

# Acute Heart Failure With and Without Concomitant Acute Coronary Syndromes: Patient Characteristics, Management, and Survival

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## ABSTRACT

**Background:** Acute coronary syndromes (ACS) may precipitate up to a third of acute heart failure (AHF) cases. We assessed the characteristics, initial management, and survival of AHF patients with (ACS-AHF) and without (nACS-AHF) concomitant ACS.

**Methods and Results:** Data from 620 AHF patients were analyzed in a prospective multicenter study. The ACS-AHF patients (32%) more often presented with de novo AHF (61% vs 43%;  $P < .001$ ). Although no differences existed between the 2 groups in mean blood pressure, heart rate, or routine biochemistry on admission, cardiogenic shock and pulmonary edema were more common manifestations in ACS-AHF ( $P < .01$  for both). Use of intravenous nitrates, furosemide, opioids, inotropes, and vasopressors, as well as noninvasive ventilation and invasive coronary procedures (angiography, percutaneous coronary intervention, coronary artery bypass graft surgery), were more frequent in ACS-AHF ( $P < .001$  for all). Although 30-day mortality was significantly higher for ACS-AHF (13% vs 8%;  $P = .03$ ), survival in the 2 groups at 5 years was similar. Overall, ACS was an independent predictor of 30-day mortality (adjusted odds ratio 2.0, 95% confidence interval 1.07–3.79;  $P = .03$ ).

**Conclusions:** Whereas medical history and the manifestation and initial treatment of AHF between ACS-AHF and nACS-AHF patients differ, long-term survival is similar. ACS is, however, independently associated with increased short-term mortality. (*J Cardiac Fail* 2014;20:723–730)

**Key Words:** Acute heart failure, acute coronary syndromes, management, survival.

Acute heart failure (AHF) is an important reason for hospitalization in Western countries and is associated with poor prognosis. Coronary artery disease (CAD) is a major cause of heart failure (HF),<sup>1</sup> and acute coronary syndromes (ACS)

are the precipitating factor in up to one-third of AHF cases.<sup>2–4</sup> Data suggest that AHF patients may have a worse prognosis when they have CAD, concomitant ischemia, or both.<sup>5–7</sup> Regarding treatment of concomitant ACS, AHF guidelines mainly emphasize the importance of coronary angiography and revascularization.<sup>1,8,9</sup>

Overall, patients presenting with AHF have much higher mortality rates than ACS patients.<sup>10</sup> Increased short- and long-term mortality is associated with concomitant HF in the setting of unstable angina pectoris (UAP) or myocardial infarction (MI).<sup>11–14</sup> In addition, data suggest that compared with ACS patients without HF, ACS patients with complicating HF are less likely to receive the recommended therapies.<sup>11–14</sup>

Of note, ACS patients have often been excluded from AHF trials, leaving characteristics and treatment of AHF precipitated by ACS (ACS-AHF) inadequately described. In the present study, we evaluated ACS-AHF patients compared with AHF patients with no concomitant ACS (nACS-AHF) regarding clinical profile, management, and survival.

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## Methods

The Finnish Acute Heart Failure Study (FINN-AKVA) prospectively enrolled 620 consecutive patients hospitalized for AHF in 14 Finnish hospitals during 3 months in 2004.<sup>4</sup> Inclusion in the study required confirmation of an AHF diagnosis during the hospital stay. The study included patients with de novo (new-onset) AHF and those with exacerbation of chronic HF. Patients were enrolled only once in the study, and data on their medical history, clinical presentation, and initial management were recorded in detail. Oral medication was registered both on admission and at discharge. On the basis of clinical presentation, patients were classified according to the ESC 2005 guideline classification into 5 groups<sup>15,16</sup>:

1. Cardiogenic shock (CS): evidence of tissue hypoperfusion (eg, oliguria) and low blood pressure (systolic blood pressure <90 mm Hg or need for vasopressors to maintain perfusion) caused by HF after correction of preload.
2. Pulmonary edema: acute heart failure with severe respiratory distress, crackles on lung auscultation, and pulmonary edema on chest x-ray, usually with O<sub>2</sub> saturation <90% on room air.
3. Acute decompensated HF: signs and symptoms of AHF not fulfilling the criteria of hypertensive crisis, pulmonary edema, or CS.
4. Hypertensive AHF: Signs and symptoms of AHF accompanied by high blood pressure (>160 mm Hg) and relatively preserved left ventricular function (ejection fraction >40%) with congestion or pulmonary edema on chest radiograph.
5. Right HF: AHF predominantly due to right ventricular failure. Signs and symptoms of decreased cardiac output, distension of jugular veins, enlarged liver, and severe edema.

