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REVIEW

Dissecting the ''CHF Admission'': An Evidence-Based Review of the Evaluation and Management of Acute Decompensated Heart Failure for the Hospitalist

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Acute decompensated heart failure (ADHF) is one of the most common conditions managed by hospitalists. Here we review the most recent evidence applicable to hospitalists for the diagnosis, risk stratification, and management of patients presenting with ADHF. By following a structured approach based on the patient's symptoms, history, physical examination, and laboratory testing, the clinician can make the diagnosis of heart failure efficiently. Because patients exhibit a wide spectrum of risk for adverse outcomes, both in the hospital and after discharge, assessing for clinical factors associated with these outcomes is essential. Congestion should be managed primarily with diuretics, and vasodilators may be helpful in certain patients. Given high rates of readmission, hospitalists should ensure that patients received evidence-based therapy, heart failure education is performed, and follow-up is in place before discharge. *Journal of Hospital Medicine* 2012;7:439–445. © 2012 Society of Hospital Medicine

Caring for patients with acute decompensated heart failure (ADHF) is one of the core competencies of practice in hospitalist medicine. ''Congestive heart failure'' remains the most common discharge diagnosis as recorded in the National Hospital Discharge Survey, with over 1.1 million hospitalizations for heart failure in 2004.[1] Furthermore, with the disproportionate growth in the population over age 65 that will occur over the next 20 years, heart failure prevalence will grow from its current value of 2.8% to 3.5% by 2030.[2] This will result in an additional 3 million Americans with chronic heart failure, thereby sustaining ADHF as the most common reason for hospital admission. Despite an average hospital stay of 5 days, the readmission rate for heart failure was 26.9% at 30 days in a 2003-2004 analysis of Medicare data.[3] This high readmission rate is the target of reform as part of the recently passed Patient Protection and Accountability Act. Starting in fiscal year 2013, acute-care hospitals with higher-than-expected readmission rates for heart failure will have a reduction in reimbursement for these admissions.[4] Thus, there is substantial incentive for hospitalists to focus on providing the highest quality of care for patients with ADHF. Here we review the most recent evidence applicable to hospitalists for the diagnosis, risk stratification, and management of patients presenting with ADHF.

DIAGNOSIS

The hospitalist can establish the ADHF diagnosis efficiently by applying a structured approach based on the patient's symptoms, history, physical examination, and laboratory testing. The typical symptoms of ADHF include dyspnea, orthopnea, paroxysmal nocturnal dyspnea (PND), and lower extremity edema. In particular, patients complaining of PND and/or orthopnea are likely to have ADHF.[5,6] Patients may also report chest congestion or chest pain in an atypical pattern. A history of rapid weight gain suggests fluid overload, hence determination of the patient's ''dry weight'' is important to establish a target for congestive therapy. Patients with advanced systolic heart failure may also complain of nausea, abdominal pain, and abdominal fullness from ascites.[7] In a patient with dyspnea, a history of heart failure, myocardial infarction, or coronary artery disease, all make the diagnosis of ADHF more likely.[5]

Performing a careful physical examination on a patient presenting with suspected ADHF will not only establish the diagnosis of heart failure, but also determine the hemodynamic profile. Patients presenting with ADHF can be separated into 4 hemodynamic profiles, based on vital sign and physical exam parameters: the presence or absence of congestion (''wet or dry''), and the presence or absence of adequate

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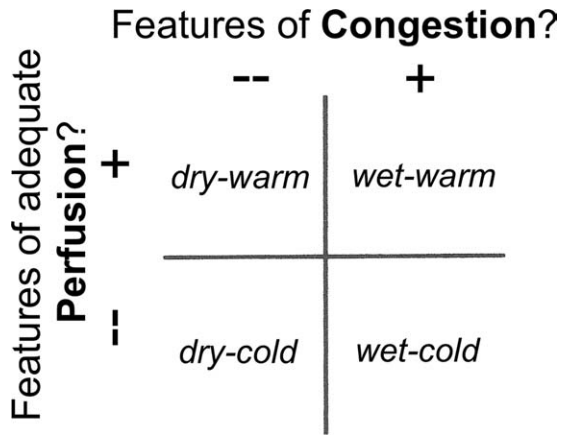

