

Relationship Between COVID-19 and Cardiovascular Disease in Critically Ill Patients: A Systematic Review of the Literature

Sergio Andrés Puerto Horta ^{1,*}, Laura Fernanda Monroy Tovar ², Maria Catalina Gaviria Pérez ², José Daniel Charry Cuellar ³

¹ Department of Internal Medicine, Hospital Británico de Buenos Aires, Buenos Aires, Argentina.

² Department of Epidemiology, Hernando Moncaleano University Hospital, Neiva, Colombia.

³ Center for Research and Innovation (CIINA), Universidad Navarra Foundation (UNINAVARRA), Neiva, Colombia.

* Correspondence: sergiopuerto903@hotmail.com.

Abstract: Cardiovascular complications are frequently reported in patients with severe COVID-19; however, their spectrum and clinical relevance in critically ill patients have not been fully characterized. To systematically review the literature to identify the main cardiovascular complications occurring in patients with severe COVID-19 admitted to intensive care units (ICUs). A systematic search of three electronic databases was conducted from December 1, 2019 to December 31, 2025. Observational studies including adult patients with severe COVID-19 admitted to ICUs and reporting cardiovascular complications were eligible, without language restrictions. Study selection and qualitative synthesis were performed according to predefined criteria. The primary outcomes were pre-existing cardiovascular comorbidities and cardiovascular complications occurring during ICU stay. Eight observational cohort studies met the inclusion criteria. A high prevalence of baseline cardiovascular comorbidities was consistently reported, particularly arterial hypertension, obesity, smoking history, and diabetes mellitus. The most frequently described cardiovascular complications in critically ill patients included circulatory failure, thromboembolic events, myocardial injury, cardiac arrhythmias, and cerebrovascular events. The reported frequency of these complications varied across studies. Cardiovascular complications are common among critically ill patients with COVID-19, with circulatory failure and thromboembolic and arrhythmic events being the most consistently reported. These findings highlight the need for early cardiovascular monitoring and multidisciplinary management in this population.

Keywords: COVID-19; SARS-CoV-2; Cardiovascular Diseases; Critical Care; Cohort Study.

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1. Introduction

Since its emergence in late 2019, SARS-CoV-2 infection has evolved into a global health crisis with sustained clinical and socioeconomic consequences. Although widespread vaccination and the emergence of viral variants have modified the epidemiological landscape and clinical severity over time, severe COVID-19 continues to require intensive care support in a subset of patients. Beyond its primary respiratory manifestations, COVID-19 is now recognized as a systemic disease with significant cardiovascular involvement. Reported complications include circulatory shock, myocardial injury, arrhythmias, and venous and arterial thromboembolic events. The pathophysiology of cardiovascular injury in severe COVID-19 is multifactorial, involving systemic hyperinflammation, endothelial dysfunction, dysregulated coagulation pathways, and microvascular injury, rather than a single receptor-mediated mechanism.

Critically ill patients admitted to intensive care units represent a particularly vulnerable population in whom cardiovascular complications are associated with hemodynamic instability, multiorgan dysfunction, and increased mortality. However, the incidence, spectrum, and clinical implications of these complications remain variably reported across studies, partly due to heterogeneity in study design, patient populations, and evolving pandemic phases. In this context, a systematic synthesis of the available evidence focused specifically on ICU patients is warranted to better characterize the cardiovascular manifestations of severe COVID-19 and to clarify their association with adverse outcomes.

2. Methodology

This systematic review was conducted in accordance with the recommendations of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses, which provide a structured framework for transparent reporting of evidence-based research. A comprehensive literature search was performed in the Medline, ScienceDirect, and LILACS databases to identify relevant studies meeting the predefined eligibility criteria. The search covered publications from December 1, 2019, to December 31, 2025. Four trained authors independently conducted the search and study selection process.

2.1 Inclusion and exclusion criteria

All descriptive studies, including prospective or retrospective cohort studies, were included if they enrolled patients aged 18 years or older who were admitted to the intensive care unit with a diagnosis of SARS-CoV-2 infection confirmed by molecular testing (PCR) or a positive antigen test, and who developed cardiovascular complications during their ICU stay. Articles consisting of case reports or letters to the editor were excluded. Studies that did not report cardiovascular complications or mortality as outcomes, or that contained incomplete or imprecise data, were also excluded.

