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Prognostic implications of high-sensitivity cardiac troponin in patients with suspected myocardial infarction with or without psoriasis



To the Editor: Psoriasis is a chronic, immune-mediated skin disease that is potentially associated with cardiometabolic abnormalities.¹ High-sensitivity cardiac troponins (hs-cTn) I and T are the gold-standard biomarkers for the diagnosis of myocardial infarction (MI).² However, it is unknown whether the prognostic implications of hs-cTn and biochemical profiles (including lipid levels and inflammatory markers) differ between patients with psoriasis and those without psoriasis presenting with suspected MI.

We conducted a retrospective, population-based cohort study through linkage among the Danish Civil

Registration System, Patient Registry, Prescription Registry, and Registry of Laboratory Results for Research. We included all individuals aged ≥ 18 years who presented between 2014 and 2016 with at least 1 of the following diagnoses coded during ≥ 24 hours of hospitalization: MI, unstable angina, suspected MI, other chest pain, or chest pain not otherwise specified. Psoriasis was defined as ≥ 1 International Classification of Diseases, Tenth Revision, code for psoriasis on or before the date of inclusion or if patients had ≥ 2 filled prescriptions for topical calcipotriol. The primary end point was major adverse cardiovascular events or a composite of recurrent MI, ischemic stroke, or death because of cardiovascular (CV) causes within the first 30 days and at 31 to 365 days, respectively. The secondary outcomes were recurrent MI, ischemic stroke, death

Table I. Demographics of patients with versus without psoriasis and with versus without an elevated troponin I concentration

Variable	Subjects without psoriasis (n = 47,397)		Subjects with psoriasis (n = 2041)	
	Normal troponin (n = 30,659)	Elevated troponin (n = 16,738)	Normal troponin (n = 1297)	Elevated troponin (n = 744)
Age (y)	58 (47-70)	71 (60-81)	63 (53-72)	71 (62-79)
Female sex	15,020 (49.0%)	5901 (35.3%)	669 (51.6%)	278 (37.4%)
Income				
SES: lowest	5506 (18.0%)	4079 (24.4%)	184 (14.2%)	135 (18.2%)
SES: below average	5476 (17.9%)	3963 (23.7%)	253 (19.5%)	196 (26.3%)
SES: average	6082 (19.8%)	3382 (20.2%)	276 (21.3%)	158 (21.2%)
SES: above average	6644 (21.7%)	2795 (16.7%)	304 (23.4%)	128 (17.2%)
SES: highest	6951 (22.7%)	2519 (15.1%)	280 (21.6%)	127 (17.1%)
Comorbidities				
Baseline DM	3423 (11.2%)	3041 (18.0%)	201 (15.5%)	174 (23.4%)
Hypertension	10,050 (32.8%)	9168 (54.8%)	512 (39.5%)	422 (56.7%)
Ischemic heart disease	4263 (13.9%)	2439 (14.6%)	203 (15.7%)	116 (15.6%)
Heart failure	1460 (4.7%)	1591 (9.5%)	68 (5.0%)	72 (9.7%)
Atrial fibrillation	2289 (7.5%)	1856 (11.1%)	120 (9.3%)	85 (11.0%)
Ischemic stroke	690 (2.3%)	554 (3.3%)	33 (2.5%)	21 (2.8%)
Chronic kidney disease	604 (2.0%)	1029 (6.2%)	42 (3.2%)	65 (8.7%)
COPD	1564 (5.1%)	1470 (8.8%)	90 (6.9%)	84 (11.3%)
Psoriasis duration (y)	-	-	9.5 (4.6-15.9)	9.9 (4.9-16.6)
Active psoriasis	-	-	399 (30.8%)	253 (34.0%)
Psoriatic arthritis	61 (0.2%)	25 (0.2%)	124 (9.6%)	61 (8.2%)
Psoriatic arthritis duration (y)	6.3 (3.1-11.3)	7.5 (2.6-9.8)	7.2 (3.9-12.6)	8.8 (3.2-14.3)
Rheumatoid arthritis	613 (2.0%)	361 (2.2%)	41 (3.2%)	32 (4.3%)
Rheumatoid arthritis duration (y)	8.6 (4.3-15.6)	9.9 (4.2-16.0)	7.6 (3.4-16)	8.35 (3.6-13.9)
Spondylitis	178 (0.6%)	82 (0.5%)	13 (1.0%)	8 (1.1%)
Spondylitis duration (y)	7.4 (3.6-15.6)	9.9 (3.8-16.4)	9.4 (5.4-16.2)	12.3 (2.6-19.0)