FIG. 1. The 4 hemodynamic profiles of patients with acute decompensated heart failure (ADHF). Patients presenting with ADHF can be separated into 4 hemodynamic profiles based on the presence or absence of adequate perfusion (left), and the presence or absence of features of congestion (top). This figure adapted from Nohria et al. with the permission of Elsevier Limited.[8]

perfusion (''warm or cold'') (Figure 1).[8] Parameters indicating the presence of congestion include: orthopnea, elevated jugular venous pulsation (JVP), lower extremity edema, hepatojugular reflux, ascites, and a loud P2 heart sound. Notably, rales are an uncommon physical finding in patients with ADHF, likely because pulmonary lymphatics compensate for chronically elevated filling pressures in such patients.[9,10] Parameters indicating inadequate perfusion include: hypotension (mean arterial pressure <60 mmHg), proportional pulse pressure <25%, cool extremities, altered mental status, and poor urine output (<0.5 mL/kg/hr). We recommend assigning the patient to 1 of these 4 hemodynamic profiles, as the profile correlates with invasive hemodynamic measurements of pulmonary capillary wedge pressure and cardiac index, guides management, and predicts outcome.

Natriuretic peptide testing may help establish or exclude a diagnosis of ADHF. A recent expert consensus paper on natriuretic peptide testing recommends cutpoints for both B-type natriuretic peptide (BNP) and N-terminal proBNP (NT-BNP) that indicate a very low (BNP <100 or NT-BNP <300), intermediate (BNP 100-400 or NT-BNP 300-1800), and high (BNP >400 or NT-BNP >1800) probability of heart failure[11] (Figure 2). However, 2 common conditions affect the utility of BNP testing. First, obese patients have lower levels, and thus a lower rule-out cutpoint of 54 pg/mL is recommended when using BNP, whereas the cutpoint for NT-BNP remains the same.[12,13] Second, in patients with renal dysfunction, levels are increased, and thus higher rule-out cutpoints of 200 pg/mL (for BNP) and 1200 pg/mL (for NT-BNP) are recommended for patients with a glomerular filtration rate <60 mL/min.[14,15] For patients with longstanding heart failure and chronically elevated levels of natriuretic peptides, there is a correlation between BNP levels and left ventricular filling pressure,[16] but the change is more helpful than the absolute levels; a 50% increase over baseline, in conjunction with symptoms, usually reflects ADHF.[11]

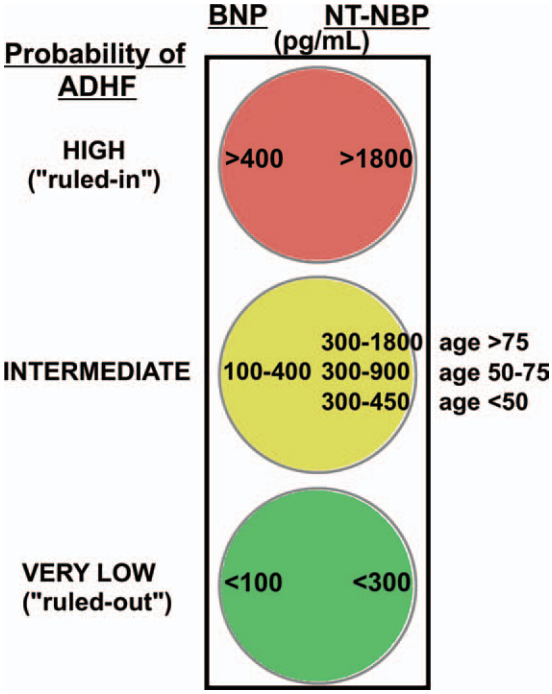

FIG. 2. Cutpoints for natriuretic peptide testing. In patients presenting with dyspnea, the levels of BNP and NT-BNP can help establish or exclude a diagnosis of ADHF. A BNP <100 or NT-BNP <300 correlate with a very low probability of ADHF, so the diagnosis is "ruled-out." A BNP of 100-400 or NT-BNP of 300-1800 (with the upper limit varying by age) is intermediate, so other clinical criteria should be used to establish or exclude the diagnosis. A BNP >400 or NT-BNP >1800 correlates with a high probability of ADHF. **Abbreviations:** ADHF, acute decompensated heart failure; BNP, B-type natriuretic peptide; NT-BNP, N-terminal proBNP.

