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Characteristics and prognosis in acute myocarditis and unexplained acute chest pain: a nationwide longitudinal cohort study

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ABSTRACT

Aims Acute myocarditis (AM) is a disease with variable prognosis, ranging from complete recovery to end-stage heart failure (HF) and death but often challenging to differentiate from unexplained acute chest pain (UCP) in the acute setting. This study examines the short-tem and long-term outcomes of AM compared with UCP, focusing on the risk of HF development.

Methods We used the Swedish Web System for Enhancement of Evidence-Based Care in Heart Disease Evaluated According to Recommended Therapies-registry to identify patients >16 years admitted to hospital between 1 January 1998 and 31 December 2018 with either AM or UCP. Patients were followed for outcomes including mortality, rehospitalisation and HF development over both short-term (30 days) and long-term periods. Cox proportional hazards models were used to compare the risks, adjusting for demographic and clinical-related factors.

Results A total of 3792 patients with AM and 109 934 patients with UCP were included. Median follow-up time was 7.8 years (Q1, Q3; 3.4, 12.3). AM patients were younger compared with UCP patients, median age 37 years (Q1, Q3; 26, 52) vs 59 years (Q1, Q3; 49, 69) and more likely to be men (79.9% vs 51.4%, p<0.001). Comorbidity burden was less pronounced within the AM cohort. Chest pain was the most common presenting symptom in both groups. Mortality rate at 30 days (OR 3.75, 95% CI 1.9 to 7.3, p<0001) as well as long term (OR 2.0, 95% CI 1.69 to 2.39, p<0.001) were significantly higher in AM patients compared with UCP. AM patients were more likely to develop HF during follow-up (OR 2.3, 95% CI 1.81 to 2.93, p<0001).

Conclusions AM is associated with worse short-term and long-term outcomes compared with UCP, including a higher risk of developing HF even after the first year.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ To differentiate patients with acute myocarditis (AM) from unexplained chest pain (UCP) diagnostically can be challenging. AM studies are often based on a small cohort of patients and there are few studies describing the clinical characteristics of patients with UCP. There are also limited comparative data between the two patient groups.

WHAT THIS STUDY ADDS

⇒ In this large nationwide register-based follow-up study, AM is associated with worse short-term and long-term outcomes compared with UCP, including a higher risk of developing heart failure even after the first year.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ These results underscore the importance of accurately distinguishing AM from UCP in clinical practice. Further research is needed to develop robust risk stratification strategies for AM patients to better predict long-term complications.

INTRODUCTION

Acute myocarditis (AM) is an inflammation of the heart muscle due to infectious, toxic or autoimmune processes.[1] The clinical presentation of AM can vary wildly, ranging from mild symptoms, such as chest pain and fever, to severe presentation including fulminant haemodynamic collapse and sudden death.[2 3] The long-term prognosis also differs significantly depending on the severity of the initial inflammation and the extent of heart muscle damage, with outcomes ranging from complete recovery to the development of advanced heart failure (HF), the need for heart transplantation or death.[2] Treatment is mostly supportive, but in certain cases, immunosuppressive treatments are needed.[4] Men are more susceptible to AM than women, especially at a younger age.[5]

As high-sensitivity troponin assays and cardiac MR (CMR) imaging become integral to clinical practice, the AM population is undergoing notable changes, with milder cases now being detected more frequently.[6] Notably, multiple recent studies indicate

that these patients tend to have a favourable prognosis.[3 7 8]

AM often mimics acute coronary syndrome (ACS), presenting with sudden chest pain and ECG abnormalities[9] making the differential diagnosis challenging. This overlap in clinical presentation often requires invasive coronary angiography to rule out obstructive coronary artery disease. Despite its clinical significance, the published data on AM, particularly among patients with symptoms suggestive of myocardial infarction, remain sparse and are limited to case reports and case series on young males.[10] Culprit-free coronary angiography is found in 5%–13% of patients with suspected ACS,[11] with many of these patients later diagnosed with AM.[12] Another group of patients presenting with acute chest pain and culprit-free coronary angiography are classified as unexplained acute chest pain (UCP). UCP is characterised by sudden, severe chest discomfort with no obvious cause. The diagnosis is typically made once alternative explanations (such as ACS) are ruled out. Studies show that patients with UCP are at increased risk of cardiovascular events.[13–15] Both AM and UCP have better prognosis than acute myocardial infarction.[16] Accurately differentiating AM from UCP poses a considerable clinical challenge, as both conditions share similar presentations but necessitate distinct diagnostic and therapeutic strategies.[17 18] Additionally, the lack of comparative outcome data emphasises the pressing need for further investigation in this area.