The search strategy was developed based on the PICO methodology, addressing the research question: "Cardiovascular complications in patients with COVID-19 admitted to the intensive care unit." Keywords included "COVID-19," "SARS-CoV-2," "cardiovascular diseases," "critical care," and "cohort study." The search was conducted using a combination of standardized vocabulary (EMTREE or MeSH terms) without language restrictions.

2.2 Risk of bias assessment

The methodological quality of the review process was evaluated using the AMSTAR-2 tool. The review met key methodological standards, including a comprehensive literature search across multiple databases without language restrictions, independent study selection and data extraction by four trained reviewers, and transparent reporting of eligibility criteria and study selection.

Given the observational design of the included studies and the descriptive objectives of the review, a separate formal risk-of-bias assessment at the individual study level was not performed. Methodological aspects relevant to observational research were considered during study selection and were qualitatively integrated into the interpretation of findings. Methodological quality was considered during the study selection process. Consequently, potential sources of bias inherent to observational designs were not formally quantified but were acknowledged when interpreting the findings. No formal assessment of publication bias was performed, which was considered appropriate given the limited number of included studies.

3. Results

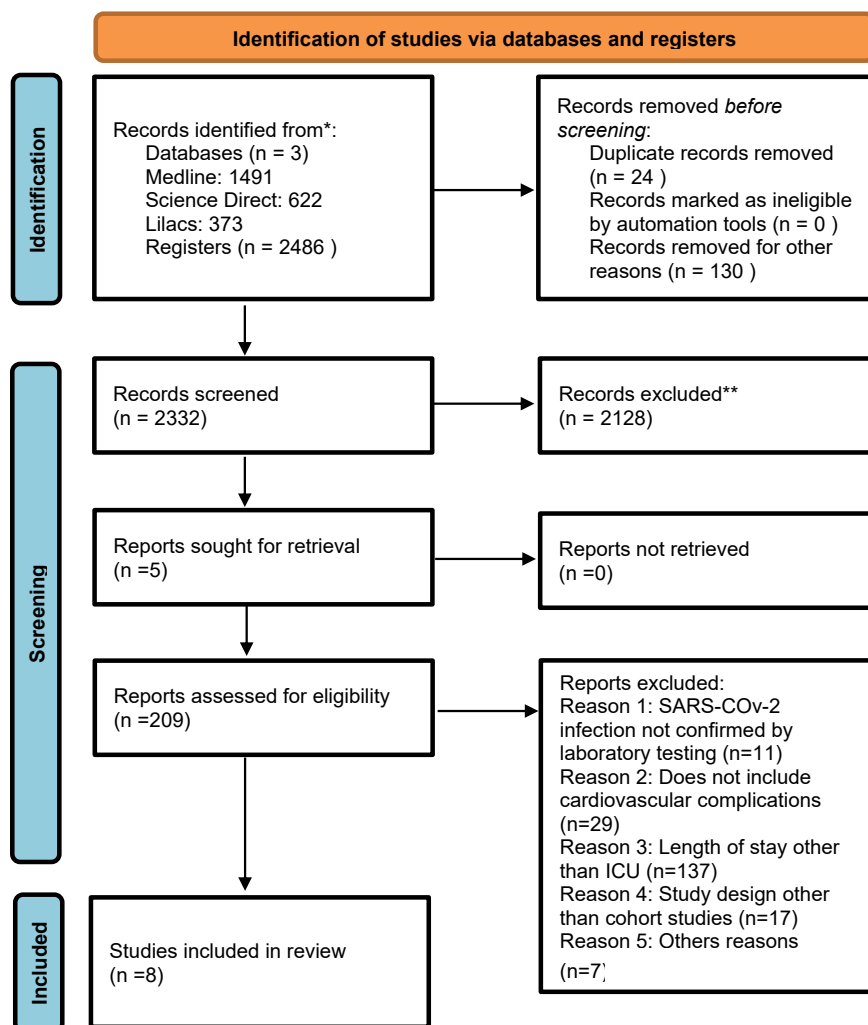
A total of 2,486 publications were initially identified through the search strategy across the selected databases. During preliminary screening, 154 records were excluded,

including 24 duplicates and 130 records for which full-text access was not immediately available. Of these 130 records, responses were obtained for 62 after contacting authors and attempting institutional and inter-library access. Following full-text review, 53 were excluded for not involving ICU populations and 9 for not meeting the predefined cohort study design criteria. For the remaining records, full texts could not be obtained despite repeated attempts.

