CARDIOVASCULAR MEDICINE

Coronary artery disease and outcome in acute congestive heart failure

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Objectives: To quantify the prognostic impact of coronary artery disease (CAD) on patients with acute heart failure (HF).

Design: Prospective cohort study of 217 consecutive patients presenting with acute HF to the emergency department. Treatment, hospitalisation, the use of revascularisation procedures, and survival were observed during follow up of up to three years.

Results: CAD was present in 153 patients (71%). Patients with and without CAD were similar with respect to age and sex. Although adequate HF treatment was initiated more rapidly among patients with CAD, their initial outcomes including hospitalisation rate, time to discharge, and total treatment cost were significantly worse. Moreover, despite higher use of angiotensin converting enzyme inhibitors and β blockers during follow up, patients with CAD had a significantly lower survival rate. Cumulative survival at 720 days was 48.7% of patients with CAD as compared with 76.4% of patients without CAD ($p = 0.0004$). In Cox regression analysis the presence of CAD increased the risk of death by more than 250% (hazard ratio 2.57, 95% confidence interval 1.50 to 4.39, $p = 0.001$). This strong association persisted after multivariate adjustments. The use of coronary angiography and coronary revascularisation procedures was low, both at initial presentation and during follow up.

Conclusion: CAD is a strong and independent predictor of mortality among patients with acute HF. Whether, for example, less restrictive use of revascularisation procedures in this elderly HF population can improve the outcome for patients with CAD warrants further study.

Coronary artery disease (CAD) is a major cause of heart failure (HF).[1–3] Previous studies of patients with chronic stable cardiomyopathy have shown the importance of distinguishing between ischaemic and non-ischaemic cardiomyopathy, because the diagnosis influences management. Smoking cessation, lipid lowering treatment, and revascularisation are important interventions in patients with left ventricular dysfunction due to CAD.[1–5] Moreover, the diagnosis of CAD seems to influence prognosis, as CAD is an independent predictor of long term mortality for patients with chronic cardiomyopathy.[4–8] The role of CAD as a prognostic factor in patients with acute HF is poorly defined. Therefore, this study sought to evaluate the impact of CAD on short and long term outcome of patients presenting with acute congestive HF to the emergency department.

METHODS

Setting and study population

This study specifically evaluated the impact of CAD on short and long term outcome of patients with acute congestive HF recruited in the BASEL (B-type natriuretic peptide for the acute shortness of breath evaluation) study.[9] The BASEL study was a prospective, randomised, single blind study conducted in the emergency department of the University Hospital Basel, Switzerland, from May 2001 to April 2002. The study was carried out according to the principles of the Declaration of Helsinki and approved by our local ethics committee. Written informed consent was obtained from all participating patients.

A total of 452 consecutive patients presenting with acute dyspnoea as the primary complaint and no obvious traumatic cause of dyspnoea were enrolled. Among these, 217 patients had acute congestive HF. Patients with severe renal disease (serum creatinine > 250 μmol/l) and patients in cardiogenic shock were excluded. No other exclusion criteria in the BASEL trial may have excluded patients with CAD or biased their enrolment into that trial. CAD was defined as the presence of at least one of the following criteria: a significant (> 70% diameter stenosis) lesion on coronary angiography, a history of myocardial infarction, percutaneous coronary intervention or coronary artery bypass grafting, and documented myocardial ischaemia during exercise testing.

Routine clinical assessment

All patients underwent an initial clinical assessment that in general included clinical history, physical examination, electrocardiography, pulse oximetry, blood tests including arterial blood gas analysis (when indicated), and chest radiography. Echocardiography and pulmonary function tests were strongly recommended in the emergency department on an outpatient basis or in the hospital if the patient was admitted. The final discharge diagnosis of acute congestive HF was based on all information available after discharge of the patient including response to treatment and necropsy data for those patients who died in hospital. B-type natriuretic peptide (BNP) concentrations were available for the diagnosis for half the patients and were determined in a blinded fashion for the other half.