Chest radiography will establish the presence or absence of pulmonary congestion. Classic teaching is that congestion starts with cephalization (pulmonary capillary wedge pressure 10-15 mmHg), progresses to Kerley B lines (15-20 mmHg), then to interstitial edema (20-25 mmHg), and finally to alveolar edema (>25 mmHg).[17] In patients presenting with dyspnea, any of these findings helps to establish the diagnosis of ADHF.[5]

MECHANISMS AND TERMINOLOGY

Data from ADHF registries show that hemodynamically stable patients presenting to the hospital with ADHF are an approximately equal mix of heart failure with reduced ejection fraction (HFrEF; ejection fraction <50%) and heart failure with preserved ejection fraction (HFpEF; ejection fraction ≥50%).[18,19] The important differences between these groups with regards to pathophysiology and etiology have been reviewed elsewhere.[20] Establishing the heart failure mechanism (ie, reduced or preserved EF) is important because the medical management is distinct. Patients with HFrEF are more likely to be male, younger in age, to have ischemic heart disease, and to present

TABLE 1. Clinical Criteria for the Application of Current Heart Failure DRG Codes to Patients With ADHF

ICD-9	DRG Code	Severity Subclass	Clinical Criteria	Hemodynamic Profile
Acute decompensated heart failure				
428.21	Systolic, acute	MCC	New diagnosis, clinical features of low-output or "cold" state, EF ≤30	Dry-cold
428.23	Systolic, acute on chronic	MCC	Established diagnosis, clinical features of low-output or "cold" state, EF ≤30	Dry-cold
428.41	Combined systolic and diastolic, acute	MCC	New diagnosis, clinical features of congestion, EF <50	Wet-warm or wet-cold
428.43	Combined systolic and diastolic, acute on chronic	MCC	Established diagnosis, clinical features of congestion, EF <50	Wet-warm or wet-cold
428.31	Diastolic, acute	MCC	New diagnosis, clinical features of congestion, EF ≥50	Wet-warm
428.33	Diastolic, acute on chronic	MCC	Established diagnosis, clinical features of congestion, EF ≥50	Wet-warm
Chronic heart failure				
428.22	Systolic, chronic	CC	No previous symptoms, or history of clinical features of low-output state but currently compensated, EF <50	Dry-warm
428.40	Combined systolic and diastolic, chronic	CC	History of clinical features of congestion but currently compensated, EF <50	Dry-warm
428.32	Diastolic, chronic	CC	History of clinical features of congestion but currently compensated, EF ≥50	Dry-warm
Other				
428.1	Left heart failure	CC	Clinical features of congestion, mechanism and EF is unknown	Wet-warm or wet-cold
428.20	Systolic heart failure, unspecified	CC	Clinical features of low-output, acuity is unknown	Dry-cold
428.0	Congestive heart failure, unspecified	NCC	Clinical features of right-heart failure	Not applicable

NOTE: From the clinical assessment, 3 pieces of information are needed: the acuity, the hemodynamic profile, and the ejection fraction. **Abbreviations:** ADHF, acute decompensated heart failure; CC, complication; DRG, Diagnosis Related Group; EF, ejection fraction; ICD-9, International Classification of Diseases, Ninth Revision; NCC, non-complication; MCC, major complication.

with normal or low blood pressure. Patients with HFpEF are more likely to be female, older in age, to have hypertension or diabetes mellitus, and to present with elevated blood pressure.[18,19]

The terminology used for inpatient heart failure coding has been the subject of renewed focus. For fiscal year 2008, the Centers for Medicare and Medicaid Services (CMS) overhauled its Diagnosis Related Group (DRG) system to better account for the severity of illness of hospitalized patients.[21] In this revision, the existing DRG codes for heart failure were subdivided into 3 severity subclasses: major complication, complication, and non-complication. Payment to hospitals for a heart failure DRG was changed to be proportional to the level of complication. Thus, for the first time, the clinicians' assessment of the acuity of heart failure determines the level of payment to the hospital. Not surprisingly, this has led to initiatives by hospitals to improve clinicians' coding of inpatients hospitalized with heart failure. A major impediment is that there are no established criteria for the application of each DRG code. Table 1 presents recommended clinical criteria for the application of these codes to patients with ADHF.