The remaining 2332 records underwent title screening, resulting in the exclusion of 2128 articles. Subsequently, 5 records were retrieved and, following consensus among the 3 reviewers, were deemed eligible for further evaluation. Ultimately, 209 articles were assessed for eligibility according to the predefined inclusion criteria, methodological rigor, and overall study quality. Of these, 201 were excluded: 137 studies involved patients who were not admitted to an intensive care unit; 29 did not report cardiovascular complications; 17 employed study designs other than cohort studies; 11 included patients without laboratory-confirmed SARS-CoV-2 infection; and 7 were interventional studies or were deemed to have low methodological quality.

Following the selection process, eight studies fulfilled all eligibility criteria and were included in the final review: four identified through MEDLINE, none from ScienceDirect, and four from LILACS (Figure 1). Twenty-two percent of the included studies were conducted in China, while the remaining studies originated from Denmark (12.5%), London (12.5%), the United States (12.5%), Spain (12.5%), Brazil (12.5%), and Mexico (12.5%). Most of the studies were retrospective in design (n = 6; 75%), whereas two studies (25%) were prospective.

Figure 1. Information Search Process.



4. Review

4.1 Clinical Data and Comorbidities

The systematic review was based on a pooled sample of 6,412 patients admitted to the intensive care unit (ICU) with COVID-19, for whom data on comorbidities, cardiovascular complications, and mortality were reported. Table 1 presents the characteristics of the included studies. Sample sizes varied considerably (67–5,019 patients). The mean age across studies was approximately 60 years. Most cohorts were predominantly male, although the proportion of male participants varied across studies.

Regarding comorbidities, hypertension was the most frequently reported condition (7 studies; 3,631 patients across studies), followed by obesity (3 studies; 2,813 patients), history of smoking (3 studies; 2,224 patients), and diabetes mellitus (7 studies; 2,387 patients). Other comorbidities were reported across studies (Table 2).

Table 1. Main Features.

Reference	Country	Study Design	Total Patients	Main Outcome	Quality Assessment
Vijayabharathy et al. [6]	Denmark	Multicenter retrospective cohort	155	Patients with cardiac arrhythmias had higher mortality compared with those without arrhythmias (63% vs. 39%), corresponding to a relative risk (RR) of 1.63 (95% CI: 1.19–2.24; $p = 0.005$).	Good
Gao et al. [5]	London	Multicenter retrospective cohort	109	Patients who developed new-onset atrial fibrillation (NOAF) during ICU stay were older (median 65 years [IQR 59–71] vs. 58 years [IQR 51–64]; $p = 0.001$) and more likely to have underlying heart failure (33% vs. 2%; $p = 0.03$) and chronic kidney disease (44% vs. 16%). NOAF was associated with increased in-hospital mortality (OR: 5.4; 95% CI: 1.7–17; $p = 0.004$).	Good
Ferrando et al. [9]	Wuhan, China	Single-center retrospective cohort	79	The incidence of tachyarrhythmias was significantly higher among non-survivors compared with survivors ($p = 0.04$). In Cox regression analysis, older patients with ventricular tachycardia had an increased risk of death (HR: 3.30; 95% CI: 1.52–7.15; $p = 0.002$; and HR: 1.05; 95% CI: 1.02–1.07; $p < 0.001$). Overall mortality was 72% (81% in men and 54% in women; $p = 0.01$).	Moderate
Hernández-Cárdenas et al. [3]	Andorra	Multicenter prospec-	663	Cardiovascular complications were	Good