End points

Time to treatment was defined as the time interval from presentation to the emergency department to the initiation of treatment specific for acute congestive HF. This included the

Abbreviations: ACE, angiotensin converting enzyme; BASEL, B-type natriuretic peptide for the acute shortness of breath evaluation; BNP, B-type natriuretic peptide; CAD, coronary artery disease; HF, heart failure; TACTICS-TIMI 18, treat angina with Aggrastat and determine cost of therapy with an invasive or conservative strategy-thrombolysis in myocardial infarction 18

Table 1 Baseline characteristics of patients with acute congestive heart failure

Variable	All patients (n = 217)	CAD (n = 153)	No CAD (n = 64)	p Value*
Age (years)	75 (11)	75 (11)	73 (13)	0.226
Female sex	93 (43%)	66 (43%)	27 (42%)	0.897
History				
Systemic hypertension	138 (64%)	103 (67%)	35 (55%)	0.078
Diabetes mellitus	67 (31%)	56 (37%)	11 (17%)	0.005
Chronic obstructive pulmonary disease	52 (24%)	41 (27%)	11 (17%)	0.130
Asthma	4 (2%)	1 (1%)	3 (5%)	0.078
Pulmonary embolism	6 (3%)	5 (3%)	1 (2%)	0.673
Pneumonia	27 (12%)	22 (14%)	5 (8%)	0.259
Other pulmonary disease	15 (7%)	14 (9%)	1 (2%)	0.073
Any pulmonary disease	88 (41%)	69 (45%)	19 (30%)	0.035
Depressive disorder	14 (7%)	10 (7%)	4 (6%)	1.000
Stroke or peripheral vascular disease	60 (28%)	50 (33%)	10 (16%)	0.010
Chronic kidney disease	85 (39%)	66 (43%)	19 (30%)	0.064
Deep vein thrombosis	15 (7%)	10 (7%)	5 (8%)	0.772
Symptoms				
Dyspnoea†				0.465
Slight hill	26 (12%)	20 (13%)	6 (9%)	
Level ground	122 (56%)	89 (58%)	33 (52%)	
At rest	67 (31%)	43 (28%)	24 (38%)	
Paroxysmal nocturnal dyspnoea	102 (47%)	76 (50%)	26 (41%)	0.223
Nocturia	86 (40%)	68 (44%)	18 (28%)	0.025
Weight gain	38 (18%)	27 (18%)	11 (17%)	0.935
Weight loss	21 (10%)	16 (11%)	5 (8%)	0.624
Chest pain	85 (39%)	63 (41%)	22 (34%)	0.349
Nausea	32 (15%)	22 (14%)	10 (16%)	0.813
Coughing	93 (43%)	64 (42%)	29 (45%)	0.636
Expectoration	59 (27%)	44 (29%)	15 (23%)	0.422
Fever	32 (15%)	27 (18%)	5 (8%)	0.091
Vital status				
Systolic blood pressure (mm Hg)	147 (31)	147 (32)	146 (31)	0.813
Diastolic blood pressure (mm Hg)	88 (21)	88 (19)	88 (24)	0.897
Heart rate (beats/min)	97 (26)	95 (25)	101 (29)	0.161
Temperature (℃)	37.2 (0.9)	37.2 (0.9)	37.3 (0.8)	0.558
Signs				
Tachypnoea (>20 breaths/min)	96 (44%)	68 (44%)	28 (44%)	0.925
Increased jugular venous pressure	49 (23%)	35 (23%)	14 (22%)	0.872
Hepatojugular reflux	34 (16%)	21 (14%)	13 (20%)	0.223
Rales	130 (60%)	99 (65%)	31 (48%)	0.026
Wheezing	30 (14%)	21 (14%)	9 (14%)	0.948
Hyperresonant percussion	16 (7%)	12 (8%)	4 (6%)	0.783
Dullness	24 (11%)	20 (13%)	4 (6%)	0.163
Lower extremity oedema	101 (47%)	72 (47%)	29 (45%)	0.814
Cyanosis	17 (8%)	10 (7%)	7 (11%)	0.271
Laboratory tests				
Ejection fraction (%)‡	40 (30–55)	40 (25–50)	40 (35–60)	0.168
GFR (ml/min/1.73 m^2)	53 (28)	50 (28)	61 (27)	0.009
Haemoglobin (g/l)	130 (24)	127 (24)	135 (22)	0.013
Serum albumin (g/l)	33 (6)	33 (5)	34 (7)	0.284
Troponin I (μg/l)	0.5 (0.3–2.1)	0.6 (0.3–2.7)	0.3 (0.3–1.1)	0.003
B-type natriuretic peptide (pg/ml)	822 (410–1300)	888 (474–1300)	682 (222–1300)	0.048

