Heart Failure Complicating Acute Myocardial Infarction

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KEYWORDS

• Heart failure • Myocardial infarction • Beta blockers • Angiotensin-converting enzyme inhibitors • Nitrates • Aldosterone antagonists • Digoxin • Positive inotropic drugs

KEY POINTS

- Factors predisposing the older person with acute myocardial infarction (MI) to develop heart failure (HF) include increased prevalence of prior MI and multivessel coronary artery disease, decreased left ventricular (LV) contractile reserve, impairment of LV diastolic relaxation, and an increased prevalence of hypertension, LV hypertrophy, diabetes mellitus, valvular heart disease, and renal insufficiency.
- The American College of Cardiology/American Heart Association (ACC/AHA) guidelines state that class I indications for the use of angiotensin-converting enzyme (ACE) inhibitors during acute MI are (1) patients within the first 24 hours of a suspected acute MI with ST-segment elevation in 2 or more anterior precordial leads, or with clinical HF in the absence of significant hypotension or known contraindications to the use of ACE inhibitors or (2) patients with MI and an LV ejection fraction 40% or less or patients with clinical HF based on systolic pump dysfunction during and after convalescence from acute MI.
- The ACC/AHA class I indications for use of early intravenous beta blockade in patients with acute MI are (1) patients without a contraindication to beta-blockers who can be treated within 12 hours of onset of MI, (2) patients with continuing or recurrent ischemic pain, and (3) patients with tachyarrhythmias, such as atrial fibrillation with a rapid ventricular rate, or hypertension.

Factors predisposing the older person with acute myocardial infarction (MI) to develop heart failure (HF) include an increased prevalence of prior MI and multivessel coronary artery disease, decreased left ventricular (LV) contractile reserve, impairment of LV diastolic relaxation, and an increased prevalence of hypertension, LV hypertrophy, diabetes mellitus, valvular heart disease, and renal insufficiency.[1–4] Women with acute MI are more likely to be older and to develop HF than men with acute MI.[5–8] Prior HF was present in 12% of 124 subjects younger than 70 years of age and in 17% of 137 subjects 70 years of age and older with acute MI.[5] HF occurred during acute MI in 33% of the 124 subjects younger than 70 years of age and in 56% of the 137 subjects 70 years of age and older.[5] Dyspnea due to HF was the initial clinical manifestation of acute MI in 35% of 110 subjects older than 62 years of age (mean age: 82 years) with acute MI.[9]

HF complicating acute MI is associated with a high mortality.[10] HF occurred during acute MI in 40% of 30 subjects who died at 1-year follow-up and in 9% of 202 subjects who were alive at

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1-year follow-up.[5] In the Multicenter Postinfarction Program, the 1-year mortality rate was 28% in 123 subjects with pulmonary congestion occurring during acute MI versus 5.5% in 744 subjects with no pulmonary congestion occurring during acute MI.[11] An analysis of 790 surviving subjects from the Multicenter Postinfarction Program and 1060 placebo-treated subjects from the Multicenter Diltiazem Postinfarction Trial showed at 2-year follow-up that the cardiac mortality hazard ratios were 1.43 for subjects with mild or moderate pulmonary congestion occurring during acute MI and 4.20 for subjects with severe pulmonary congestion during acute MI.[12] Of 86, 771 subjects with acute MI, HF was present at admission or developed during hospitalization in 15.2% of men and 16.8% of women aged 55 to 74 years, and in 25.6% of men and 27.1% of women aged 75 to 85 years.[13] Of 63,853 subjects discharged alive without HF, 8058 subjects (12.6%) were hospitalized with or died of HF at 3.2-year median follow-up.[13]

Pulmonary venous hypertension with pulmonary congestion and a low cardiac output may complicate acute MI. Pulmonary congestion occurs when the pulmonary capillary wedge pressure exceeds 18 mm Hg.[14] Peripheral hypoperfusion occurs when the cardiac index falls below 2.2 L/min/m^2.[14] The greater the extent of injury to the left ventricle, the lower the LV ejection fraction and the higher the incidence of clinical manifestations of HF. At follow-up, a low LV ejection fraction is an independent predictor of mortality in subjects with HF associated with acute MI.[5,12,15]

Older patients with prior MI and HF have a higher mortality at follow-up if they have an abnormal LV ejection fraction than if they have a normal LV ejection fraction.[16,17] **Table 1** shows the mortality rates in older men and in older women with prior MI and HF at 1-year, 2-year, 3-year, 4-year, and 5-year follow-up.[17] The mortality rates were similar in men versus women with normal or abnormal LV ejection fraction.[17] Older patients with an abnormal LV ejection fraction had a 2.2 times higher mortality rate than older patients with a normal LV ejection fraction after controlling other prognostic variables.[17]

Table 1
Mortality rates in older men and women who have congestive heart failure and prior myocardial infarction

Mortality (y)	Normal LV Ejection Fraction (n = 276) (%)	Abnormal LV Ejection Fraction (n = 340) (%)
1	19	41
2	39	65
3	49	78
4	56	86
5	74	92

Data from Aronow WS, Ahn C, Kronzon I. Prognosis of congestive heart failure after prior myocardial infarction in older men and women with abnormal versus normal left ventricular ejection fraction. Am J Cardiol 2000;85:1382–4.

GENERAL MEASURES

In general, management of HF complicating acute MI is similar in older and younger patients. Underlying causes of HF should be treated when possible.[18,19] Precipitating factors of HF should be identified and treated.[18,19] The LV ejection fraction must be measured in patients with HF associated with acute MI to guide therapy.[18] Echocardiography with Doppler may be helpful in determining the presence and severity of valvular heart disease, such as aortic stenosis or mitral regurgitation; LV diastolic dysfunction due to LV hypertrophy; LV wall-motion abnormalities caused by acute myocardial ischemia; and complications of acute MI, including ventricular rupture, ventricular septal rupture, papillary muscle rupture, ruptured chordae tendineae, papillary muscle dysfunction, LV aneurysm, intracardiac thrombi, pericardial effusion with and without cardiac tamponade, and right ventricular infarction.[5,19–23]

HEMODYNAMIC MONITORING

Invasive hemodynamic monitoring may be necessary to guide the therapy for some patients with acute MI and a pulmonary capillary wedge pressure of 18 mm Hg or a cardiac index !2.2 L/min/m^2. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines state that class I indications for balloon flotation right-heart catheter monitoring during acute MI include (1) severe or progressive HF or pulmonary edema, (2) progressive hypotension when unresponsive to fluid administration or when fluid administration may be contraindicated, and (3) suspected mechanical complications of acute MI, such as ventricular septal defect, papillary muscle rupture, or pericardial tamponade if an echocardiogram has not been performed.[24]

OXYGEN

Experimental data have shown that breathing oxygen may limit ischemic myocardial injury.[25] Patients with acute MI and HF may have hypoxemia