		tive cohort		more frequent among non-survivors, including shock (42% vs. 14%; $p < 10^{-13}$) and arrhythmias (24% vs. 11%; $p < 10^{-4}$). Non-survivors required greater use of vasoactive drugs. ICU mortality was 31% (203 patients).	
Wang et al. [12]	México	Single-center prospective cohort	67	Vasopressor support was required in 59% of patients and was more common among non-survivors than survivors (76% vs. 45%; $p < 0.05$). Overall mortality was 44.7%. Male sex was more prevalent among non-survivors (80% vs. 54%; $p < 0.05$).	Good
Soares Brandão et al. [8]	Wuhan, China	Single-center retrospective cohort	77	Patients with myocardial injury were older (68.4 ± 10.1 vs. 62.1 ± 13.5 years; $p = 0.022$), had more cardiovascular comorbidities (34.1% vs. 11.1%; $p = 0.017$), and were more frequently smokers (53.6% vs. 22.2%; $p < 0.01$). Cardiovascular complications and mortality rates were significantly higher in this group.	Good
Hayek et al. [4]	Brazil	Single-center retrospective cohort	243	Risk factors for thromboembolic events included admission D-dimer $>3,000$ ng/mL ($p < 0.001$) and major bleeding. These factors were also identified by logistic regression for venous thromboembolism (DVT and PE).	Moderate
Vijayabharathy et al. [6]	United States	Multicenter retrospective cohort	5019	Patients with cardiac arrest more frequently had hypertension (67.8% vs. 60.5%; $p < 0.001$) and a history of smoking (55.5% vs. 49.5%; $p < 0.01$). They required ≥ 2 vasopressors more often and had significantly higher mortality (93.2% vs. 32.2%; $p < 0.001$).	Moderate

NOAF. New-onset atrial fibrillation. HF. Heart failure. CKD. Chronic kidney disease. CV. cardiovascular complications. VT. ventricular tachycardia. DVT. Deep vein thrombosis. PE. Pulmonary embolism.

4.2 Cardiovascular Complications

Regarding cardiovascular complications, shock was the most frequently reported event (six studies), followed by deep vein thrombosis (three studies), cardiac arrhythmias

(six studies), pulmonary embolism (three studies), acute myocardial infarction (three studies), and cerebrovascular events (three studies). Notably, most studies reported shock as a composite clinical outcome without detailed hemodynamic characterization, and did not consistently differentiate between septic, cardiogenic, or obstructive etiologies. Consequently, the specific pathophysiological mechanisms underlying hemodynamic deterioration could not be systematically delineated. The reported incidence of these complications varied substantially across studies. Mortality was also frequently reported, with 2,580 deaths documented across the included cohorts; however, no pooled incidence estimates were calculated due to heterogeneity in study design and patient populations (Table 3).

Table 2. Prevalence of Comorbidities Among Patients Reported in Included Studies.

References	HTA	DM	HF	OB	CVD	SH	CVD	IHD	TA	BA	DL	COPD	Asthma	CKD	CA	TE
Wetterslev et al. [7]	68	32	-	-	-	20	-	15	22	2	14	-	6	13	12	-
Vijayabharathy et al. [6]	61	46	4	25	-	-	-	17	-	-	40	-	-	22	1	-
Gao et al. [5]	40	17	2	-	10	-	-	10	-	-	-	3	-	-	2	-
Ferrando et al. [9]	329	152	9	-	-	-	-	-	-	90	28	17	38	-	72	-
Hernández-Cárdenas et al. [3]	8	14	-	33	-	-	-	-	-	-	-	-	-	-	-	-
Wang et al. [12]	39	17	-	-	-	30	18	2	2	-	-	3	-	4	2	-
Soares Brandão et al. [8]	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	32
Hayek et al. [4]	3086	2110	512	2813	-	2174	-	676	-	-	433	-	819	227	-	-

HTA. Hypertension; HF. Heart Failure; OB. Obesity; IHD. Ischemic Heart Disease; CVD. Cardiovascular Disease; DM. Diabetes Mellitus; TA. Tachyarrhythmias; BA. Bradyarrhythmias; DL. Dyslipidemia; SH. Smoking History; COPD. Chronic Obstructive Pulmonary Disease; CKD. Chronic Kidney Disease; CA. Malignancies; TE. Thromboembolic Disease.