We used data from the national registry SWEDEHEART (Swedish Web System for Enhancement of Evidence-Based Care in Heart Disease Evaluated According to Recommended Therapies) which includes all patients admitted to hospitals with coronary care units (CCU) in Sweden. Our aim was to characterise patients with AM in comparison to those with UCP and investigate the prognosis in each group.

METHODS

Database

The SWEDEHEART registry continuously registers all patients admitted to hospitals with CCU; Register of Information and Knowledge about Swedish Heart Intensive Care Admissions is one component of the SWEDEHEART registry. By 2008, all Swedish hospitals with a CCU were participating in the registry. The full protocol has been published previously,[17] and detailed information and the complete protocol are available online at http://www.ucr.uu.se/swedeheart/. On admission, patients receive written information about SWEDEHEART. According to Swedish law, written consent is not required because quality control is an inherent element of hospital and other care. Research based on the registry is approved by an institutional ethics committee and all personal identifiers are removed from the SWEDEHEART data file when used for research purposes.[18]

Source data are continuously validated by an external monitor via comparison of the information in the registry with hospital patient records. A 94% agreement has been observed between the registered information and the source data in patients' records.[19]

Long-term survival data were obtained by merging the SWEDEHEART database with the Swedish Cause of Death Register, based on the patient's unique 10-digit personal identification number.[17]

Validation of the diagnosis

Studies that have assessed the accuracy of AM diagnosis based on hospital record reviews have reported a correctness rate of over 80% in previous publications.[20]

Patient selection and outcomes

The present study included all consecutive patients aged >16 years admitted between 1 January 1998 and 31 December 2018 and reported to SWEDEHEART with the diagnosis AM or UCP. Diagnoses were coded at the treating physician's discretion according to the International Classification of Diseases version 10 (ICD-10). AM was defined as a discharge diagnosis with one of the following main ICD codes: I40.0, I40.1, I40.8, I40.9, I41.0, I41.1, I41.2, I41.8 or I01.2. UCP was defined as discharge diagnosis R07.4.

The primary outcome was all-cause mortality at 30 days and long-term (median follow-up time 7.8 years). The risk of myocardial infarction, development of HF, significant bleeding and stroke was also studied. Patients were followed until death, emigration or end of follow-up (December 2022).

Statistics

Continuous variables are reported as mean±SD or as median and quartile (Q1–Q3), according to normal or non-normal distribution. Student's t-test or Mann-Whitney U test was used to compare continuous variables, as deemed appropriate. χ^2 test was used for categorical variables. Kaplan-Meier curves were compared with the use of log-rank statistics. However, the underlying assumption of proportional hazards in the Cox model through follow-up was not met (treatment-time interaction, $p<0.001$). Comparisons between groups were, therefore, performed by multivariable adjusted logistic regression with follow-up time included as log-transformed offset variable, with the use of an estimated SE for the difference. Differences with values of $p<0.05$ were considered statistically significant. Software packages used were Stata V.18 and R (V.4.3.1).