Our study did not include patients with high-output HF. All patients gave their written informed consents. FINN-AKVA was approved by local Ethics Committees and was conducted in concordance with the Declaration of Helsinki.

We investigated characteristics at presentation, initial management, and long-term survival of ACS-AHF and nACS-AHF patients as a secondary analysis of the FINN-AKVA cohort. Investigators at each participating center assessed factors causing AHF (UAP, MI, infection, arrhythmias) for each patient. ACS was defined as UAP or MI. Troponin T (TnT) levels were measured with the use of a Roche Elecsys 2010 assay on admission and at the 48-hour time point. The cutoff value for elevated TnT was 0.03 µg/L. Initial treatment included intravenous (IV) medication such as nitrates, furosemide, opioids, inotropes (levosimendan or dobutamine), and vasopressors (dopamine, noradrenaline, or adrenaline) as well as noninvasive ventilation (NIV) within the first 48 hours after admission. Invasive diagnostic and therapeutic coronary procedures (angiography, percutaneous coronary intervention [PCI], and coronary artery bypass graft surgery [CABG]) were recorded during the index hospitalization. In addition, we compared the doses of beta-blockers (BB), angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), and spironolactone at discharge with recommended target doses.<sup>1</sup> Documentation included length of stay (LOS) as well as admissions to cardiac (CCU) and intensive (ICU) care units. All-cause mortality was determined for all patients up to 5 years after the index hospitalization from the national Population Register Center, as was the time of death.

Statistical analyses were with the use of SPSS 21 statistical software (IBM Corp, Armonk, New York), with results presented as numbers and percentages, mean with standard deviation, or median with interquartile range for variables not normally distributed. We used the  $\chi^2$  test for comparison of categorical variables, the *t* test and Mann-Whitney *U* test for continuous variables, and the Kaplan-Meier method for survival analyses, and we compared survival between groups with the use of the log-rank test. To evaluate the independent effect of ACS on mortality, we performed multivariable logistic regression, adjusting for potential confounders, including sex, age, medical history (previous history of HF, CAD, hypertension, diabetes, cerebrovascular disease, and chronic obstructive pulmonary disease) as well as systolic blood pressure, anemia (defined as hemoglobin <120 g/L for women and <130 g/L for men), hyponatremia (sodium <135 mmol/L), and estimated glomerular filtration rate (calculated with the use of the CKD-EPI equation) on admission. Odds ratios (ORs) are shown with 95% confidence intervals (CIs). We considered *P* values of <.05 to be statistically significant.

## Results

Patients' mean age was  $75 \pm 10$  years; one-half were women. ACS was a precipitating factor for 32% of patients, and of these only 39% had a history of HF; thus, most ACS-AHF presented as de novo AHF. As presented in [Table 1](#), ACS-AHF patients more frequently had a history of CAD, MI, diabetes, or hypercholesterolemia. The clinical presentation of CS and pulmonary edema was more frequent among ACS-AHF patients, but at presentation no major differences existed between these groups overall in parameters or in biochemistry, apart from TnT and N-terminal pro-B-type natriuretic peptide ([Table 1](#)).

ACS-AHF patients received IV treatments and NIV more often ([Table 2](#)). IV furosemide was the most common treatment in both groups, given to as many as 85% of ACS-AHF patients. IV nitrate was frequent in ACS-AHF (69%); in contrast, fewer than one-third of nACS-AHF patients received it. A striking difference appeared in the use of inotropes and vasopressors, with a 5-fold greater use of the latter in ACS-AHF. In addition, NIV use was twice as common in nACS-AHF.

Invasive coronary procedures differed markedly between the groups. In ACS-AHF, coronary angiography and revascularization (PCI/CABG) during the index hospitalization were more frequent ([Table 2](#)). A rather large proportion of revascularized ACS-AHF patients underwent CABG (38%). Although 43% of nACS-AHF patients had elevated TnT levels, only 8% of them underwent an angiography; revascularization rates also were very low. ACS-AHF patients were more frequently admitted to a CCU or an ICU and had longer LOS ([Table 2](#)).

Prescription of cardiac medications increased during hospitalization in both groups ([Fig. 1](#)). Before hospitalization, ACS-AHF patients more frequently received lipid-lowering agents and antithrombotics, whereas furosemide, spironolactone, and warfarin were more common among nACS-AHF patients. No difference existed between the groups

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