Data are presented as mean (SD), median (interquartile range), or number of patients (%).
*Patients with coronary artery disease (CAD) versus patients without CAD; †one patient in both groups had dyspnoea only while walking up a steep incline; ‡available for 148 patients.
GFR, glomerular filtration rate.

administration of diuretics, morphine, glyceryl trinitrate, or angiotensin converting enzyme (ACE) inhibitors. Total days in hospital, total treatment cost, and all cause mortality were prospectively assessed during follow up. The calculation of total days in hospital and total cost of hospitalisations summed up all hospitalisations after the initial presentation to the emergency department. Since ratios of costs to charges have not been defined for the majority of services and departments at our institution, nor that of other hospitals in the area, hospital charges were used as the most appropriate estimate of the true costs.[10 11] To avoid an imbalance owing to differences in reimbursement or charges associated with different types or classes of insurance, charges were standardised according to the actual rates for patients with general insurance who were living in Basel. Total treatment cost did include total cost of hospitalisations and medication but not regular outpatient visits to a private doctor.

A single trained researcher contacted patients six, 12, and 24 months after the initial presentation for a telephone interview. In addition, referring physicians were contacted in case of uncertainties regarding health status or hospitalisations. The administrative databases of the patients' hometowns were assessed to ascertain the vital status of those patients who could not be contacted by telephone. All information derived from contingent hospital readmission records or provided by the referring physician or by the outpatient clinic was reviewed and entered on to the computer database.

Statistical analysis

Data were statistically analysed with the SPSS/PC version 12.0 (SPSS Inc, Chicago, Illinois, USA) software package. A significance level of 0.05 was used. Data were compared by the *t* test, Mann-Whitney U test, Fisher's exact test, and χ^2

Table 2 Impact of CAD on outcome

Variable	CAD (n = 153)	No CAD (n = 64)	p Value
Initial outcome			
Time to treatment (min)	49 (19–163)	105 (24–195)	0.051
Hospital admission	140 (92%)	51 (80%)	0.015
Admission to intensive care	45 (29%)	13 (20%)	0.167
Time to discharge (days)	13 (6–20)	9 (1–17)	0.042
30 day mortality	21 (14%)	6 (9%)	0.377
Initial total treatment cost ($)	6 374 (3 732–9 606)	4 432 (756–8 670)	0.006
Discharge medication			
ACE inhibitor or ARB	116 (84%)	41 (70%)	0.027
β Blocker	90 (65%)	27 (46%)	0.013
Diuretic	127 (92%)	45 (76%)	0.002
Glyceryl trinitrate	61 (44%)	9 (15%)	<0.001
Aspirin	75 (54%)	17 (29%)	0.001
Anticoagulation	66 (48%)	36 (61%)	0.081
Long term outcome			
Total days in hospital			
At 90 days	14 (7–25)	11 (2–21)	0.047
At 180 days	16 (8–29)	14 (3–22)	0.095
At 360 days	18 (8–36)	17 (5–30)	0.250
Treatment cost ($)			
A 90 days	7 413 (4 435–12 364)	5 082 (1 606–9 472)	0.005
At 180 days	7 981 (4 755–14 033)	6 109 (2 511–10 328)	0.016
At 360 days	10 415 (5 384–18 365)	8 122 (3 376–14 558)	0.078
Cumulative survival (%)			
At 360 days	64.1 (3.9)	80.0 (5.0)	
At 720 days	48.7 (4.1)	76.4 (5.3)	0.0004*

Data are presented as median (interquartile range), number of patients (%), or mean (SE).
*By log rank test.
ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker.

test as appropriate. All hypothesis testing was two tailed. Cox regression analysis was used to identify predictors of death in univariate and multivariate analyses.

RESULTS

Baseline characteristics

A total of 153 patients (71%) had CAD. Patients with and without CAD were similar with respect to age and sex (table 1). Differences in baseline characteristics were a higher incidence of diabetes mellitus, stroke or peripheral vascular disease, and pulmonary disease in patients with CAD. Symptoms, signs, and vital status were also similar with the exception of nocturia and rales, both being more common among patients with CAD. Laboratory tests showed important differences between the groups. Haemoglobin and glomerular filtration rate were significantly lower, whereas troponin I and BNP were significantly higher in patients with CAD.