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Table I. Cont'd

Variable	Subjects without psoriasis (n = 47,397)		Subjects with psoriasis (n = 2041)	
	Normal troponin (n = 30,659)	Elevated troponin (n = 16,738)	Normal troponin (n = 1297)	Elevated troponin (n = 744)
Medication				
Statins	8.6 (28.0%)	5579 (33.3%)	447 (34.5%)	265 (35.6%)
Antiplatelets	7587 (24.8%)	5509 (33.0%)	401 (31.0%)	276 (37.1%)
Proton pump inhibitor	7283 (23.8%)	4400 (26.3%)	364 (28.1%)	213 (28.6%)
NSAID	5081 (16.6%)	2437 (14.6%)	262 (20.2%)	115 (15.5%)
Anticoagulants	2278 (7.4%)	1839 (10.1%)	118 (9.1%)	72 (9.7%)
Methotrexate	258 (0.8%)	164 (1.0%)	68 (5.2%)	47 (6.3%)
Acitretin	10 (<0.1%)	6 (<0.1%)	7 (0.5%)	5 (0.7%)
Cyclosporin	20 (0.1%)	36 (0.2%)	<3	<3
Biological agents	252 (0.8%)	152 (0.9%)	76 (5.9%)	45 (6.1%)
Admission diagnosis				
Chest pain	12,325 (40.2%)	1757 (10.5%)	520 (40.1%)	72 (9.7%)
Suspected MI	15,063 (49.1%)	3096 (18.5%)	621 (47.9%)	143 (19.2%)
UA	1684 (5.5%)	688 (4.1%)	84 (6.5%)	33 (4.4%)
MI	1587 (5.2%)	11,197 (67.0%)	72 (5.6%)	496 (66.7%)
Troponin results*				
Troponin T hs ≤ 14 ng/L	11 (9.0-14.0)	63 (27.0-255.0)	12 (9.0-14.0)	73 (29.0-267.0)
hs-Troponin ≤ 25 ng/L	4 (3.0-9.0)	220 (68.0-1254.0)	5 (2.0-8.0)	321.5 (107.0-1295.0)
Tnl-Ultra ≤ 40 ng/L	10 (10.0-15.0)	289.5 (96.0-1620.0)	10 (10.0-15.0)	351.0 (83.0-1350.0)
Tnl Flex reagent ≤ 45 ng/L	15 (15.0-15.0)	295 (100.0-1338.0)	15 (15.0-15.0)	172.0 (100.0-914.0)
Blood test results, other				
Hgb (mmol/L)	8.6 (8.0-9.2)	8.4 (7.6-9.1)	8.5 (7.9-9.1)	8.3 (7.5-9.0)
CRP (mg/L)	3.0 (2.9-6.0)	5.9 (2.9-20.0)	3.0 (2.9-7.0)	7.3 (3.0-22.0)
Total cholesterol (mmol/L)	4.7 (3.9-5.5)	4.7 (3.8-5.6)	4.6 (3.8-5.6)	4.6 (3.8-5.5)
LDL cholesterol (mmol/L)	2.5 (1.9-3.3)	2.8 (2.0-3.6)	2.6 (1.8-3.3)	2.7 (1.9-3.5)
HDL cholesterol (mmol/L)	1.3 (1.0-1.6)	1.2 (1.0-1.5)	1.3 (1.0-1.6)	1.3 (1.0-1.5)
Triglyceride (mmol/L)	1.4 (0.9-1.4)	1.2 (0.8-1.8)	1.5 (1.0-2.2)	1.2 (0.8-1.8)
Glucose (mmol/L)	6.0 (5.4-6.9)	7.1 (6.0-8.9)	6.1 (5.5-7.2)	7.2 (6.1-9.1)
HbA1c (mmol/L)	37 (34.0-41.0)	39 (36.0-44.0)	38 (35.0-42.0)	39 (36.0-43.0)
Creatinine (μmol/L)	74 (64.0-86.0)	85 (70.0-106.0)	74 (64.0-86.0)	85 (69.0-105.0)
eGFR (mL/min/1.73 m ²)	84 (71.0-90.0)	72 (54.0-88.0)	83 (68.0-90.0)	72 (53.0-87.0)

