### Review Article Factors influencing the development of heart failure after PCI in patients with acute coronary syndrome: a meta-analysis

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Abstract: Objective: To systematically analyze the factors associated with heart failure (HF) development after percutaneous coronary intervention (PCI) in patients with acute coronary syndrome (ACS). Methods: Relevant literature on risk factors for HF following PCI in ACS patients were retrieved from PubMed, Embase, The Cochrane Library, Web of Science, and Medline buildup to September 2024. Two independent investigators conducted literature screening, quality assessment, and data extraction based on inclusion and exclusion criteria. Meta-analysis was performed using Stata 12.0 software. Results: A total of 20 papers were included, comprising 45,578 patients of whom 4,345 ACS patients developed HF after PCI. Meta-analysis identified several predictors of post-PCI HF in ACS patients, including advanced age [odds ratio (OR) =1.04, 95% confidence intervals (CI): 1.03-1.06], female gender (OR=1.43, 95% CI: 1.18-1.72), history of hypertension (OR=1.54, 95% CI: 1.31-1.80), history of diabetes mellitus (OR=1.55, 95% Cl: 1.39-1.72), previous myocardial infarction (OR=1.58, 95% Cl: 1.11-2.23), anterior wall myocardial infarction (OR=2.22, 95% CI: 1.89-2.61), reduced left ventricular ejection fraction (LVEF) (OR=1.40, 95% CI: 1.21-1.62), elevated white blood cell count (OR=1.14, 95% CI: 1.07-1.22), atrial fibrillation [hazard ratio (HR) =2.14, 95% CI: 1.11-4.12], increased heart rate (OR=1.03, 95% CI: 1.02-1.04), elevated Pentraxin-3 (PTX3) levels (OR=2.67, 95% CI: 1.45-4.93), and decreased myocardial contractility (HR=1.18, 95% CI: 1.10-1.26). Notably, complete revascularization (HR=0.29, 95% CI: 0.10-0.86) was identified as a protective factor. Sensitivity analysis confirmed the robustness of these findings. Conclusions: Advanced age, female gender, history of hypertension and diabetes, previous myocardial infarction, anterior wall myocardial infarction, decreased LVEF at admission, increased white blood cell count, atrial fibrillation at admission, increased heart rate, elevated PTX3 levels, and impaired myocardial contractility were risk factors for HF development after PCI in ACS patients. Conversely, complete revascularization was associated with a lower risk of post-PCI HF.

Keywords: Acute coronary syndrome, PCI, heart failure, influencing factors, meta-analysis

#### Introduction

Acute coronary syndrome (ACS) is a common cardiovascular condition characterized by clinical manifestations such as angina pectoris and tachycardia [1]. Currently, percutaneous coronary intervention (PCI) is one of the most effective treatment strategies for ACS, as it can alleviate clinical symptoms. However, despite its therapeutic benefits, some patients remain at risk of developing adverse cardiac events after PCI, with heart failure (HF) being one of the most severe complications [2, 3]. HF is associated with significant impairment of cardiac function, leading to symptoms such as dyspnea, weakness, and edema, which markedly reduce patients' quality of life and worsen their prognosis [4]. Given that HF prevention is a primary focus in post-PCI management, identifying the key risk factors associated with HF development in ACS patients is essential for improving patient outcomes, enhancing quality

#### Table 1. PubMed search strategy

#1 ("Acute Coronary Syndrome"[Mesh]) OR (((((Acute Coronary Syndromes[Title/Abstract]) OR (Coronary Syndrome, Acute[Title/Abstract])) OR (Coronary Syndromes, Acute[Title/Abstract])) OR (Syndrome, Acute Coronary[Title/Abstract])) OR (Syndromes, Acute Coronary[Title/Abstract]))

#2 ("Percutaneous Coronary Intervention"[Mesh]) OR ((((((((Coronary Intervention, Percutaneous[Title/ Abstract]) OR (Coronary Interventions, Percutaneous[Title/Abstract])) OR (Intervention, Percutaneous Coronary[Title/Abstract])) OR (Intervention, Percutaneous Coronary[Title/Abstract])) OR (Percutaneous Coronary Interventions[Title/Abstract])) OR (Percutaneous Coronary Revascularization[Title/Abstract])) OR (Coronary Revascularization, Percutaneous[Title/Abstract])) OR (Coronary Revascularizations, Percutaneous[Title/Abstract])) OR (Percutaneous Coronary Revascularizations, Percutaneous[Title/Abstract])) OR (Percutaneous Coronary Revascularizations[Title/Abstract])) OR (Revascularization, Percutaneous Coronary[Title/Abstract])) OR (Revascularizations, Percutaneous Coronary[Title/Abstract])) OR (Revascularization, Percutaneous

#3 ("Heart Failure"[Mesh]) OR ((((((((((((((Cardiac Failure[Title/Abstract]) OR (Heart Decompensation[Title/Abstract])) OR (Decompensation, Heart[Title/Abstract])) OR (Congestive Heart Failure[Title/Abstract])) OR (Heart Failure, Congestive[Title/Abstract])) OR (Heart Failure, Right-Sided[Title/Abstract])) OR (Heart Failure, Right Sided[Title/Abstract])) OR (Right-Sided Heart Failure[Title/Abstract])) OR (Right Sided Heart Failure[Title/Abstract])) OR (Heart Failure, Left-Sided[Title/Abstract])) OR (Heart Failure, Left Sided[Title/Abstract])) OR (Left-Sided Heart Failure[Title/Abstract])) OR (Left Sided Heart Failure[Title/Abstract])) OR (Myocardial Failure[Title/ Abstract]))

#4 #1 AND #2 AND #3

of life, and guiding personalized therapeutic strategies. High-quality evidence plays a crucial role in clinical decision-making by providing reliable guidance for healthcare professionals [5]. However, existing studies on the risk factors for HF after PCI in ACS patients are heterogeneous and fragmented, making it challenging to derive consistent clinical recommendations. Therefore, this study aims to systematically quantify the available clinical evidence through meta-analysis, identify the key factors associated with post-PCI HF in ACS patients, and provide an evidence-based foundation for optimizing personalized treatment strategies, implementing effective interventions, and preventing postoperative HF.

#### Information and methods

#### PROSPERO registration

This study has been registered with PROSPERO (CRD42024595789).

#### Inclusion and exclusion criteria

Inclusion criteria: Studies were included if they met the following criteria: (1) Study design: cohort studies and case-control studies published in English; (2) Population: patients diagnosed with ACS, including unstable angina pectoris, non-ST-segment elevation myocardial infarction (N-STEMI), and ST-segment elevation myocardial infarction (STEMI); (3) Outcome measure: incidence of HF after PCI; (4) Clearly defined diagnostic criteria for both ACS and HF; and (5) Reported influencing factors, predictive factors, or associated factors for HF occurrence after PCI in ACS patients.

Exclusion criteria: Studies were excluded if they met any of the following criteria: (1) systematic evaluations, case reports, reviews, abstracts, conference papers, or animal experiments; (2) studies with incomplete or non-extractable data, where attempts to contact the authors were unsuccessful; (3) duplicated studies or studies without full-text availability; (4) studies lacking relevant outcome measures or with poorly defined outcome metrics; (5) non-English publications; and (6) literature deemed to be of low quality.