PRECIPITANTS AND ETIOLOGY

For patients presenting for the first time with a diagnosis of ADHF (de novo), a thorough evaluation should be performed to determine the mechanism and etiology of the patient's left ventricular dysfunction. After the initial history and physical exam, the American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommend checking basic laboratory studies, an electrocardiogram, and an echocardiogram.[22] The full assessment recommended by the ACC/AHA is detailed in Supporting Online Table 1 (in the online version of this article). Cardiac ischemia is the most common etiology of HFrEF, accounting for about 50% of cases. The common, non-ischemic causes of systolic heart failure include atrial fibrillation, aortic stenosis, illicit cardiotoxic drugs (cocaine, methamphetamine), medical cardiotoxic drugs (adriamycin), as well as primary myocardial disorders such as myocarditis, idiopathic, or peripartum cardiomyopathy. HFpEF is most commonly associated with long-standing hypertension and diabetes mellitus, but can also be caused by infiltrative, hypertrophic, and constrictive cardiomyopathies.

For patients with a history of heart failure, it is important to identify the precipitant for the decompensation, as it may be treated or avoided in the future. When no clear precipitant is identified, this is most concerning, as it indicates the patient's tenuous cardiac function. In the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry, approximately 61% of patients were found to have at least 1 precipitating factor.[23] The most common precipitants were respiratory process in 15.3%, acute coronary syndrome in 14.7%, arrhythmia in 13.5%, uncontrolled hypertension in 10.7%, medication non-compliance in 8.9%, worsening renal function in 8.0%, and dietary non-compliance in 5.2%.

RISK STRATIFICATION

Patients hospitalized with ADHF are at a significantly elevated risk for death, both during their hospitalization and after discharge. Numerous studies have shown that multiple clinical parameters assessed during the hospitalization, such as vital signs and laboratory values, predict outcome.[6,8,24,25] Some of the most elegant parameters are physical exam findings. As introduced above, the "wet-cold" hemodynamic profile assessed at admission predicts increased mortality

TABLE 2. Indications for Cardiology Consultation in Patients with ADHF

Results of Evaluation	Indication for Referral	Purpose of Referral
Hypotension, "cold" hemodynamic profile	Inadequate perfusion	Pulmonary artery catheterization, inotropic therapy
Ischemic symptoms, positive troponin, abnormal ECG, echocardiogram with focal wall motion abnormalities	Cardiac ischemia	Coronary angiography and coronary intervention if indicated
Atrial fibrillation	Arrhythmia	Consideration of a rhythm control strategy
Ejection fraction ≤35%	Severe left ventricular systolic dysfunction	Implantable cardiac defibrillator and/or biventricular pacemaker
High diuretic dose requirements or decreasing urinary response to diuretics	Diuretic resistance	Consideration for vasodilator therapy or ultrafiltration
Increasing blood urea nitrogen and serum creatinine, decreasing urine output	Worsening renal function	Consideration of inotropic therapy

Abbreviations: ADHF, acute decompensated heart failure; ECG, electrocardiogram.

and urgent transplantation at 1 year.[8] One of the most powerful risk stratification schemes for in-hospital mortality is that developed from the Acute Decompensated Heart Failure (ADHERE) national registry. Three clinical parameters, blood urea nitrogen (BUN) >43 mg/dL, systolic blood pressure <115 mmHg, and serum creatinine >2.75 mg/dL, stratified patients into risk groups. Patients exhibiting all 3 parameters had a 22% in-hospital mortality compared with 2% for patients with none of the 3 parameters.[24]