COPD, Chronic obstructive pulmonary disease; CRP, C-reactive protein; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; Hgb, hemoglobin; hs, high-sensitivity; LDL, low-density lipoprotein; MI, myocardial infarction; NSAID, non-steroidal anti-inflammatory drug; SES, socioeconomic status; UA, unstable angina.

*The numbers next to each troponin assay signify their 99th percentile upper reference limit.

because of CV causes, death because of any cause, and coronary revascularization.

Multivariable logistic regression was used to calculate odds ratios, with corresponding 95% confidence intervals, of the association of elevated hs-cTn level (and the presence of psoriasis) with each end point. Interactions between hs-cTn and psoriasis were explored using the likelihood-ratio test for regression models with and without the interaction term. A 2-sided *P* value of <.05, when corrected for multiple testing using the Benjamini-Hochberg procedure, was considered statistically significant.

A total of 47,397 patients without psoriasis and 2041 with psoriasis were enrolled. The demographics are shown in Table I. We found no significant differences in hs-cTn concentrations at initial

presentation between patients with psoriasis and those without psoriasis. The concentrations of routine blood parameters also did not appear to differ between patients with psoriasis and those without psoriasis. Individuals with elevated hs-cTn concentrations were older and more likely to be men compared with those who presented with normal hs-cTn concentrations. The prevalence of comorbidities and the use of medications were generally higher among subjects with an elevated hs-cTn concentration; however, no differences were apparent between those with psoriasis and those without psoriasis (Table I). We did not observe significant interactions between hs-cTn and psoriasis (Table II).

Our study found no significant differences in hs-cTn concentrations between patients with psoriasis

Table II. Odds ratios for events in patients without versus with an elevated troponin concentration stratified by the diagnosis of psoriasis*