Table 3. Cardiovascular and Thromboembolic Complications Reported in Included Studies.

Reference	PE	DVT	AMI	ACV	Arrhythmia	HF	Shock	Cardiac Arrest	Mortality
Wetterslev et al. [7]	-	12	15	-	57	-	125	-	74
Vijayabharathy et al. [6]	3	21	2	-	2	-	-	-	38
Gao et al. [5]	-	-	-	3	19	-	24	-	57
Ferrando et al. [9]	72	-	-	12	97	22	150	26	203
Hernández-Cárdenas et al. [3]	-	-	-	-	-	-	40	-	30
Wang et al. [12]	-	-	-	-	19	-	3	-	58
Soares Brandão et al. [8]	19	9	6	3	-	-	-	-	77
Hayek et al. [4]	-	-	-	-	48	-	1617	701	2043

PE. Pulmonary Embolism; DVT. Deep Vein Thrombosis; AMI. Acute Myocardial Infarction; ACV. Cerebrovascular Accident; Arrhythmia. Cardiac Arrhythmia; HF. Heart Failure; Shock. Circulatory Shock; Cardiac Arrest. Cardiac Arrest; Mortality. In-Hospital Mortality. “-” Indicates Data Not Reported.

4.3 Circulatory Failure

Across six studies reporting shock among ICU patients with COVID-19, the reported incidence ranged from 0% to 59.7%. In the cohort described by Ferrando et al., which included 663 patients, circulatory failure was significantly more frequent among non-survivors compared with survivors (42% vs. 14%; $p < 10^{-13}$) and was independently associated with increased mortality (odds ratio [OR]: 2.15; 95% CI: 1.35–3.43; $p = 0.0013$) [9]. Similarly, Hernández-Cárdenas et al. reported that 59% of patients required vasopressor support with norepinephrine. A significantly higher proportion of non-survivors

required norepinephrine compared with survivors (76% vs. 45%; $p < 0.05$) [3]. The overall mortality rate in this cohort was 44.7%. Additionally, 65% of participants were male, with a significantly higher proportion of men among non-survivors compared with survivors (80% vs. 54%; $p < 0.05$) [3].

4.4 Cardiac Arrest

Two studies reported the occurrence of cardiac arrest among ICU patients with COVID-19, with incidence rates ranging from 0% to 13.96%. In the cohort described by Salim S. Hayek et al., patients who experienced cardiac arrest more frequently had chronic arterial hypertension (67.8% vs. 60.5%; $p < 0.001$) and were more commonly smokers (55.5% vs. 49.5%; $p < 0.01$) [4]. Additionally, patients with cardiac arrest were more likely to require at least two vasopressors compared with those without cardiac arrest (51% vs. 29%; $p < 0.001$) and had significantly higher in-hospital mortality rates (93.2% vs. 32.2%; $p < 0.001$) [4].

4.5 Cardiac Arrhythmias

A total of six studies reported cardiac arrhythmias in ICU patients with COVID-19, the reported incidence ranged from 0% to 36.7%. In the study by Xue Lin et al., tachyarrhythmias were significantly more frequent among non-survivors compared with survivors ($p = 0.04$) [5]. In a Cox regression model, ventricular tachyarrhythmias and advanced age were independently associated with increased mortality, with hazard ratios (HRs) of 3.30 (95% CI: 1.52–7.15; $p = 0.002$) and 1.05 (95% CI: 1.02–1.07; $p < 0.001$), respectively. Overall mortality in this cohort was 72%, with higher mortality among men than women (81% vs. 54%; $p = 0.01$) [5].