#### Literature search

This study was conducted according to the Meta-analysis of Observational Studies in Epidemiology (MOOSE) guidelines [6]. A comprehensive electronic search was performed in PubMed, Embase, The Cochrane Library, Web of Science, and Medline from their inception to September 2024. The search terms included "acute coronary syndrome", "unstable angina pectoris", "percutaneous coronary intervention", "coronary intervention, percutaneous", "heart failure", and "cardiac failure", along with other relevant keywords. The detailed search strategy for PubMed is provided in **Table 1**.

#### Literature screening and data extraction

Two independent researchers (Qian-Zhu Jiang and Qian Xu) screened and extracted data from eligible studies based on the inclusion and exclusion criteria. The screening results were cross-checked, and any discrepancies were initially resolved through discussion. If consensus could not be reached, a third researcher (Dong-Mei Wan) made the final decision on study inclusion. The extracted data included the first author, year of publication, country, sample size, number of cases, and risk factors associated with HF occurrence after PCI.

#### Literature quality assessment

The Newcastle-Ottawa Scale (NOS) [7] was used to assess the quality of the included studies. The NOS evaluation of cohort and case-control studies comprises three aspects: selection of study participants, comparability between groups, and outcome measurements. The scale has a total score of 9 points, with studies classified as low quality (0-4 points). moderate quality (5-6 points), and high quality  $(\geq 7 \text{ points})$ . Two independent, highly trained researchers (Qian-Zhu Jiang and Qian Xu) evaluated the quality of the included studies according to the NOS criteria and cross-checked the results. Any discrepancies were first resolved through discussion. If a consensus could not be reached, a third researcher made the final decision.

#### Statistical methods

EndNote X9.1 software was used for literature de-duplication and screening, while Stata 12.0 was employed for statistical analysis. The odds ratio (OR) or hazard ratio (HR) was used as the effect size measure. Notably, due to the limited number of studies available for each risk factor, when both OR and HR were reported in a study, HR values were treated as OR for the purpose of meta-analysis. The 95% confidence intervals (CI) were calculated to assess outcome variability. Heterogeneity among the included studies was evaluated using the Q test and  $I^2$  statistic. If P>0.10 or I<sup>2</sup><50%, heterogeneity was considered insignificant, and a fixed-effects model was applied for meta-analysis. If P≤0.10 and I<sup>2</sup> ≥50%, significant heterogeneity was assumed, and a random-effects model was used. In cases of substantial heterogeneity, subgroup analysis, meta-regression, and sensitivity analysis were performed to identify potential sources of variability. Sensitivity analysis was conducted by omitting each study in turn to assess the robustness of the pooled results and determine the influence of individual studies on the overall risk estimates. Egger's linear regression test and Beggs' test were used to detect publication bias. Differences were considered statistically significant at P<0.05.

#### Results

#### Literature screening

A total of 7,862 documents were retrieved from the selected databases. After applying the inclusion and exclusion criteria, 20 eligible studies were included in the final analysis [8-27]. The literature screening process is shown in **Figure 1**.

## Characteristics of included studies and quality assessment

A total of 20 studies [8-27] were included in the meta-analysis, involving 45,578 patients, among whom 4,345 ACS patients developed HF after PCI. The included studies consisted of 6 case-control studies and 14 cohort studies; conducted in various countries, including China [8, 12, 18, 21, 24, 26], Switzerland [9], Denmark [10, 19, 20], Germany [11], South Korea [13], France [14, 22], Poland [15], Serbia [23], the United States [25], Israel [27], and other European regions [16, 17]. Quality evaluation of the literature showed that 16 studies were of high quality, while 4 studies were of moderate quality, with NOS scores ranging from 6 to 8. Factors were included in the metaanalysis if they were reported in at least two studies. A total of 13 influencing factors were extracted from the included literature for multivariate analysis, including age, gender, hypertension, diabetes mellitus, previous myocardial infarction, anterior wall myocardial infarction, left ventricular ejection fraction (LVEF), white blood cell count, atrial fibrillation, complete revascularization, heart rate, pentraxin-3 (PTX3), and myocardial contractility. The latter was assessed using overall longitudinal strain. postsystolic shortening (PSS) wall number, and myocardial performance index MPI. Detailed information on the included studies and their

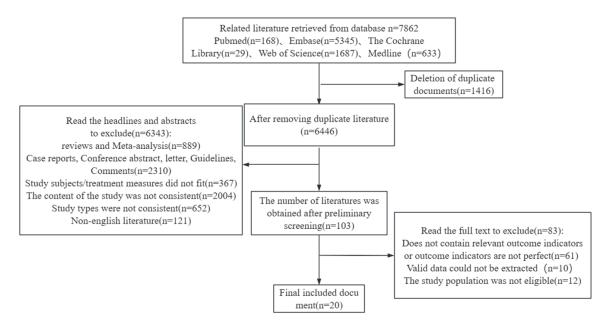


Figure 1. Literature screening process.

quality assessment results is presented in **Table 2**.

#### Meta-analysis results and forest plot

Age: Seven studies [12, 14, 17, 21, 22, 26, 27] reported the impact of age on the occurrence of HF after surgery in ACS patients. The heterogeneity test yielded P<0.001 and I<sup>2</sup>=84.2%, suggesting substantial heterogeneity across studies, and thus a random-effects model was applied for the meta-analysis. The results demonstrated that age was a risk factor for HF development after PCI in ACS patients (OR=1.04, 95% CI: 1.03-1.06, P<0.05), suggesting a positive correlation between age and HF risk. Specifically, each one-year increase in age was associated with a 4% higher risk of HF post-PCI, as detailed in Figure 2. Sensitivity analysis (Figure 3) revealed that excluding any single study did not alter the overall direction of the results, indicating the robustness and reliability of the findings. However, substantial heterogeneity persisted regardless of study exclusion, and its source could not be excluded. To explore potential sources of heterogeneity, meta-regression analysis was performed, incorporating variables such as geographic region (Asia vs. Europe), HF definitions and diagnostic criteria, single-center vs. multi-center study design, timing of HF occurrence (postoperative hospitalization vs. postoperative rehospitalization), and ACS subtypes (ACS vs. STEMI). However, none of these factors adequately explained the observed heterogeneity. Potential sources of heterogeneity were further analyzed, with surgical-related factors (e.g., surgical techniques) and postoperative management strategies being identified as possible contributors. Variability in patient characteristics and treatment protocols may have influenced the relationship between age and HF development, further contributing to heterogeneity.