Variable	Without psoriasis (n = 47,397)			With psoriasis (n = 2041)			P for interaction
	OR	95% CI	P	OR	95% CI	P	
End point at 30 d							
Major adverse cardiovascular events							
Unadjusted	11.1	10.0-12.4	<.001	8.2	5.0-13.5	<.001	.248
Adjusted	6.8	6.0-7.7	<.001	4.8	2.7-8.9	<.001	.348
Myocardial infarction							
Unadjusted	8.1	7.2-9.2	<.001	4.9	2.9-8.3	<.001	.065
Adjusted	6.6	5.7-7.7	<.001	3.7	2.0-7.2	<.001	.075
Ischemic stroke							
Unadjusted	3.3	2.2-5.0	<.001	-	-	-	-
Adjusted	1.9	1.2-3.1	.012	-	-	-	-
Death from any cause							
Unadjusted	16.9	14.1-20.3	<.001	30.4	9.4-97.9	<.001	.332
Adjusted	6.4	5.0-8.1	<.001	11.2	2.6-49.2	.001	.292
Cardiovascular death							
Unadjusted	33.4	24.6-45.2	<.001	-	-	-	-
Adjusted	10.8	7.5-15.5	<.001	-	-	-	-
Coronary revascularization							
Unadjusted	9.1	8.5-9.7	<.001	7.1	5.2-9.7	<.001	.123
Adjusted	8.9	8.3-9.7	<.001	7.4	5.1-10.7	<.001	.212
End point at 31-365 d							
Major adverse cardiovascular events							
Unadjusted	4.4	3.9-4.9	<.001	3.2	2.0-5.4	<.001	.276
Adjusted	2.4	2.1-2.8	<.001	2.0	1.1-3.8	.026	.610
Myocardial infarction							
Unadjusted	4.5	3.9-5.3	<.001	3.3	1.6-6.6	.001	.378
Adjusted	2.8	2.3-3.4	<.001	2.5	1.0-6.4	.051	.928
Ischemic stroke							
Unadjusted	1.7	1.3-2.1	<.001	1.7	0.5-6.1	.378	.939
Adjusted	1.2	0.9-1.7	.294	1.4	0.3-7.7	.634	.814
Death from any cause							
Unadjusted	3.9	3.6-4.3	<.001	2.8	1.9-4.2	<.001	.111
Adjusted	1.6	1.4-1.8	<.001	1.2	0.7-2.0	.468	.250
Cardiovascular death							
Unadjusted	6.6	5.5-7.9	<.001	3.7	1.8-7.7	<.001	.134
Adjusted	2.7	2.2-3.5	<.001	1.6	0.6-3.9	.326	.169
Coronary revascularization							
Unadjusted	2.0	1.8-2.3	<.001	1.1	0.6-2.0	.678	.047
Adjusted	1.50	1.3-1.7	<.001	1.10	0.5-2.3	.803	.323

OR, Odds ratio.

*The regression models were adjusted for age, sex, diabetes, ischemic heart disease, heart failure, chronic obstructive pulmonary disease, statin use, total cholesterol, estimated glomerular filtration rate, and C-reactive protein.

and those without psoriasis, and we generally observed a striking similarity in the biochemical profiles between these 2 groups. These findings may suggest that the previously reported increased incidence of CV disease in patients with psoriasis is predominantly driven by traditional, modifiable CV risk factors rather than systemic inflammation. In keeping with this, in a comparative study from Denmark,^{3,4} traditional CV risk factors, including family history, physical activity, and smoking, were

more frequently observed in patients with psoriasis than in the general population. Nevertheless, because observational studies cannot establish causality, only association, further research is warranted.

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Conflicts of interest

With no relation to the work reported in this article, Dr Guldmann has received honoraria as a speaker for Medistim ASA. With no relation to the work reported in

this paper, Dr Pareek has received honoraria as a speaker for AstraZeneca, Bayer, Boehringer Ingelheim, and Janssen-Cilag, and for serving on advisory boards for AstraZeneca and Janssen-Cilag. With no relation to the work reported in this paper, Dr Hjuler has been a consultant and adviser for AbbVie, LEO Pharma, and Novartis and has received speaking fees from AbbVie, LEO Pharma, Novartis, Janssen, and CSL Behring. With no relation to the work reported in this paper, Dr Egeberg has received research funding from Pfizer, Eli Lilly, Novartis, Bristol-Myers Squibb, AbbVie, Janssen Pharmaceuticals, the Danish National Psoriasis Foundation, the Simon Spies Foundation, and the Kgl Hofbundtmager Aage Bang Foundation and honoraria as a consultant and/or speaker from AbbVie, Almirall, LEO Pharma, Galápagos NV, Sun Pharmaceuticals, Samsung Bioepis Co Ltd, Pfizer, Eli Lilly and Company, Novartis, Galderma, Dermavant, UCB, Mylan, Bristol-Myers Squibb, and Janssen Pharmaceuticals. Drs Kaiser and Kragholm have no conflicts of interest to declare.

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