Vijayabharathy et al. [6] reported new-onset atrial fibrillation (NOAF) in 16 patients (14.6%) during ICU stay, and cardiac troponin T levels were elevated above the 99th percentile upper reference limit in 91% of patients. Individuals who developed NOAF were significantly older (median 65 years [IQR 59–71] vs. 58 years [IQR 51–64]; $p = 0.001$) and more likely to have pre-existing chronic heart failure (33% vs. 2%; $p = 0.03$) and chronic kidney disease (44% vs. 16%) [6]. ICU length of stay was longer among survivors with NOAF compared with those who remained in sinus rhythm (42 days [IQR 37–44] vs. 32 days [IQR 21–40]; $p = 0.03$). The presence of NOAF was independently associated with increased in-hospital mortality (OR: 5.4; 95% CI: 1.7–17; $p = 0.004$) [6].

In the study by Wetterslev et al., the incidence of arrhythmias in the ICU was 37% (57/155; 95% CI: 30–45), of which 68% were new-onset events [7]. Supraventricular tachycardias, particularly atrial fibrillation/flutter, were the most common arrhythmias (95%), whereas ventricular arrhythmias (3%) and conduction blocks (2%) were uncommon. Sixty-day mortality was 48%, and patients with arrhythmias had significantly higher mortality compared with those without arrhythmias (63% vs. 39%; RR: 1.63; 95% CI: 1.19–2.24; $p = 0.005$) [7].

4.6 Myocardial Injury

Three studies reported the occurrence of myocardial injury, with incidence rates ranging from 0% to 9.67%. In the study by Hao Qian et al., patients with myocardial injury had significantly higher all-cause mortality and cardiovascular-related mortality compared with those without myocardial injury (85.3% vs. 63.9%; $p = 0.029$ and 14.6% vs. 0%; $p = 0.027$, respectively) [5]. Myocardial injury at admission was independently associated with increased 28-day mortality (HR: 2.20; 95% CI: 1.29–3.74; $p = 0.004$) [5]. Patients with myocardial injury were older (68.4 ± 10.1 vs. 62.1 ± 13.5 years; $p = 0.022$), had a higher prevalence of cardiovascular comorbidities (34.1% vs. 11.1%; $p = 0.017$), including coronary artery disease (19.6% vs. 2.8%; $p = 0.032$), and were more frequently smokers (53.6% vs. 22.2%; $p < 0.01$). Cardiovascular complications were significantly more frequent in the myocardial injury group (41.5% vs. 13.9%; $p < 0.01$) [5].

4.7 Thromboembolic Events

Four studies reported thromboembolic complications, with incidence rates ranging from 0% to 19.2%. In a Brazilian cohort of 243 patients reported by Soares Brandão et al., thromboembolic events occurred in 14.8% of patients. Venous thromboembolic events included deep vein thrombosis (3.7%) and pulmonary embolism (7.8%), whereas arterial events comprised stroke (1.2%), myocardial infarction (2.5%), and peripheral arterial occlusion (1.2%) [8].

Patients with D-dimer levels >3,000 ng/mL at admission had a significantly higher incidence of thromboembolic events compared with those with lower levels (40.6% vs. 14.3%), corresponding to an OR of 4.11 (95% CI: 1.82–9.24; $p < 0.001$) [8]. Overall mortality in this cohort was 33.5%. Furthermore, a D-dimer cutoff value >1,140.5 ng/mL, identified through ROC curve analysis, was associated with shorter overall survival and a 4.09-fold increased risk of death (HR: 4.09; 95% CI: 2.47–6.79) [8].

4.8 Cerebrovascular Events

Three studies reported cerebrovascular events among ICU patients with COVID-19, with incidence rates ranging from 0% to 10.8%. In the cohort by Wei Wu and Shuyang Zhang conducted in Wuhan, overall mortality was 72% (81% in men and 54% in women; $p = 0.01$), with cerebrovascular disease accounting for 3.5% of all causes of death [5]. In a separate Spanish cohort, the presence of cerebrovascular disease was associated with an increased probability of death (OR: 3.52; 95% CI: 0.96–12.86; $p = 0.057$) [9].