Gender: Three studies [16, 22, 25] investigated the effect of gender on the occurrence of HF after PCI in ACS patients. The heterogeneity test yielded P=0.031 and I<sup>2</sup>=71.1%, indicating substantial heterogeneity across studies. Therefore, a random-effects model was used for the meta-analysis. The results indicated that female gender was a risk factor for the development of HF in ACS patients following PCI. Specifically, female patients had a 1.43fold higher risk of developing HF after PCI compared to their male counterparts (OR=1.43, 95% CI: 1.18-1.72, P<0.05), as shown in Figure 4. Sensitivity analysis (Figure 5) revealed that the results remained stable, with no directional changes after excluding any individual study, indicating the robustness and reliability of the results. The primary source of heterogeneity was identified in the study by Auffret

Serial No.	Author	Year of publication	Nation	Total sample size	Type of study	Gender (male/female)	Postoperative HF (case)	Influencing factors	NOS score
1	Wen Wen [8]	2023	China	416	Cohort studies	324/92	41	13(14(15)	8
2	Tomandlova [9]	2015	Switzerland	262	Cohort studies	185/77	70	1112	8
3	Ravnkilde [10]	2021	Denmark	235	Prospective + retrospective cohort study	181/54	57	(16)	7
4	Petrosyan [11]	2021	Germany	169	Prospective cohort study	135/34	5		7
5	Lieyou Li [12]	2021	China	377	Retrospective cohort study	324/53	54	1418109	6
6	Dong-Hyeon Lee [13]	2010	South Korea	137	Prospective cohort study	108/29	91	(12)	7
7	Leboube [14]	2023	France	407	Prospective cohort study	343/64	23	1319	8
8	Kowara [15]	2017	Poland	278	Retrospective cohort study	210/68	35	20	7
9	Elia [16]	2023	Center of Europe	14699	Retrospective cohort study	10121/4578	1225	2	7
10	Filippo [17]	2023	Center of Europe	14699	Retrospective cohort study	10121/4578	1225	14159234571	7
11	Chen [18]	2014	Taiwan, China	959	Retrospective cohort study	787/172	67	26	6
12	Brainin [19]	2019	Denmark	428	Retrospective cohort study	314/114	155	27)28)	7
13	Bjerregaard [20]	2024	Denmark	386	Retrospective cohort study	282/104	140	2930	7
14	Li Li [21]	2021	China	778	Case-control study	582/196	108	3113118	8
15	AUFFRET [22]	2016	France	6282	Case-control study	4850/1432	589	12343711336	6
16	Rajic [23]	2018	Serbia	148	Case-control study	111/37	16	83	7
17	Xu [24]	2018	China	405	Case-control study	351/54	85	353637	6
18	Kelly [25]	2011	United States	2946	Case-control study		153	5724	7
19	Cheng [26]	2022	China	609	Case-control study	498/111	97	138397	7
20	Massalha [27]	2016	Israel	958	Prospective cohort study	771/187	109	15674	7

Table 2. Basic characteristics of the included literature

Note: ① Age, ② Sex (female), ③ Hypertension, ④ Diabetes mellitus, ⑤ Previous myocardial infarction, ⑥ Anterior wall myocardial infarction, ⑦ Left ventricular ejection fraction (LVEF), ⑧ White cell count, ⑨ Atrial fibrillation, ⑩Complete Revascularization, ⑪ Heart rate, ⑫ Pentraxin-3 (PTX3), ③ Lymphocyte count, ④ Eosinophils (EO) count, ⑤ EO %, ⑥ Myocardial contractility-reduced overall longitudinal strain, ⑪ Glomerular filtration rate, ⑱ Killip functional class, ⑲ Left ventricular end-systolic pressure (LVFP) during initial hospitalization, ⑳ Platelet distribution width, ⑳ Chronic kidney disease, ㉒ Pulmonary disease, ㉓ GRACE risk score, ㉒ Peripheral arterial disease, ㉓ Cardiogenic shock on admission, ㉓ Admission glucose level, ㉒ Myocardial contractility-contraction postsystolic shortening (PSS) wall number, ⑳ Postsystolic index (PSI), ㉓ Myocardial contractility-myocardial function index (MPI), ⑳ Decreased systolic ejection time (ET), ⑨ D-dimer, ㉓ Arrhythmia history, ㉓ Systolic blood pressure, ㉓ Neutrophil count, ֍ Hs-CRP, ـ MT-proBNP, ㉑ Number of diseased veins, 휗 Peak cardiac troponin I (TNI) levels, ⑲ Left ventricular end-diastolic diameter (LVEDD).

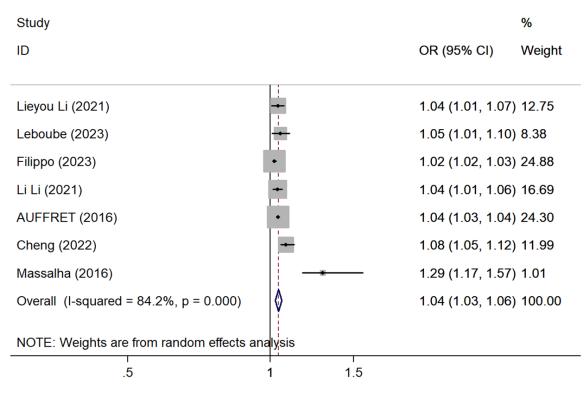
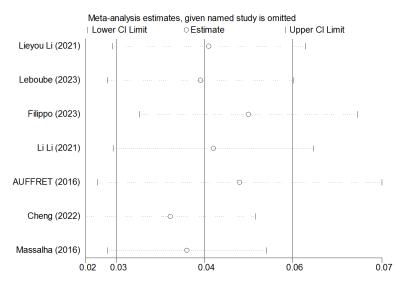


Figure 2. Meta-analysis of the effect of age on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

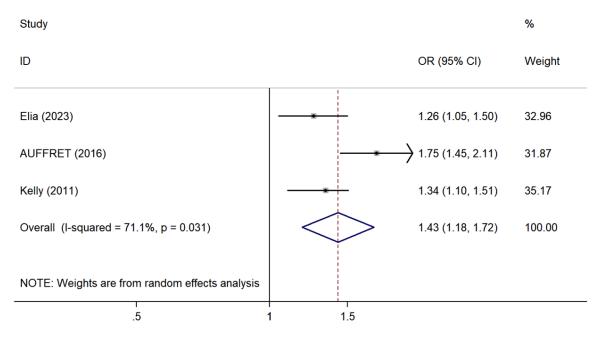


**Figure 3.** Sensitivity analysis of the effect of age on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

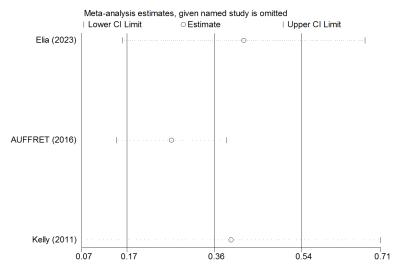
et al. (2016) [22], which focused on HF occurring during hospitalization after PCI. This was in contrast to the other two studies, which predominantly assessed long-term outcomes.