BNP and troponin also have a role in risk stratification of patients with ADHF. In the ADHERE registry, for every increase in the BNP of 400 pg/mL, the odds of risk-adjusted mortality increased by 9%, in patients with both HFrEF and HFpEF.[26] Similarly, an elevated admission troponin was associated with an in-hospital mortality of 8.0%, versus 2.7% for troponin-negative patients[27]; notably almost half of patients with a positive troponin had no history of ischemic heart disease. In the future, refinement and widespread application of these risk stratification methods should allow clinicians to triage patients to determine their location (eg, observation unit, inpatient, intensive care unit) and type of treatment (eg, oral or intravenous diuretic, vasodilator, inotrope).[28]

In the community, hospitalists care for many patients with ADHF without input from a cardiologist.[29] However, there are several situations where the patient is at an increased risk of adverse outcomes, and therefore in which we recommend consulting a cardiologist (Table 2). Patients with hypotension, a "cold" hemodynamic profile, or worsening renal function due to poor cardiac function are at an especially elevated risk and should be considered for advanced therapies such as mechanical circulatory support or heart transplantation.

CONGESTION AND DIURESIS

The syndrome of heart failure is due primarily to elevation of left ventricular filling pressures resulting in congestion, and therapies aimed at reducing congestion are of primary importance.[30] For 50 years, treatment with diuretic medications has been the mainstay of therapy for patients admitted with ADHF. Vasodilator agents, specifically nitroglycerin and sodium nitroprusside, may also be beneficial in patients presenting with ADHF and hypertension.[22] In 1 study, patients with acute pulmonary edema treated with high-dose nitroglycerin experienced fewer adverse events as compared to those treated with high-dose diuretics alone, suggesting that nitrates can more rapidly decrease congestion and thereby improve outcomes.[31] Unfortunately vasodilators are underutilized, with only 5.8% of patients with elevated blood pressure (>160 mmHg) treated with nitrates in the OPTIMIZE-HF registry.[9]

Diuretic choice, dosing, and administration method have traditionally been highly variable between practitioners. Oral diuretics are generally not preferred initially for patients with ADHF because of concerns of inadequate absorption from an edematous bowel and slow onset of action.[32] For a patient who is not on diuretics as an outpatient, an initial dose of 40 mg intravenous furosemide is reasonable. For a patient with chronic heart failure on outpatient loop diuretic therapy, the Diuretic Optimization Strategies Evaluation (DOSE) study provides insight into diuretic dosing and administration. Patients were randomized to an administration route (bolus dosing every 12 hours or continuous infusion) and a dosing strategy (low-dose or high-dose).[33] There were no differences in the primary endpoint of patient-reported global assessment of symptoms, or the primary safety endpoint of change in serum creatinine from baseline to 72 hours between the bolus and continuous infusion groups or between the low-dose and high-dose groups. However, patients in the high-dose group had decreased dyspnea at 72 hours, decreased body weight at 72 hours, increased fluid loss at 72 hours, and decreased NT-BNP at 72 hours. These improvements came at the expense of a mild increase in creatinine. Therefore, in hospitalized patients with ADHF on outpatient furosemide, these data support initiation of high-dose furosemide with a daily intravenous dose equal to 2.5 times their daily outpatient oral dose, using either bolus or continuous infusion.

All patients being treated with diuretic therapy should have close fluid intake and output monitoring, fluid restriction of 1500 to 2000 mL per day, a 2 gram sodium diet, and at least daily electrolyte monitoring. For patients with inadequate diuresis (generally less than 1 L per day in a patient with moderate volume overload), several options are available. If the

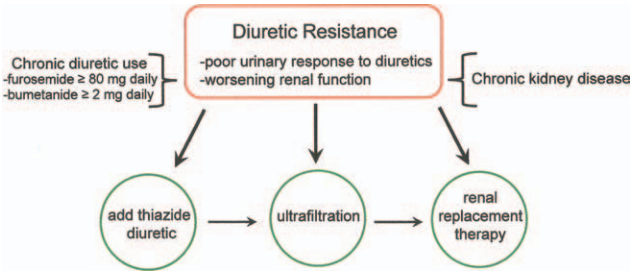

FIG. 3. Strategies for management of diuretic resistance. Thiazide diuretics can be added to loop diuretics for enhanced diuresis. For patients with severe fluid overload, ultrafiltration may be beneficial. In the setting of worsening urine output and renal function, renal replacement therapy may be needed.