RESULTS

The study population comprised 3792 patients with AM and 109 934 patients with UCP. The baseline characteristics are shown in table 1. Patients with AM were younger (median age 37 (Q1, Q3; 26, 62) vs 59 years (Q1, Q3; 49, 69), more often male and had a lower burden of common comorbidities compared with patients with UCP. The

Table 1 Patients characteristics at admission to the cardiac care unit

	AM	UCP	P value
N	3792	109 934	
Age (median (IQR))	37 (26, 52)	59 (49, 69)	<0.001
Male, n (%)	3028 (79.9)	56 533 (51.4)	<0.001
Female, n (%)	764 (20.1)	53 401 (48.6)	<0.001
Smoking (%)			<0.001
Never smoker	58.5	49.3	
Past smoker	17.6	27.8	
Smoker	20.2	19.5	
Unknown	3.7	3.4	
Diabetes, n (%)	152 (4.0)	9554 (8.7)	<0.001
Hypertension, n (%)	140 (3.7)	13 964 (12.7)	<0.001
Previous stroke, n (%)	49 (1.3)	4336 (3.9)	<0.001
Previous HF, n (%)	46 (1.2)	2626 (2.4)	<0.001
Previous cancer (%)	34 (0.9)	1300 (1.2)	0.11
Previous CABG (%)	10 (0.3)	1812 (1.6)	<0001
Previous PCI (%)	8 (0.2)	1429 (1.3)	<0001

AM, acute myocarditis; CABG, coronary artery by-pass grafting; HF, heart failure; PCI, percutaneous coronary intervention; UCP, unexplained chest pain.

prevalence of diabetes was 4.7% and 8.7% in the AM and UCP groups, respectively (p<0.001), while hypertension was found in 3.7% and 12.7% of patients of AM and UCP, respectively (p<0.001).

The most common presenting symptom for patients with AM was chest pain (82.9%), followed by dyspnoea (3.1%). Chest pain was also the most common presenting symptom in the UCP group (93.4%). In our material, a very small proportion presented with cardiac shock or resuscitated cardiac arrest, but the rate of cardiac shock/resuscitated cardiac arrest was significantly higher in the AM group relative to the UCP group (0.6% and 0.2%, respectively, for cardiac shock and 0.7% vs <0.1% for resuscitated cardiac arrest, p<0.001). Compared with patients with UCP, AM patients displayed higher levels of cardiac troponin (10 (0.92, 127) ng/L vs 0.03 (0.01, 2) ng/L; p<0.001) as well as C reactive protein (29 (6, 75) mg/L vs 5 (2.2, 8) mg/L; p<0.001) (table 2). ECG changes in the form of ST-segment elevation were more common in patients with AM (43.5% vs 4.7%, p<0.001, see table 3).

Registration of medication at discharge from hospital showed that patients with AM were less often treated with ACE inhibitors than patients with UCP (4% vs 10.5 %; p<0.001). The same was true for angiotensin receptor blockers (3.3% vs 8.8%; p<0.001) and diuretics (3.8% vs 12.4%; p<0.001). For more details on medications, see table 4.

Table 2 Presenting symptoms, clinical and laboratory findings at admission

	AM	UCP	P value
N	3792	109 934	
SPB mm Hg (median, IQR)	130 (120, 145)	148 (130, 165)	<0001
DBP mm Hg (median, IQR)	80 (70, 90)	84 (75, 93)	<0001
Pulmonary rales, n (%)			<0001
No	3625 (95.6)	107 075 (97.4)	
Yes	117 (3.1)	2089 (1.9)	
Presenting symptoms, n (%)			<0001
Chest pain	3144 (82.9)	102 678 (93.4)	
Dyspnoea	118 (3.1)	1209 (1.1)	
Cardiac shock	23 (0.6)	220 (0.2)	
Other symptoms	345 (9.1)	4507 (4.1)	
Resuscitated cardiac arrest (%)	26 (0.7)	n/a (<0.1)	<0001
VT/VF	15 (0.4)	n/a (<0.1)	
Laboratory findings			
Troponins (median, IQR)	10.0 (0.9, 127.0)	0.03 (0.01, 2.0)	<0.001
CRP (median, IQR)	29.0 (6.0, 75.0)	5.0 (2.2, 8.0)	<0.001
b-glucose (median, IQR)	6.0 (5.4, 6.9)	5.8 (5.2, 6.7)	<0.001
Haemoglobin (median, IQR)	144 (135, 152)	141 (132, 150)	<0.001
HbA1c (median, IQR)	36 (34, 39)	38 (35, 43)	<0001
Creatinine (median, IQR)	78 (68, 89)	75 (65, 88)	<0001
Cholesterol (total) (median, IQR)	4.3 (3.7, 5.1)	5.2 (4.5, 6.0)	<0001

Variables where the cumulative percentage does not add up to 100% are due to missingness.
AM, acute myocarditis; CRP, C reactive protein; DBP, diastolic blood pressure; HbA1c, haemoglobin A1c; SBP, systolic blood pressure; UCP, unexplained acute chest pain; VF, ventricular fibrillation; VT, ventricular tachycardia.