Diabetes mellitus: Five studies [12, 17, 22, 25, 27] evaluated the impact of diabetes mellitus on the development of HF after PCI in ACS patients. The heterogeneity test vielded P=0.626 and I<sup>2</sup>=0.0%, suggesting no significant heterogeneity among the studies. Therefore, a fixed-effects model was employed for the meta-analysis. The results showed that diabetes mellitus was a risk factor for postoperative HF in ACS patients. The risk of developing HF after PCI in diabetic ACS patients was 1.55 times higher compared to non-diabetic ACS patients (OR=1.55, 95% CI: 1.39-1.72, P<0.05), as

illustrated in **Figure 6**. Sensitivity analysis (**Figure 7**) confirmed the stability of the results, showing no directional changes after excluding any individual study, indicating that the results were consistent and reliable.



**Figure 4.** Meta-analysis of the effect of gender on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).



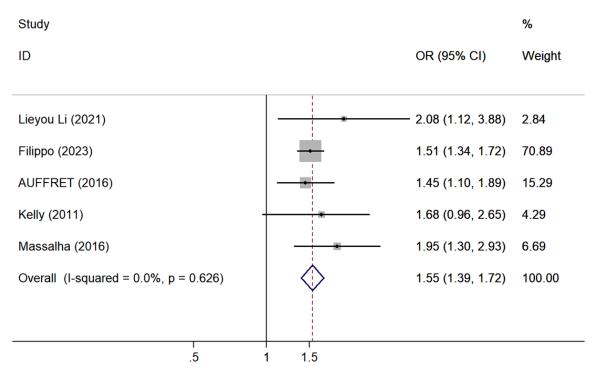
**Figure 5.** Sensitivity analysis of the effect of gender on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

Hypertension: Three studies [14, 21, 22] explored the effect of hypertension on the development of HF after PCI in ACS patients. The heterogeneity test showed P=0.325 and  $l^2=11.0\%$ , suggesting acceptable heterogeneity among studies. Consequently, a fixed-effects model was used for the meta-analysis. The analysis showed that hypertension was a risk factor for postoperative HF in ACS patients.

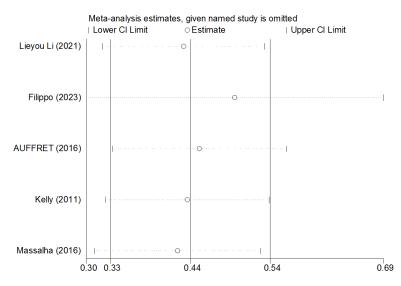
ACS patients with hypertension had a 1.54-fold increased risk of developing HF after PCI compared to those without hypertension (OR=1.54, 95% CI: 1.31-1.80, P<0.05), as detailed in **Figure 8**. The sensitivity analysis (**Figure 9**) showed that excluding any individual study did not alter the direction of the results, further supporting their robustness and reliability.

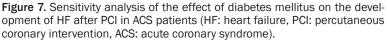
Previous myocardial infarction: Three studies [17, 25, 27] examined the effect of a history of myocardial infarction on the occurrence of HF after surgery in ACS patients. The heterogeneity test reveal-

ed P=0.027 and I<sup>2</sup>=72.3%, suggesting substantial heterogeneity. Therefore, a random-effects model was applied for the meta-analysis. The results showed that previous myocardial infarction was a risk factor for postoperative HF in ACS patients. ACS patients with a history of myocardial infarction had a 1.58-fold higher risk of developing HF after PCI compared to those without such a history (OR=1.58, 95% CI:



**Figure 6.** Meta-analysis of the effect of diabetes mellitus on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

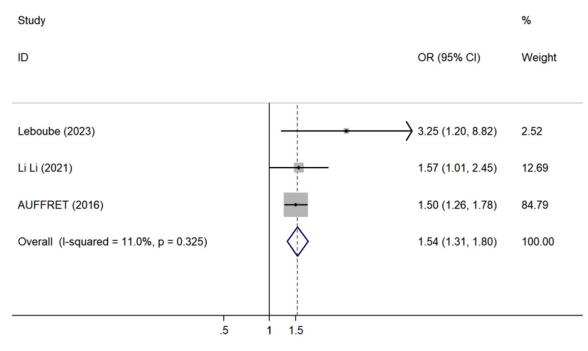




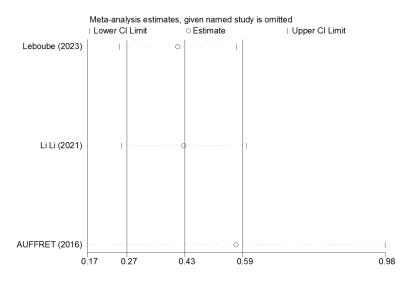
1.11-2.23, P<0.05), as depicted in **Figure 10**. The sensitivity analysis (**Figure 11**) showed that excluding any individual study did not alter the direction of the results, confirming their stability and reliability. The primary source of heterogeneity was identified in the study by Filippo et al. [17], which included ACS patients with three distinct types of stable angina, NS-TEMI, and STEMI. This differed from the other two studies, which primarily focused on STEMI patients, a subtype of ACS.

Myocardial infarction of the anterior wall: Two studies [22, 27] investigated the effect of anterior wall myocardial infarction as a clinical presentation on the development of HF after PCI in ACS patients. The heterogeneity test showed P=0.407 and  $I^2$ = 0.0%, suggesting no significant inter-study heterogeneity. Therefore, a fixed-effects model was used for the meta-

analysis. The results showed that anterior wall myocardial infarction was a risk factor for the postoperative development of HF in ACS patients. The risk of developing HF after PCI in ACS patients with anterior wall myocardial infarction was 2.22 times higher compared to



**Figure 8.** Meta-analysis of the effect of hypertension on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).



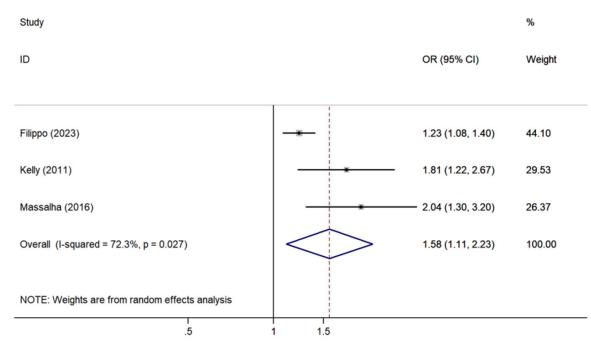
**Figure 9.** Sensitivity analysis of the effect of hypertension on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

ACS patients with other clinical manifestations (OR=2.22, 95% CI: 1.89-2.61, P<0.05), as shown in Figure 12.