urinary response to a furosemide dose is inadequate, the dose should be doubled and the urinary response followed. If there has been inadequate diuresis in a patient with a low serum albumin or significant proteinuria, furosemide should be switched to bumetanide, which is not protein-bound and thus will achieve higher concentrations in the tubule.[34]

Longstanding treatment with loop diuretics leads to decreased renal responsiveness and an increased dose required to maintain euvolemia. Patients taking furosemide 80 mg daily or above (or an equivalent dose of other loop diuretics) are designated as "diuretic-resistant."[35,36] Diuretic resistance is associated with more severe heart failure, more advanced chronic kidney disease, and worsening renal function with the use of intravenous diuretics.[35,37,38] There are no consensus recommendations available to guide the management of diuretic resistance, but several options exist. First, a thiazide diuretic, such as metolazone, can be given before the loop diuretic.[39] This combination is frequently able to initiate a brisk diuresis, but patients require close monitoring for hypokalemia and worsening renal function. Recently, ultrafiltration has emerged as an option. In the Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Congestive Heart Failure (UNLOAD) trial, patients with congestion treated with ultrafiltration had more weight and fluid loss at 48 hours compared to patients treated with intravenous furosemide, without any significant differences in renal function.[40] For patients with oliguria and renal dysfunction, initiation of renal replacement therapy may be needed. We present an algorithm for the management of diuretic resistance in Figure 3.

The endpoints for discontinuation of diuretic therapy remain unclear. Traditionally, alleviation of the patient's congestive symptoms, edema, and attainment of the patient's self-reported "dry weight" have served as endpoints for diuretic therapy. A more accurate approach may be daily assessments of the JVP, as normalization of the JVP may be a more accurate method to assess for euvolemia. When euvolemia has been achieved, patients should be switched to "maintenance" therapy at a diuretic dose of one-fourth to one-half the total daily dose used for diuresis. Patients should be observed for 24 hours on oral diuretic therapy to ensure that their fluid intake and output are balanced. Generally, we aim for slightly negative fluid balance (less than 500 mL) on an oral diuretic regimen prior to discharge, assuming some relaxation of the salt and fluid restriction once the patient is discharged home.

NEUROHORMONAL THERAPIES

Activation of neurohormonal systems, specifically the renin-angiotensin-aldosterone and beta-adrenergic pathways, are the major mechanisms for disease progression in HFrEF, and agents which block these pathways improve functional status and survival in these patients. In the OPTIMIZE-HF registry, patients treated with beta-blockers on admission had a lower in-hospital mortality.[25] Although beta-blockers are often discontinued in patients with ADHF, continuation of beta-blocker treatment is associated with decreased mortality and rehospitalization at 60 to 90 days.[41] While beta-blocker initiation is often deferred to the outpatient setting, patients who receive a beta-blocker at hospital discharge are 31 times more likely to be treated with a beta-blocker at 60 to 90 day follow-up.[42] Only 3 agents, metoprolol succinate, carvedilol, and bisoprolol, have survival benefit in large clinical trials of systolic heart failure, and therefore are the only recommended agents.[22] In the hospital, hypotension is a common reason for suspension or discontinuation of beta-blocker therapy. However, in the absence of symptoms such as light-headedness, patients with systolic blood pressure as low as 85 mmHg will benefit from beta-blocker treatment.[43] Thus, we recommend continuation or initiation of an evidence-based beta-blocker for all patients hospitalized with systolic heart failure in the absence of symptomatic hypotension, systolic blood pressure <85 mmHg, second or third degree heart block, or the need for intravenous inotropic therapy.