Short time outcome (during hospitalisation and at 30 days)

Patients with AM presented higher mortality at 30 days compared with patients with UCP (OR 3.75, 95% CI 1.9 to 7.3, p<0.001) when analysed by multivariable adjusted logistic regression. The mortality at 30 days is shown in figure 1.

The risk of developing HF during hospitalisation was increased for the AM cohort compared with patients with UCP (OR 5.4, 95% CI 4.44 to 6.49, p<0.001).

Table 3 Electrocardiographic findings at admission and at discharge

	AM, n (%)	UCP, n (%)	P value
N	3257	94 602	
ECG rhythm admission			<0.001
Sinus	3156 (96.9)	91 980 (97.2)	
Atrial fibrillation/ flutter	78 (2.4)	1868 (2.0)	
Other	53 (1.6)	754 (0.8)	
QRS complex			<0.001
Normal	2780 (85.3)	78 159 (82.6)	
Pacemaker	9 (0.3)	479 (0.5)	
Left bundle branch block	50 (1.5)	2586 (2.7)	
Pathologic Q-wave	77 (2.4)	3267 (3.5)	
Right bundle branch block	53 (1.6)	2100 (2.2)	
Other	254 (7.8)	6164 (6.5)	
ST-segment changes			<0.001
Normal	1036 (31.8)	64 564 (68.2)	
ST-elevation	1434 (44.0)	4406 (4.7)	
ST-depression	167 (5.1)	7245 (7.7)	
Pathologic T-wave	317 (9.7)	7762 (8.2)	
Other	289 (8.9)	8620 (9.1)	
Discharge ECG			0.002
Sinus	2979 (95.5)	86 626 (96.6)	
Atrial fibrillation/ flutter	50 (1.6)	1205 (1.3)	
Other	29 (0.9)	466 (0.5)	

Variables where the cumulative percentage does not add up to 100% are due to missingness.
AM, acute myocarditis; UCP, unexplained acute chest pain

Table 4 Medication at discharge from hospital

	AM, n (%)	UCP, n (%)	P value
ACE-inhibitors			<0.001
No	3160 (95.4)	84 480 (89.0)	
Yes	131 (4.0)	10 002 (10.5)	
Angiotensin II receptor blockers			<0.001
No	2472 (95.9)	59 310 (90.6)	
Yes	86 (3.3)	5773 (8.8)	
Oral anticoagulants			<0.001
No	3253 (98.2)	92 044 (97.0)	
Warfarin	33 (1.0)	2309 (2.4)	
Dabigatran	0 (0.0)	26 (<1)	
Rivaroxaban	4 (0.1)	40 (<1)	
Apixaban	52 (0.1)	90 (0.1)	
Digitalis			<0.001
No	3274 (98.9)	93 356 (98.5)	
Yes	17 (0.5)	1091 (1.2)	
Diuretics			<0.001
No	3169 (95.7)	82 695 (87.2)	
Yes	125 (3.8)	11 769 (12.4)	
MRA			0.003
No	1171 (98.3)	15 983 (97.0)	
Spironolactone	3 (0.3)	214 (1.3)	
Eplerenon	2 (0.2)	8 (<1)	
Statins			<0.001
No	3146 (95.0)	81 096 (85.5)	
Yes	154 (4.4)	13 344 (14.1)	
Other lipid lowering agents			0.048
No	3095 (99.2)	89 269 (99.0)	
Ezetimib	4 (0.1)	275 (0.3)	
Fibrates	0 (0.0)	83 (0.1)	
Other	2 (0.1)	135 (0.1)	
Nitrates			<0.001
No	3454 (98.3)	90 472 (95.5)	
Yes	35 (1.1)	3896 (4.1)	

Variables where the cumulative percentage does not add up to 100% are due to missingness.
AM, acute myocarditis; UCP, unexplained acute chest pain.