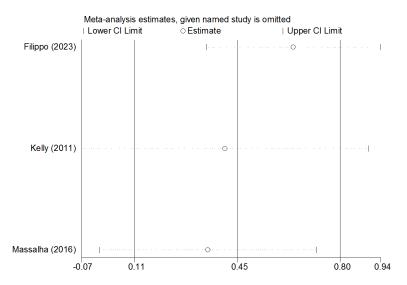
Left ventricular ejection fraction (LVEF): Five studies [17, 22, 25, 26, 27] reported the influence of decreased admission LVEF on the occurrence of HF after PCI in ACS patients. The

heterogeneity test yielded P<0.001 and I<sup>2</sup>=94.8%, suggesting substantial heterogeneity among the included studies, and thus a randomeffects model was applied for the meta-analysis. The results showed that a reduced admission LVEF was a risk factor for postoperative development of HF in ACS patients. In ACS patients undergoing PCI, those with a decreased admission LVEF had a 1.40fold higher risk of developing postoperative HF compared to those with a normal admission LVEF (OR=1.40, 95% CI: 1.21-1.62, P<0.05), as shown in Figure 13. Sensitivity analysis (Figure 14) confirmed the

robustness of these findings, as the direction of the results remained unchanged after the exclusion of any single study, indicating high stability and reliability. The primary source of heterogeneity, aside from variations in effect size indicators (OR and HR) across studies, was the study by Kelly et al. (2011) [25], which recruited participants from 123 medical cen-



**Figure 10.** Meta-analysis of the effect of prior myocardial infarction on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).



**Figure 11.** Sensitivity analysis of the effect of prior myocardial infarction on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

ters worldwide, encompassing a broader geographic distribution and a more diverse study population than the other four included studies.

White blood cell count: Two studies [21, 23] assessed the association between white blood cell count and HF development in ACS patients

after PCI. The test for heterogeneity showed P=0.233 and I<sup>2</sup>=11.0%, suggesting acceptable heterogeneity, and thus a fixed-effects model was employed for the meta-analysis. The results revealed that an elevated white blood cell count was a risk factor for postoperative HF in ACS patients; for every one-unit increase in white blood cell count, the risk of HF after PCI in ACS patients increases by 14% (OR=1.14, 95% CI: 1.07-1.22, P<0.05), as shown in Figure 15.

Atrial fibrillation: Two studies [12, 17] reported the impact of atrial fibrillation (AF) at admission on the occurrence

of postoperative HF in ACS patients. The heterogeneity test yielded P=0.100,  $I^2$ =63.1%, suggesting significant heterogeneity among studies. Therefore, a random-effects model was used for the meta-analysis. The results showed that ACS patients with AF had a higher risk of developing HF after PCI compared to those without AF (HR=2.14 95%CI: 1.11-4.12,

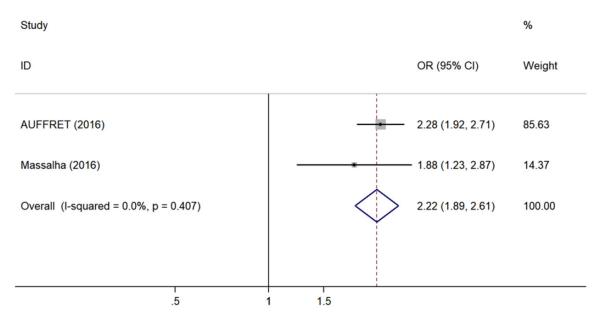


Figure 12. Meta-analysis of the effect of anterior wall myocardial infarction on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

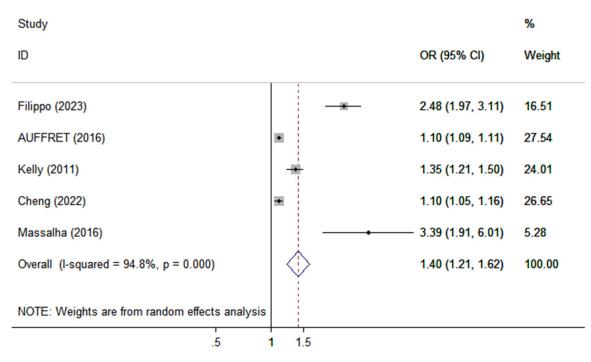


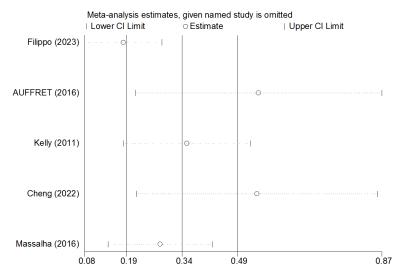
Figure 13. Meta-analysis of the effect of decreased admission LVEF on the development of HF after PCI in ACS patients (LVEF: left ventricular ejection fraction, HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

P<0.05), as detailed in **Figure 16**. An analysis of heterogeneity sources revealed that the primary contributing factor was study design differences. Specifically, Lieyou Li et al. (2021) [12] conducted a single-center study, whereas Filippo et al. (2023) [17] performed a multi-cen-

ter study, which may have introduced variability in patient populations and treatment protocols.

Complete revascularization: Two studies [12, 17] investigated the relationship between com-

#### Factors affecting the development of heart failure after PCI



**Figure 14.** Sensitivity analysis of the effect of decreased admission LVEF on the development of HF after PCI in ACS patients (LVEF: left ventricular ejection fraction, HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

plete revascularization and HF development in ACS patients after PCI. The heterogeneity test vielded P=0.024 and I<sup>2</sup>=80.4%, indicating substantial heterogeneity. Accordingly, a randomeffects model was applied for the meta-analysis. The results indicated that complete revascularization was associated with a lower risk of HF in ACS patients after PCI; ACS patients who underwent complete revascularization had only a 29% risk of developing HF after PCI compared to those who did not undergo complete revascularization (HR=0.29, 95% CI: 0.10-0.86, P<0.05), as detailed in Figure 17. To analyze the main sources of heterogeneity, study design differences were identified as a key factor. Similar to the AF analysis, Lieyou Li et al. (2021) [12] conducted a single-center study. whereas Filippo et al. (2023) [17] was a multicenter study, likely contributing to the observed heterogeneity.

*Heart rate:* Three studies [21, 22] reported the impact of admission heart rate on the development of HF in ACS patients after PCI. The heterogeneity test yielded P=0.422, I<sup>2</sup>=0.0%, indicating no heterogeneity among the included studies. Therefore, a fixed-effects model was used for the meta-analysis. The results demonstrated that increased heart rate at admission was a risk factor for HF in ACS patients after PCI (OR=1.03, 95% CI: 1.02-1.04, P<0.05). This finding suggests that, after adjusting for poten-

tial confounding factors, each one-unit increase in heart rate at admission is associated with a 1.03-fold increase in the risk of developing HF in ACS patients after PCI, as shown in **Figure 18**.

Pentraxin-3 (PTX3): Two studies [12, 17] evaluated the relationship between PTX3 concentration and the risk of HF in ACS patients after PCI. The heterogeneity test yielded P=0.436 and I<sup>2</sup>=0.0%, suggesting no heterogeneity across studies. Thus, a fixedeffects model was used for the meta-analysis. The results showed that increased PTX3 concentration was a risk factor for the development of HF

in ACS patients after PCI (OR=2.67, 95% CI: 1.45-4.93, P<0.05). After adjusting for potential confounders, each one-unit increase in PTX3 concentration was associated with a 2.67-fold increase in the risk of developing HF in ACS patients after PCI, as shown in **Figure 19**.

