified by the Institute of Medicine as central to improving resource use and patient flow. A recent study suggests increased OU utilization has the potential to save $3.1 billion and avoid 2.4 million inpatient admissions [54]. Appropriate OU utilization in ADHF management may contribute to this cost savings. While hospitalization assists with detection of occult disease, and is clearly an inflection point which marks a threshold independently predicting worse outcomes, the impact of hospitalization on post-discharge events has not been well elucidated [52, 55, 56]. Of the potential 50 % of patients who present with ADHF and have no high risk features, an ED-based OU may be a safe and appropriate alternative to admission to manage these patients and facilitate early discharge [57]. An ED-based OU has the potential to provide the resources necessary to monitor blood pressure, heart rate, urine output and weight during a 23-hour observation period, which is also adequate time for many patients to have near-complete resolution of symptoms with standard therapy [58]. Additional diagnostic testing can also be easily arranged in an OU setting, including formal echocardiography, electrolytes, and cardiac biomarkers.

Conclusions

ADHF is a dynamic and heterogeneous clinical entity. The economic burden of ADHF is significant and coordination of patient care begins in the ED. The decisions made by ED physicians regarding patient care often set the trajectory for patient disposition. Lack of research and investigation leaves many questions unanswered regarding optimal and efficient patient care in the ED as well as appropriate disposition. ED physicians have a unique set of skills and resources that allow them to immediately assess and treat the ADHF patient effectively, including the widespread use of ultrasound in the ED. In spite of these resources, the ED physician has little to aid him/her in deciding the ultimate disposition of the patient. The result is that most ED patients with ADHF are admitted to the hospital. However, available data, from mostly inpatient studies, would suggest that there is at least a portion of patients who may be suited for ED discharge, or a brief period of management in an ED OU. Our practices for treating ADHF have not changed significantly over the last 20 years. The cornerstones of ADHF therapy remain the same, including vasodilators and diuretics, which impart significant subjective improvement within 4-6 hours in the ED. However, the long term effects and interplay of both HF exacerbations and the subsequent treatment are not well studied. Given the growing rate of survival after myocardial infarctions, it is obvious that the prevalence and incidence of HF and ADHF will continue to grow. The need for an evidence–based, systematic approach regarding patient treatment and disposition from the ED is imperative. This research has implications for the clinical outcomes of hundreds of thousands of individuals affected each year with HF, as well as for the overall economic burden of HF on the healthcare system.

Conflict of Interest Courtney Fay Horton declares that she has no conflict of interest.

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