Initial outcome

Although adequate treatment was initiated more rapidly in patients with CAD, initial outcome was worse. Hospitalisation rate, time to discharge, and total treatment cost were significantly higher in patients with CAD (table 2). Discharge medication in patients with CAD more often included ACE inhibitors or angiotensin receptor blockers, β blockers, diuretics, nitrates, and aspirin as compared with patients without CAD.

Long term follow up

Clinical 360 day follow up data were available for all 217 patients (100%). The median time interval to last patient contact or patient death was 678 days. Patients with CAD had a significantly lower survival rate. Cumulative survival at 720 days was 48.7% of patients with CAD as compared with 76.4% of patients without CAD (p = 0.0004; fig 1). Total treatment cost remained significantly higher for patients with CAD at 90 and 180 days and still tended to be much higher at 360 days.

CAD as a predictor of death

Cox regression analysis showed that CAD was a powerful predictor of death in this cohort. In univariate analysis, the presence of CAD increased the risk of death by 257% (hazard ratio (HR) 2.57, 95% confidence interval (CI) 1.50 to 4.39; p = 0.001). This strong association persisted after adjustment for age and sex (HR 2.41, 95% CI 1.41 to 4.13; p = 0.001). When diabetes mellitus, stroke or peripheral vascular disease, and pulmonary disease were entered as additional variables in the model, CAD remained a strong predictor of death (HR 2.24, 95% CI 1.28 to 3.89; p = 0.001). Additionally including those laboratory test that differed between the groups in the model (haemoglobin, glomerular filtration rate, troponin I, and BNP) resulted in a hazard ratio for CAD of 2.10 (95% CI 1.07 to 4.13: p = 0.032). Interestingly, left ventricular ejection fraction, which was available for 148 patients, was not a significant predictor of death (p = 0.259).

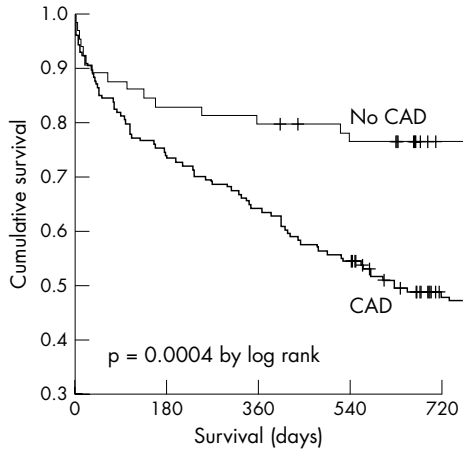

Figure 1 Cumulative survival of patients with and without coronary artery disease (CAD).

Table 3 Use of procedures related to coronary revascularisation

Variable	CAD (n = 153)	No CAD (n = 64)	p Value
At initial presentation			
Coronary angiography	27 (18%)	4 (6%)	0.033
PCI	8 (5%)	0	0.109
CABG	10 (7%)	0	0.036
Myocardial perfusion SPECT	13 (9%)	0	0.012
Exercise ECG	12 (8%)	0	0.020
During follow up			
Coronary angiography	19 (12%)	3 (5%)	0.136
PCI	10 (7%)	1 (2%)	0.181
CABG	2 (1%)	1 (2%)	1.000
Myocardial perfusion SPECT	31 (20%)	5 (8%)	0.025

Data are presented as number of patients (%).
CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; SPECT, single photon emission computed tomography.

Use of coronary revascularisation procedures

As table 3 shows, the use of coronary angiography and coronary revascularisation procedures was low for patients with CAD. At initial presentation, only 12% of patients with CAD received either percutaneous or surgical revascularisation.

DISCUSSION

This study showed the impact of CAD on outcome of patients presenting with acute congestive HF to the emergency department. Most important, more than 70% of patients had CAD, and these patients with CAD had a significantly lower survival rate. Moreover, CAD remained an independent predictor of death in multivariate analysis. Assessment of cardiac markers at presentation showed that patients with CAD had more extensive myocardial necrosis (troponin) and haemodynamic stress (BNP) than did patients without CAD. In addition, although the groups were of similar age, we noted higher co-morbidity including diabetes mellitus, diffuse vascular disease, and resulting end organ damage (glomerular filtration rate) in patients with CAD. These differences may in part explain the dismal outcome of patients with CAD.