Myocardial contractility: Three studies [10, 19, 20] investigated the association between myocardial contractility and the development of HF in ACS patients after PCI. The heterogeneity test showed P=0.325 and I<sup>2</sup>=8.0%, suggesting acceptable heterogeneity among studies. Therefore, a fixed-effects model was applied for the meta-analysis. The results demonstrated that decreased myocardial contractility was associated with an increased risk of HF in ACS patients after PCI; patients with impaired myocardial contractility had an 18% higher likelihood of developing HF after surgery compared to those with normal myocardial contractility (HR=1.18, 95% CI: 1.10-1.26, P<0.05), as detailed in Figure 20. Sensitivity analysis (Figure 21) validated the robustness of these findings, as the exclusion of any single study did not alter the overall direction of the results, indicating high stability and reliability.

#### Publication bias analysis

The Begg correlation test and Egger linear regression test were utilized to assess publica-

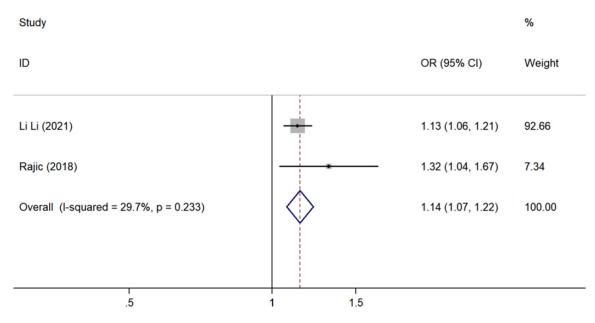


Figure 15. Meta-analysis of the effect of white cell count on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

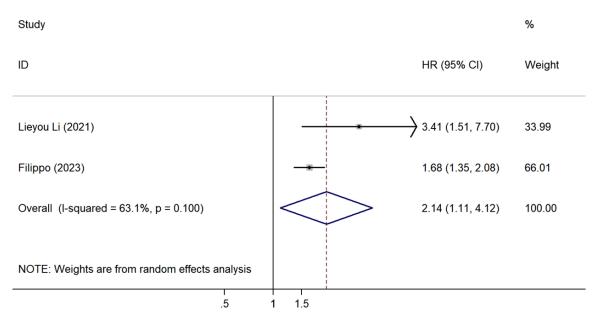


Figure 16. Meta-analysis of the effect of atrial fibrillation on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

tion bias for various risk factors, including age, gender, hypertension, diabetes mellitus, previous myocardial infarction, LVEF, and myocardial contractility, in relation to postoperative HF in ACS patients after PCI. The results showed that the *P*-values of both the Egger regression test and the Begg correlation test were greater than 0.05 for all examined variables, suggesting a low risk of publication bias and high stability of

the results. Additionally, publication bias analyses for age and diabetes were selected as representative examples, with results illustrated in **Figures 22** and **23**.

#### Discussion

Research on the factors influencing the development of HF after PCI in ACS patients has

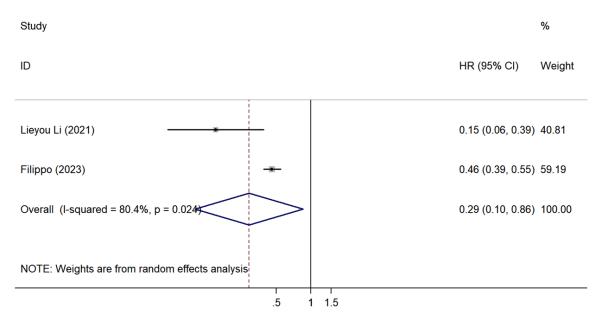
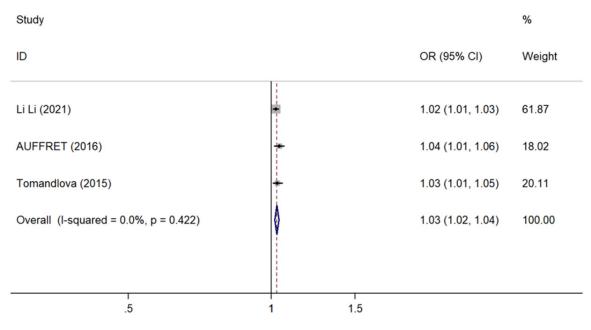
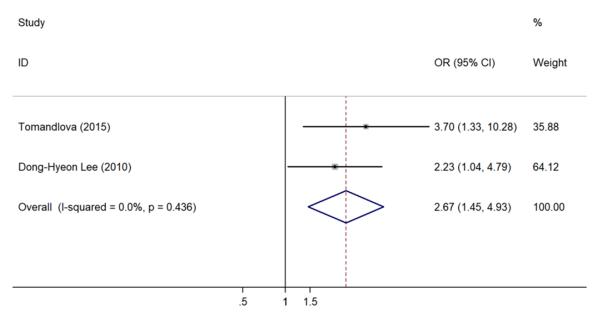


Figure 17. Meta-analysis of the effect of complete revascularization on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

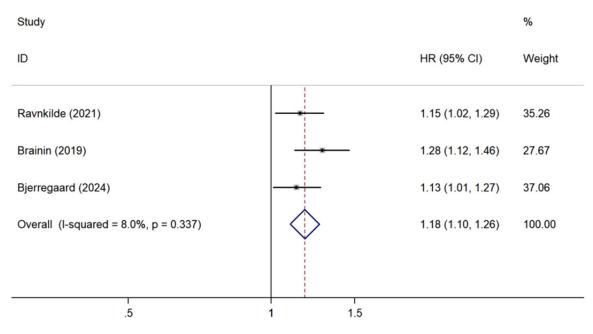


**Figure 18.** Meta-analysis of the effect of admission heart rate on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

been a major focus in clinical studies. Previous research suggests that post-PCI HF in ACS patients results from a complex interplay of factors, including pathological myocardial remodeling and excessive activation of the neuroendocrine system [28, 29]. However, existing evidence on the influencing factors of post-PCI development of HF in ACS patients remains fragmented and highly variable, limiting its applicability in clinical decision-making. In this study, we synthesized and analyzed data from 20 relevant publications and identified several potential contributors to HF after PCI in ACS patients. These include general sociodemographic factors, personal medical history, inflammation, myocardial function impairment,



**Figure 19.** Meta-analysis of the effect of PTX3 concentration on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).



**Figure 20.** Meta-analysis of the effect of myocardial contractility on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

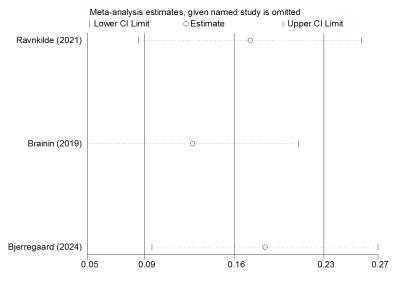
cardiac rhythm abnormalities, lesion location, and therapeutic interventions.