The number of patients admitted for AM undergoing coronary angiography increased by 3% per year during the study period (0.030, 95% CI 0.028 to 0.033, p<0.001, figure 2). In the last 4 years of the study, more than 60% of AM patients had a coronary angiography performed to rule out ACS. In comparison, 10.7% of the UCP group underwent coronary angiography.

Long-term outcome

Under a median follow up of 7.8 years (Q1, Q3; 3.4, 12.3), patients with AM displayed worse survival (OR 2.0, 95% CI 1.69 to 2.39, p<0.001) compared with UCP, as well as higher risk for HF (OR 2.3, 95% CI 1.81 to 2.93, p<0.001) and myocardial infarction (OR 1.3, 95% CI 1.11 to 1.62, p<0.001). No difference in the risk for stroke or bleeding was observed.

DISCUSSION

This large, nationwide, longitudinal cohort study demonstrated that patients with AM, despite being younger and having fewer comorbidities, experience worse short-term and long-term survival compared with those admitted with UCP. Additionally, AM patients face a significantly higher risk for developing HF, both during hospitalisation and in the long term, compared with UCP. These findings challenge the common perception of AM as a

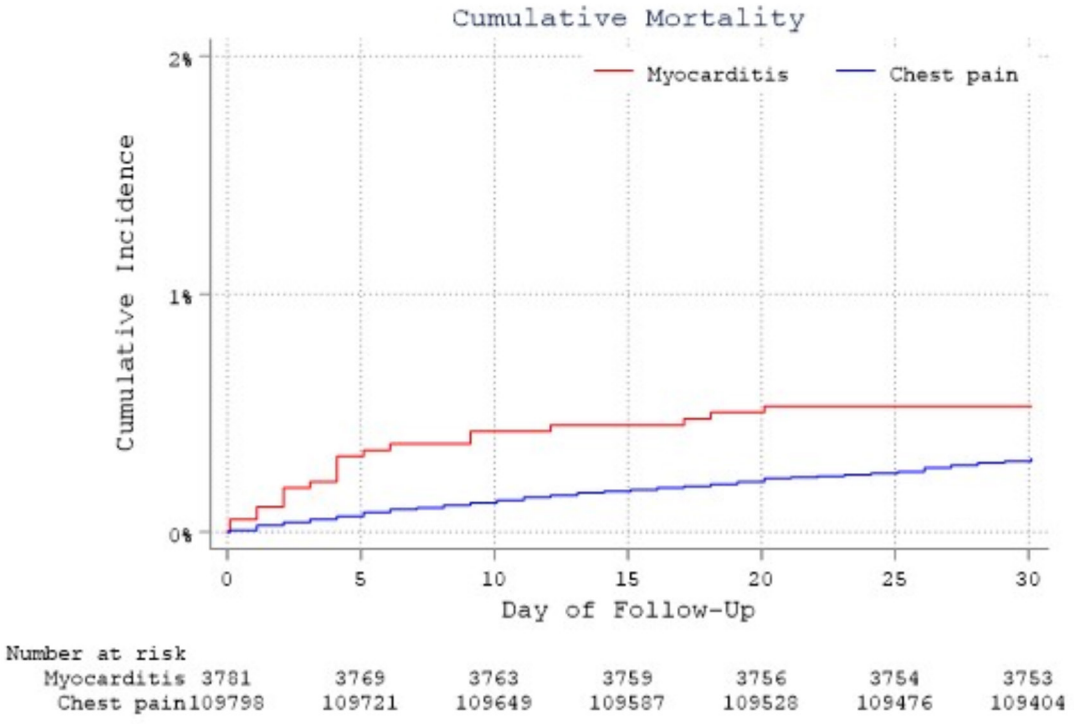

Figure 1 All-cause mortality for patients with acute myocarditis and acute unexplained chest pain at 30 days.

benign condition and emphasise the need for structured follow-up for these patients over both the short term and long term.