4.9 Mortality

Eight studies reported mortality among ICU patients with COVID-19, with mortality rates ranging from 0% to 75.3%. In the study by C. Ferrando et al., ICU mortality was 31% (203/663 patients). Cardiovascular complications were significantly more frequent among non-survivors, particularly shock (42% vs. 14%; $p < 10^{-13}$) and arrhythmia (24% vs. 11%; $p < 10^{-4}$) [9]. Inflammatory markers at ICU admission were also higher among non-survivors, including high-sensitivity cardiac troponin I (median 16 [IQR 5–53] vs. 11 [IQR 4–22]; $p = 0.006$) [9].

Cardiovascular comorbidities were more prevalent among non-survivors, including arterial hypertension (56.7% vs. 46.5%; $p = 0.018$), chronic heart failure (3.0% vs. 0.7%; $p = 0.027$), diabetes mellitus (30.1% vs. 19.6%; $p = 0.004$), and dyslipidemia (17.2% vs. 12.0%; $p = 0.084$) [9]. In the cohort reported by Hernández-Cárdenas et al., overall mortality was 44%. Sixty-five percent of participants were male, and the proportion of men was significantly higher among patients who died compared with survivors (80% vs. 54%; $p < 0.05$) [3]. Cardiovascular comorbidities were also more frequently observed among patients with fatal outcomes, including obesity (56.6% vs. 43.2%) and arterial hypertension (20% vs. 5.4%) [3].

5. Discussion

Patients who developed cardiovascular complications demonstrated a substantially higher burden of baseline cardiometabolic comorbidities, including arterial hypertension, obesity, active or prior smoking, diabetes mellitus, and chronic kidney disease, and experienced a more complicated in-hospital trajectory [9]. These findings support the hypothesis that pre-existing cardiovascular vulnerability amplifies the systemic inflammatory and prothrombotic milieu characteristic of severe SARS-CoV-2 infection.

In this systematic review restricted to critically ill patients, cardiovascular complications were frequent and encompassed a heterogeneous spectrum of clinical entities. Circulatory shock was among the most prevalent complications and was consistently associated with excess mortality. Likewise, the development of cardiac arrhythmias, myocardial injury, thromboembolic events, and cerebrovascular complications correlated

with worse clinical outcomes and a higher comorbidity burden, suggesting that cardiovascular involvement represents both a marker and mediator of disease severity.

Myocardial injury, as evidenced by elevated cardiac troponin concentrations, has been recognized as a central manifestation of COVID-19 related cardiac involvement [10]. The underlying mechanisms are likely multifactorial, including cytokine-mediated myocardial depression, endothelial dysfunction, microvascular thrombosis, supply–demand mismatch, and potential direct viral myocardial invasion [10]. In accordance with prior reports, patients with myocardial injury in the included studies exhibited significantly higher mortality and a more severe clinical course, underscoring its prognostic significance in the ICU setting [11]. The incidence of myocardial injury observed in the included studies appears lower than that reported in several large meta-analyses of hospitalized or critically ill patients, where rates frequently exceed 20%. This discrepancy may be attributable to differences in inclusion criteria, as the present review was restricted to ICU-only adult cohorts, as well as heterogeneity in troponin measurement protocols, timing of biomarker assessment, and diagnostic thresholds across studies. In several cohorts, systematic serial troponin monitoring was not reported, potentially leading to underestimation of myocardial injury incidence.

Although cardiovascular injury has been widely reported in COVID-19, it is important to contextualize these findings within the broader framework of critical illness. Myocardial dysfunction, arrhythmias, and stress-induced cardiomyopathy are well-recognized complications in any severe viral infection or septic ICU population. Therefore, the cardiovascular manifestations observed in COVID-19 may not be entirely disease-specific but rather represent an amplification of established critical care pathophysiological pathways, including systemic inflammation, catecholamine excess, and microvascular dysfunction. However, the pronounced prothrombotic phenotype and endothelial injury described in SARS-CoV-2 infection may confer a distinctive vascular component that differentiates it from classical viral myocarditis or stress cardiomyopathy.

Across most studies, non-survivors were older and predominantly male. This demographic distribution aligns with previously reported data and may reflect sex-specific differences in immune regulation, hormonal modulation, and cardiovascular risk profiles, which together may contribute to adverse outcomes. Collectively, these observations indicate that cardiovascular complications in critically ill patients with COVID-19 should be interpreted within the broader context of systemic disease severity. Early identification of high-risk phenotypes and proactive cardiovascular surveillance may therefore be essential components of comprehensive critical care management in this population.