Influence of general sociodemographic factors on the development of HF after PCI in ACS patients

Our analysis indicates that increasing age is a risk factor for HF after PCI in ACS patients. HF

has been recognized as a geriatric syndrome, with a prevalence of up to 10% among individuals aged 70 years and older [30]. Aging is often accompanied by progressive organ degeneration, impaired self-repair capacity, and functional decline, all of which increase susceptibility to HF post-PCI [31]. In addition, studies have reported that most elderly ACS patients (≥70 years old) have preexisting condi-

#### Factors affecting the development of heart failure after PCI



**Figure 21.** Sensitivity analysis of the effect of myocardial contractility on the development of HF after PCI in ACS patients (HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

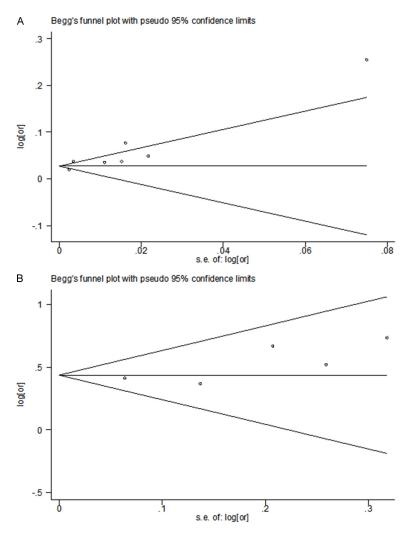
tions, such as hypertension and hyperlipidemia, along with weakened immune function and systemic deterioration, which can negatively impact postoperative prognosis [32]. Therefore, the risk of HF after PCI increases with age. Our findings also suggest that female sex is a risk factor for HF after PCI in ACS patients. A study by Donna et al. [33] found that women had a higher incidence of HF-related hospitalizations during follow-up after PCI for patients with acute myocardial infarction. Similarly, Kosmidou et al. [34] found that, despite comparable infarct sizes, smaller left ventricular volumes, and better ejection fractions, female patients had a higher rate of cardiovascular rehospitalization within one year of acute myocardial infarction than their male counterparts. These findings support our study's results, reinforcing the correlation between female sex and an increased risk of post-PCI HF. Several factors may contribute to this sex disparity. Compared to male patients, female patients often have a lower quality of life, a higher prevalence of comorbidities, and are more likely to have been prescribed β-blockers and angiotensin-converting enzyme inhibitors prior to hospitalization. The use of these medications may lead to a greater likelihood of hypertension and worse clinical outcomes [33], ultimately increasing the risk of HF in female patients.

Influence of personal disease history on the development of HF after PCI in ACS patients

The results of this study show that a history of hypertension increases the risk of HF after PCI in ACS patients. Some studies have established a correlation between hypertension and HF development. A 10-year follow-up analysis of 4,408 participants from the Cardiovascular Health Study and the Healthy Aging and Body Composition (ABC) study demonstrated that prehypertension is associated with an elevated risk of HF [35]. Conversely, long-term hypertension management has been shown to reduce the risk

of HF by 30% in younger individuals, by 50% in older adults, and by nearly 80% in elderly patients with a history of myocardial infarction [36]. The underlying mechanisms by which hypertension contributes to HF after PCI in ACS patients may include hypertension-induced microvascular damage, resulting in cardiac overload, as well as hypertension-driven left ventricular remodeling post-PCI, both of which can precipitate HF [37].

Diabetes mellitus is a well-established risk factor for HF and has been explicitly recognized in current clinical guidelines [38]. Diabetes accelerates atherosclerosis progression and has also been shown to impair myocardial function directly, contributing to cardiac dysfunction and diastolic impairment in ACS patients after PCI [39, 40]. Epidemiological investigations have reported that the prevalence of HF in diabetic patients ranges from 9%-22%, which is four times higher than in the general population [41]. Our findings are consistent with these above observations, demonstrating that ACS patients with a history of diabetes are at an increased risk of developing HF after PCI. Given these risks, clinicians should closely monitor the cardiac function of ACS patients with concomitant diabetes and implement early preventive strategies to reduce HF risk, thereby improving their prognosis. Furthermore, our study identifies prior myocardial infarction as a



**Figure 22.** Begg's anecdotal correlation test for the effect of age and diabetes mellitus on the development of HF after PCI in ACS patients (A: age, B: diabetes mellitus; HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

contributor to HF development. A history of myocardial infarction can compromise cardiac reserve capacity, making the heart more susceptible to HF when exposed to additional hemodynamic stressors.

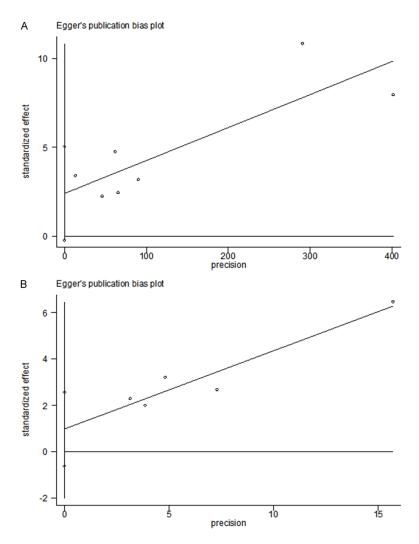
## Influence of inflammatory indicators on the development of HF after PCI in ACS patients

Inflammation plays an important role in the progression of atherosclerosis and significantly impacts clinical outcomes. Peripheral white blood cell count at admission has been identified as an independent predictor of major adverse cardiovascular events after PCI in ACS patients and serves as a critical marker for organ function impairment [42]. Consistent with these findings, our study demonstrates that elevated white blood cell count is a risk factor for HF after PCI in ACS patients. Increased white blood cell levels are closely associated with left ventricular dysfunction and myocardial injury post-PCI [43, 44], both of which contribute to the development of HF. Moreover, white blood cell count has been implicated in reperfusion injury after PCI in ACS patients [44], further exacerbating adverse clinical outcomes, including arrhythmias and impaired cardiac function, which ultimately predispose patients to HF. In addition to white blood cell count, our study found that PTX3 is also a risk factor for HF after PCI. PTX3, a long pentraxin-type protein, is involved in multiple biological processes, including immune regulation, inflammatory response, and pathogen clearance. Latini et al. [45] demonstrated that elevated PTX3 levels in STEMI patients undergoing thrombolytic therapy are associated with an increased risk of HF and serve as an independent prognostic marker for threemonth mortality. PTX3 is

actively involved in the vascular repair process, and its elevated levels may reflect extensive vascular damage, which can compromise blood supply to the heart and increase the risk of HF [46].