Consistent with prior research, our study confirmed that AM predominantly affects young male patients.[2 3 9] Recent studies have reported a very low mortality rate among patients with uncomplicated myocarditis,[3] reinforcing the view of AM as a generally benign condition. However, while the absolute number of deaths in our study was small, the mortality risk was notably higher compared

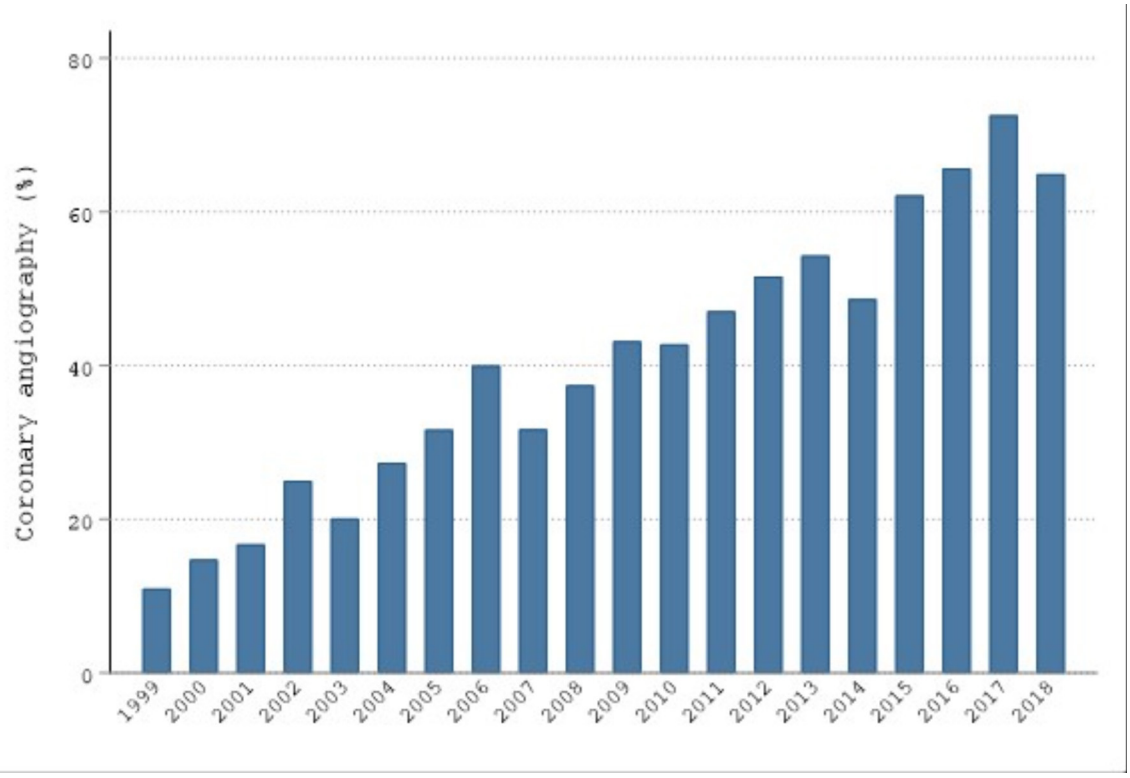

Figure 2 Percentage of patients hospitalised for suspected AM undergoing coronary angiography during 1999–2018. AM, acute myocarditis.

with UCP patients—who were both older and had more comorbidities. This discrepancy may be explained by our inclusive approach, which analysed all AM cases rather than stratifying them into uncomplicated and complicated subgroups, as some previous studies have done[3 8 21]. The higher mortality risk aligns with earlier research indicating an increased risk of 1-year mortality in myocarditis patients compared with healthy controls[20]