Current American College of Cardiology/American Heart Association and European Society of Cardiology management guidelines for patients with acute congestive HF recommend searching for and eventually treating myocardial ischaemia as the cause of decompensation.[1 2] However, coronary revascularisation procedures are notoriously underused in this elderly patient cohort in clinical practice.[1 2 12] In our study, only 12% of patients with CAD and acute congestive HF received a coronary revascularisation procedure during initial presentation. This finding is in complete agreement with data from the EuroHeart survey.[12] We hypothesise that CAD would have a less negative impact on outcome of patients with acute congestive HF if myocardial ischaemia were treated less restrictively with coronary revascularisation in these patients. Early revascularisation for acute congestive HF due to CAD and myocardial ischaemia may be as beneficial as early revascularisation for acute coronary syndromes.[13 14] Obviously, elderly patients with HF do have significant co-morbidity and a higher risk of periprocedural complications than do patients with acute coronary syndromes.[15] However, they also seem to have more to gain from revascularisation. In fact, subgroup analysis from the TACTICS-TIMI 18 (treat angina with Aggrastat and determine cost of therapy with an invasive or conservative strategy-thrombolysis in myocardial infarction 18) study showed that almost all of the benefit from early revascularisation was seen in the elderly high risk patients.[16] Moreover, elderly patients with chronic symptomatic CAD receive long term benefit from an invasive versus optimised medical treatment strategy.[17 18]

Our results extend the results of previous studies of patients with chronic and stable cardiomyopathy. To determine which of the many clinical parameters routinely collected influence mortality in patients with chronic HF, Likoff and colleagues[5] followed up 201 patients with idiopathic or ischaemic dilated cardiomyopathy for 28 months. Three characteristics at the study entry independently predicted an increased mortality risk: left ventricular ejection fraction, maximum oxygen uptake, and ischaemic cardiomyopathy. The increased mortality risk among patients with cardiomyopathy due to CAD was later confirmed by several groups.[4 6 7] Overall, these studies suggest that CAD increases mortality by 50% (hazard ratio 1.50) in patients with chronic cardiomyopathy.[4–7] Interestingly, the prognostic impact of CAD in acute HF even exceeds that in chronic cardiomyopathy. The hazard ratio for CAD was 2.57 in this study.

Limitation

CAD was defined according to established criteria.[19] As coronary angiography was not performed in all patients, those with previously undiagnosed CAD may have been incorrectly classified as not having CAD.[20] However, as the standard criteria showed an incidence of CAD of 71% in this study, the number of missed cases most likely was small. As any misclassification would only reduce the difference between groups, the prognostic impact of CAD may even be greater than calculated in our study.

Conclusion

CAD is a strong and independent predictor of mortality in patients with acute HF. Whether, for example, less restrictive use of revascularisation procedures in this elderly HF population can improve the outcome of patients with CAD warrants further study.

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IMAGES IN CARDIOLOGY

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Guidewire loss: mishap or blunder?

A 40 year old man underwent an uncomplicated operation for adhesive intestinal obstruction caused by Crohn's disease six months previously. After surgery, a cardiologist in his second year of training inserted a central venous catheter via the left subclavian vein. No problems with the catheterisation procedure were recorded. The trainee cardiologist was not familiar with central venous catheterisation or the Seldinger technique and was unsupervised. The catheter tray was not checked for guide wires after the procedure. A check chest *x* ray was reported to show no problems, and postoperative recovery was uneventful. Three weeks after discharge the patient felt mild pain and oedema on the right lower extremity, but nobody took it seriously and, after taking antibiotics for one week, the patient felt better. Six months after the initial abdominal surgery, the patient presented with posterior cervical pain and a guidewire, lost during post-surgical insertion of the central venous catheter, was shown protruding from the back of neck, like an antenna (left panel). The spiral computed tomography and three dimensional reconstruction confirmed suspicions (middle panel). A guidewire was seen extending from the saphenous vein, vena cava, right atrium, right ventricle, pulmonary artery, and lung and to the back of neck. The inner steel wire is separated from outer spring ring in the heart, and breakage of the outer spring ring is clearly visible. The inner steel wire on the back of neck was removed easily (right panel, B), but it was more difficult to remove the two parts of the outer spring ring (right panel, C1 and C2).

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