## Effect of cardiac functional status on the development of HF after PCI in ACS patients

The results of this study indicate that decreased LVEF at admission, AF at admission, and increased heart rate are risk factors for HF development following PCI in ACS patients. Consistently, a multicenter prospective cohort study by Rosa et al. [47] showed that increased heart rate is associated with a higher one-year all-cause mortality risk in HF patients, high-



**Figure 23.** Egger regression test for the effect of age and diabetes mellitus on the development of HF after PCI in ACS patients (A: age, B: diabetes mellitus; HF: heart failure, PCI: percutaneous coronary intervention, ACS: acute coronary syndrome).

lighting its detrimental impact on cardiac prognosis. Additionally, heart rate has been identified as a predictive marker for adverse cardiovascular events post-PCI [48]. Increased heart rate may result from sympathetic nervous system activation, leading to circulatory system hyperactivity, which in turn promotes HF progression [49]. LVEF serves as a key indicator of ventricular remodeling, and its reduction can lead to increased left atrial pressure, impaired smooth muscle relaxation, and decreased stroke volume, thereby increasing susceptibility to HF after emergency PCI in ACS patients [50]. Similarly, atrial fibrillation, a prevalent arrhythmia, is characterized by rapid and irregular contractions, which disrupt normal hemodynamics

and predispose patients to thrombus formation. Additionally, AF may induce tachycardia-induced cardiomyopathy, resulting in structural and functional cardiac alterations and ultimately heightening the risk of HF [51]. Furthermore, decreased myocardial contractility is a critical contributor to HF development in ACS patients post-PCI, mainly due to its direct impact on cardiac pumping efficiency. Impaired contractility hampers effective blood circulation, leading to hemodynamic instability and subsequent HF onset. Additionally, decreased myocardial contractility can increase cardiac workload, exacerbating myocardial injury and remodeling, thereby creating a vicious cycle that further exacerbates HF risk [52].

# Effect of other factors on the development of HF after PCI in ACS patients

Anterior wall myocardial infarction has been identified as a risk factor for HF in ACS patients after PCI. This is primarily due to the extensive myocardial involvement, as the anterior wall constitutes a

substantial portion of the left ventricular myocardium. Consequently, anterior wall myocardial infarction can lead to severe myocardial damage, resulting in a marked reduction in the heart's pumping capacity and an increased risk of HF. Furthermore, myocardial infarction triggers a series of structural and functional adaptations known as ventricular remodeling. Compared with infarctions in other regions, anterior wall myocardial infarction is more likely to induce profound ventricular remodeling, characterized by chamber dilation and myocyte structural alterations, which further impact cardiac pumping efficiency and exacerbate HF risk [53]. Conversely, the results of this study indicate that complete revascularization serves as

#### Table 3. Evidence quality assessment

Certainty assessment							No. of patients		Effect		
No. of studies	Study design	Risk of bias	Inconsistency	Indirectness	Imprecision	Other considerations	HF occurred after PCI	HF did not occur after PCI	Relative (95% CI)	Certainty	Importance
Gender 3	Observational research	Not serious	Serious <sup>b</sup>	Not serious	Not serious	None	1997	21960	OR=1.43, 95% CI: (1.18-1.72)	⊕⊕⊕⊖ Moderate	CRITICAL
Previous 3	myocardial infarction Observational research	Not serious	Serious <sup>b</sup>	Not serious	Not serious	None	1487	17116	OR=1.58, 95% CI: (1.11-2.23)	⊕⊕⊕⊖ Moderate	CRITICAL
Atrial fibri 2	llation Observational research	Serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	None	1279	13797	HR=2.14 95% CI: (1.11-4.12)	⊕⊕⊖⊃ Low	CRITICAL
Complete 2	Revascularization Observational research	Serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	None	1279	13797	HR=0.29, 95% CI: (0.10-0.86)	⊕⊕⊖⊃ Low	CRITICAL

<sup>a</sup>Downgrading one level for unclear randomization methods and for deviations from the intended trial interventions. <sup>b</sup>Downgrading one level for heterogeneity ≥50%.

a protective factor against HF in ACS patients following PCI. The goal of complete revascularization is to reduce myocardial ischemia, improve cardiac function, and lower the likelihood of recurrent myocardial infarction, thereby potentially improving long-term prognosis for patients. This strategy is particularly beneficial for patients with multivessel disease (MVD), as they often present with multiple ischemic territories. Several studies have indicated that PCI with complete revascularization is the preferred treatment for STEMI patients with MVD [54]. However, despite its advantages, complete revascularization is associated with several potential risks and challenges, including prolonged procedural duration, increased contrast agent usage, heightened radiation exposure, and a greater risk of contrast-induced nephropathy. Additionally, possible complications such as stent thrombosis and the noreflow phenomenon remain concerns. Moreover, whether all patients derive significant benefits from complete revascularization remains a subject of ongoing debate, and its routine implementation in clinical practice remains suboptimal. Clinical decision-making regarding complete revascularization should therefore be individualized, taking into account a comprehensive assessment of patient-specific factors, including the risk-benefit ratio, lesion severity and complexity, availability of advanced diagnostic tools, risk of contrast-induced nephropathy, and the operator's technical proficiency in managing the lesions.

Given that the analysis of gender, previous myocardial infarction, AF, and complete revascularization was based on only 2-3 studies each and that the results exhibited substantial heterogeneity, the GRADE (Grades of Recommendation, Assessment, Development, and Evaluation) framework was used to assess the quality of the pooled evidence (Table 3). The assessment showed that the credibility of the findings regarding gender and previous myocardial infarction was moderate, indicating that while further research may influence the current conclusions, the existing evidence still holds a certain reference value. However, the credibility of the results regarding AF and complete revascularization was relatively low, indicating that subsequent studies are highly likely to significantly alter the current assessment outcomes. Therefore, caution should be exercised when interpreting and applying these findings in clinical practice.

This study has several limitations: (1) The impact factors analyzed in the included studies varied, and some could not be pooled for metaanalysis. Consequently, this study may not provide a comprehensive analysis of all relevant impact factors. (2) Despite employing a rigorous and systematic search strategy, the final number of included studies was relatively small, primarily due to differences in study design and sample sources. This limitation may have introduced selection bias, potentially affecting the robustness of the findings. (3) ACS encompasses unstable angina, NSTEMI, and STEMI, each with distinct pathophysiological characteristics. However, this study did not conduct subgroup analyses for these different ACS subtypes, which may limit the specificity of the findings. (4) Due to the small number of included studies, with fewer than 10 articles reporting each outcome measure, the publication bias analysis conducted in this study may have limited validity. Therefore, the results of this analysis should be interpreted cautiously and used as a reference rather than definitive evidence.

In summary, the occurrence of HF in ACS patients after PCI is influenced by a range of factors, including age, gender, hypertension, diabetes, history of myocardial infarction, anterior wall myocardial infarction, LVEF, white blood cell count, history of AF, complete revascularization, heart rate, PTX3, and myocardial contractility. The development of HF in these patients is associated with poor clinical outcomes. Therefore, healthcare professionals should implement comprehensive strategies to reduce the incidence of HF in ACS patients, improve patient prognosis, and alleviate the associated medical burden. Additionally, several other factors, such as lymphocyte count, eosinophil count, glomerular filtration rate, Killip classification, LVFS, platelet distribution width, chronic kidney disease, pulmonary disease, the GRACE risk score, peripheral artery disease, cardiogenic shock at admission, blood glucose level at admission, ejection time, D-dimer, systolic blood pressure, neutrophil count, high-sensitivity C-reactive protein (Hs-CRP), N-terminal pro B-type natriuretic peptide (NT-proBNP), number of diseased vessels, and peak troponin I level, have been explored in

individual studies regarding their potential relationship with HF. However, due to the limited number of studies, quantitative conclusions cannot be drawn from these factors. Further research is needed to better clarify their roles in the development of HF.

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#### Disclosure of conflict of interest

None.

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