This systematic review delineates the substantial impact and clinical relevance of cardiovascular complications among critically ill patients with COVID-19 requiring intensive care support. Circulatory shock was the most frequently reported complication, followed by thromboembolic phenomena and cardiac arrhythmias. These events were consistently associated with hemodynamic instability, escalation of vasopressor therapy, prolonged invasive mechanical ventilation, and increased ICU length of stay, collectively contributing to excess short-term mortality.

The present findings are concordant with prior large-scale analyses. Kunutsor et al. demonstrated a high prevalence of cardiovascular complications in severe COVID-19 and identified shock and myocardial injury as principal determinants of adverse outcomes [10]. Similarly, the multicenter cohort reported by Lala et al. showed that myocardial injury at hospital admission independently conferred an approximate twofold increase in mortality risk [11]. Together, these data support the notion that cardiovascular involvement represents a central pathophysiological substrate of critical SARS-CoV-2 infection rather than a secondary epiphenomenon.

The underlying mechanisms appear to be multifactorial and interrelated, encompassing systemic hyperinflammation, cytokine-mediated myocardial depression, endo-

thelial dysfunction, microvascular injury, dysregulated coagulation cascades, and macro- and microthrombotic phenomena. This integrated pathobiological model provides a plausible explanation for the simultaneous occurrence of venous and arterial thromboembolic events, myocardial injury, arrhythmogenesis, and hemodynamic collapse observed in critical care populations [10,11].

New-onset arrhythmias, particularly atrial fibrillation, emerged as clinically significant events associated with markedly elevated mortality in several cohorts, exceeding 70% in selected reports from China [12]. In this context, arrhythmogenesis likely reflects a convergence of systemic inflammation, hypoxemia, autonomic imbalance, catecholamine excess, metabolic derangements, and pre-existing structural cardiac vulnerability. A high prevalence of cardiometabolic comorbidities, including arterial hypertension, diabetes mellitus, obesity, and chronic kidney disease, was consistently observed among patients with adverse outcomes. These conditions may potentiate endothelial injury, impair adaptive immune responses, and amplify the proinflammatory and prothrombotic milieu induced by SARS-CoV-2 infection, thereby lowering the threshold for cardiovascular decompensation.

Collectively, these observations underscore that cardiovascular complications in critically ill COVID-19 patients should be interpreted within the framework of systemic disease severity and host susceptibility. Early identification of high-risk phenotypes, rigorous hemodynamic and biomarker surveillance, and targeted cardiovascular supportive strategies may therefore represent essential components of comprehensive critical care management.

Despite substantial advances in delineating the clinical spectrum of COVID-19, significant mechanistic uncertainties persist. Further translational and longitudinal investigations are warranted to clarify the interaction between pre-existing cardiometabolic vulnerability and acute viral injury, with the ultimate aim of refining prognostic stratification and informing precision-based therapeutic interventions.

6. Conclusion

This systematic review underscores the considerable magnitude and prognostic significance of cardiovascular complications among critically ill patients with COVID-19 requiring intensive care. Circulatory shock, cardiac arrhythmias, and thromboembolic events emerged as the most frequently reported manifestations and were consistently associated with hemodynamic instability, escalation of organ support, and increased mortality. Rather than generalized surveillance, our findings suggest that cardiovascular monitoring strategies should be intensified in patients with a high cardiometabolic burden, particularly those with arterial hypertension and obesity, who demonstrated a disproportionately higher incidence of adverse outcomes. In such patients, early serial assessment of cardiac biomarkers, strict hemodynamic monitoring, and proactive screening for thromboembolic complications may represent pragmatic surveillance triggers in the ICU setting.

These findings emphasize the need for structured cardiovascular risk stratification and proactive surveillance strategies in ICU patients with COVID-19, particularly those with a high comorbidity burden. Early detection and timely management of cardiovascular complications may represent critical determinants of improved outcomes in this high-risk cohort.

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