Inhibitors of the renin-angiotensin-aldosterone system also have an important role in patients with HFrEF. Patients treated with angiotensin converting enzyme inhibitors (ACEI) on admission have a lower in-hospital mortality[25] and a lower likelihood of readmission or death within 60 to 90 days.[44] In practice, ACEI or angiotensin receptor blocker (ARB) treatment is frequently suspended or discontinued during treatment with diuretics out of concerns for worsening renal function, an association not borne out in trials.[38,45,46] For patients that are not able to tolerate an indicated therapy, such as a beta-blocker, ACEI, or ARB, the specific contraindication to treatment should be documented in the medical record.

For patients with HFpEF, no therapy has been shown to improve survival.[19,47] The mainstays of therapy are management of congestion, hypertension, and ventricular rate for patients with atrial

fibrillation.[22] Research into novel therapies for diastolic heart failure is ongoing.[48]

DISCHARGE

Patients hospitalized for ADHF are at an increased risk for adverse events following discharge. In an analysis of data from the Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity (CHARM) trial, the risk of death was 6-fold higher in the first month after discharge and remained elevated at 2-fold higher at 2 years after hospitalization, as compared to persons never hospitalized.[49] As yet, no model can accurately predict which ADHF patients will require readmission, though multiple clinical factors have been identified.[50] In the OPTIMIZE-HF registry, increasing admission serum creatinine, a history of chronic obstructive pulmonary disease or cerebrovascular disease, hospitalization for heart failure within the last 6 months, as well as treatment with nitrates, digoxin, diuretics, or mechanical ventilation, were all predictors of mortality and rehospitalization within 60 to 90 days after discharge.[44] Furthermore, a BNP level of greater than 350 pg/mL or less than a 50% reduction in NT-BNP during the hospital stay is also associated with an increased risk for rehospitalization or death.[51,52]

Unfortunately, few interventions reduce heart failure readmission rates. In a recent analysis of Medicare claims data, hospitals with the highest rates of early follow-up after discharge (defined as a clinic visit within 7 days of discharge) had decreased rates of readmission within 30 days.[53] Thus, early follow-up after discharge is essential. Not surprisingly, non-compliance with weight self-monitoring leads to increased readmission and mortality rates, and therefore patient education is essential.[54] The benefit of home telemonitoring programs remains controversial and requires further study.[55,56] At our center, patients are required to follow up with their internist or cardiologist within 7 days of discharge, and the patient's discharge medication list, discharge weight, and laboratory studies on the day of discharge are faxed to the outpatient provider's office to ensure a seamless transition of care.

PERFORMANCE MEASURES AND GUIDELINES

Performance measures are being assessed with greater frequency in medicine to ensure that clinicians perform key assessments and provide treatments that can improve outcomes. Acute and chronic heart failure were 2 of the first areas to be assessed. In 1996, CMS developed a set of 4 measures for inpatient heart failure care (see Supporting Online Table 2 in the online version of this article).[57] Each hospital's performance for these 4 measures is now published at the CMS website. The ACC, AHA, and the American Medical Association's Physician Consortium for Performance Improvement (AMA-PCPI) released a joint heart failure performance measurement set in 2011. This set removes 3 older recommendations (anticoagulation for patients with atrial fibrillation, discharge instructions, and smoking cessation counseling) and adds 2 new recommendations: prescription of an appropriate beta-blocker at discharge and arrangement of a postdischarge follow-up appointment.[58,59] The ACC will publish guidelines based on the ACC/AHA/AMA-PCPI measure set in early 2012. Of the extant performance measures, both ACEI/ARB and beta-blocker therapy at discharge are associated with improved outcomes.[60,61]

CONCLUSION

With the aging of the population, hospitalizations for ADHF are projected to increase substantially, creating a greater necessity for hospitalists to diagnose, risk stratify, and manage inpatients with heart failure. Once the heart failure diagnosis has been established, determining the etiology of the decompensation and estimating the patient's risk for in-hospital and postdischarge adverse events is essential. For patients with reduced systolic function, treatment with neurohormonal therapies, even while hospitalized, improves outcomes. Patients should be scheduled for follow-up within 7 days after discharge to ensure clinical stability. Hospitalists should understand and adhere to the current performance measures for heart failure, as efforts tying payment to the quality of care are likely to evolve.

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