The increased short-term and long-term risk of HF in the AM cohort is particularly striking, especially given that UCP patients in our study had a higher prevalence of comorbidities such as hypertension, a common cause of HF.[22] This underscores the importance of consistent monitoring for AM patients, especially given the variability in follow-up practices across medical centres. A prior study of patients discharged with an HF diagnosis between 1987 and 2006 demonstrated a rise in HF-related hospitalisations among young adults (aged 18–44 years) during the study period.[23] This trend suggests that undiagnosed or subclinical AM may contribute to these patterns, reinforcing the link between myocarditis and the subsequent development of HF and dilated cardiomyopathy.[21]

Our findings are consistent with previous research, which indicated a clinically significant long-term risk of HF and the need for pacing devices or implantable cardioverter defibrillator (ICD) implantation following myocarditis compared with matched controls from the general population.[24] The same has also been reported in an earlier nationwide Swedish cohort study.[20] The increased long-term risk for cardiovascular events such as myocardial infarction in AM may be partly related to inflammation, a shared pathological mechanism in myocarditis and unstable atherosclerotic plaques leading to myocardial infarction.[25–27] Further research is needed to confirm and better understand this relationship.

The increase in coronary angiographies among AM patients during the study period likely reflects evolving clinical practices, improved procedure availability and the adoption of high-sensitivity troponins, which enable the detection of lower cardiac biomarker levels. Additionally, the 2013 European Society of Cardiology position paper on AM, which recommends the exclusion of coronary artery disease, may have further contributed to this trend.[2]

Strengths and limitations

A key strength in this study lies in its nationwide design, which includes all patients admitted to CCUs across Sweden. The registry-based approach ensures minimal loss to follow-up, providing robust longitudinal data. Additionally, to our knowledge, there is very limited prior research comparing these two patient groups.

Nonetheless, there are certain limitations to consider. Although the SWEDEHEART registry has undergone rigorous validation, there remains a risk of inaccurate data reporting. As a retrospective analysis of registry data, the study is subject to inherent limitations. A key limitation is the potential for misclassification, as differentiating AM from UCP remains a considerable clinical challenge. Registry data, while valuable for large-scale observational analyses, often lack the granularity of detailed clinical, imaging and biomarker information necessary for precise diagnosis. The absence of standardised criteria across different healthcare centres may further contribute to variability in classification.

Differentiating between AM and pericarditis in clinical practice is challenging, and patients diagnosed with pericarditis were not included in our analyses. This may potentially result in missed cases of AM. Also, diagnosing AM is notoriously complex, suggesting that this study may underestimate the true incidence of hospitalisations due to this condition. Studies have shown a correlation between outcome in AM and findings on CMR.[28 29] In this study, data collection of certain parameters, including left ventricular ejection fraction and CMR findings, which would have been of interest, was not possible as they are not recorded in the SWEDEHEART registry. Therefore, the AM group could not be subclassified in, for example, non-fulminant and fulminant. Elevated admission and peak troponins (plasma cardiac troponin T >50 ng/L) are associated with worse prognosis,[30] examining differences between groups with troponin levels above and below this threshold would be interesting in future studies. In addition to troponin, there are also many new biomarkers with promising results.[31] The UCP group is heterogeneous and, particularly in the earlier years of the study period before CMR became widely available, may have included cases of non-ischaemic acute myocardial injury as well as myocardial infarction with non-obstructive coronary arteries.

CONCLUSIONS

The findings of this study indicate that patients with AM face a greater risk of both 30 days and long-term mortality as well as an increased likelihood of developing HF compared with patients with UCP. These results highlight the importance of accurately distinguishing AM from UCP in clinical practice. Further research is needed to develop robust risk stratification strategies for AM patients to better predict and mitigate long-term complications.

Contributors MB and EBol designed the study. EO provided data from the SWEDEHEART registry and performed the statistical analyses. MB drafted the first version of the manuscript. EBob, CLP, CH, NB, EO and EBol all revised and approved the final version of the manuscript. EBol is the guarantor of the manuscript.

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Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval The study was approved by the Swedish Ethical Review Authority (Dnr 2021-04026) and is in compliance with the Declaration of Helsinki.

Provenance and peer review Not commissioned; externally peer reviewed.

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