



Perioperative management of vasoplegic syndrome in cardiac surgery: a narrative review

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Abstract

Vasoplegic syndrome (VS) is a frequent and potentially life-threatening complication following cardiac surgery with cardiopulmonary bypass (CPB) and is associated with significant morbidity and mortality. It is characterized by persistent hypotension, low systemic vascular resistance, and preserved or elevated cardiac output despite adequate volume resuscitation. This narrative review provides a comprehensive and clinically focused synthesis of current evidence on the pathophysiology, risk factors, diagnosis, and perioperative management of VS. A structured literature search of the PubMed database was conducted, focusing on studies published from 2020 to 2026 and complemented by key landmark trials.

The pathophysiology of VS is multifactorial, involving systemic inflammatory activation, excessive nitric oxide production, vasopressin depletion, dysregulation of the renin–angiotensin–aldosterone system, and impaired vascular responsiveness to catecholamines. Major risk factors include preoperative use of ACE inhibitors or angiotensin receptor blockers, impaired cardiac function, prolonged CPB duration, and complex surgical procedures. Early recognition and differentiation from other causes of perioperative shock are essential for timely management.

Current therapeutic strategies emphasize a multimodal, physiology-guided approach combining catecholamines with non-adrenergic agents such as vasopressin, methylene blue, hydroxocobalamin, and angiotensin II, particularly in refractory cases. Emerging evidence supports early combination vasopressor strategies to improve hemodynamic stability and reduce catecholamine exposure. However, optimal treatment sequencing and patient selection remain uncertain.

A structured, algorithm-based approach integrating early diagnosis, targeted therapies, and advanced hemodynamic monitoring may improve clinical outcomes. Further research is needed to refine diagnostic criteria, identify reliable biomarkers, and establish evidence-based management algorithms for VS in cardiac surgery.

Keywords: angiotensin II, cardiac surgery, cardiopulmonary bypass, methylene blue, vasodilatory shock, vasoplegic syndrome

Introduction

Vasoplegic syndrome (VS) is a well-recognized complication after cardiac surgery, particularly in procedures involving cardiopulmonary bypass (CPB)^[1]. It is characterized by profound and persistent arterial hypotension associated with markedly reduced systemic vascular resistance despite a normal or increased cardiac

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Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Annals of Medicine & Surgery (2026) 88:6683–6688

Received 7 May 2026; Accepted 4 August 2026

Published online 16 September 2026

<http://dx.doi.org/10.1097/MS9.0000000000005611>

HIGHLIGHTS

- Vasoplegic syndrome is a major cause of refractory hypotension after cardiac surgery with cardiopulmonary bypass.
- Excess nitric oxide production, vasopressin depletion, and RAAS dysregulation are key mechanisms underlying vasoplegia.
- Early multimodal vasopressor therapy may improve hemodynamic stability and reduce catecholamine exposure.
- Vasopressin, methylene blue, hydroxocobalamin, and angiotensin II represent important adjunctive therapies for refractory vasoplegia.
- A physiology-guided and algorithm-based perioperative approach may improve outcomes in cardiac surgical patients with vasoplegic syndrome.

output. Clinically, VS is commonly defined by hypotension (mean arterial pressure <65 mmHg), low systemic vascular resistance, and preserved or elevated cardiac output despite adequate fluid resuscitation and high-dose vasopressor therapy. This distinctive hemodynamic profile differentiates vasoplegia from other forms of perioperative shock, such as cardiogenic or hypovolemic shock, and often necessitates escalating doses of vasopressors to maintain adequate perfusion pressure.

The incidence of VS varies depending on the definition used and the patient population, but it has been reported in up to 25% of patients undergoing cardiac surgery with CPB^[1]. Severe forms are associated with significant morbidity, including acute kidney injury, prolonged mechanical ventilation, extended intensive care unit (ICU) stay, and increased health-care costs. Moreover, refractory vasoplegia has been linked to higher perioperative mortality rates, underscoring its clinical significance^[2].

The pathophysiology of VS is complex and multifactorial, involving an interplay among systemic inflammatory response, endothelial dysfunction, dysregulation of vasoregulatory pathways, and impaired vascular responsiveness to catecholamines. CPB plays a central role in triggering this cascade through blood contact with artificial surfaces, ischemia–reperfusion injury, and activation of inflammatory mediators^[1], ultimately leading to excessive vasodilation and circulatory collapse^[3].

From a clinical perspective, early recognition of vasoplegia is essential but remains challenging in the perioperative setting, where multiple factors may contribute to hypotension. A structured diagnostic approach is therefore required to differentiate VS from other causes of hemodynamic instability. Prompt and appropriate management is critical, as delayed treatment may lead to progressive organ dysfunction and worse outcomes^[2].

Advances in our understanding of vasoplegia have led to the development of targeted therapeutic strategies beyond traditional catecholamines. These include vasopressin, methylene blue, hydroxocobalamin, and angiotensin II, which address distinct pathophysiological pathways involved in vasodilation. Consequently, a multimodal, physiology-guided approach is increasingly advocated.

This narrative review aims to provide a comprehensive overview of the perioperative management of VS in cardiac surgery, focusing on current evidence, emerging therapies, and practical clinical strategies to optimize patient outcomes.

Methods

A narrative literature review was conducted to synthesize current evidence regarding the perioperative management of VS in cardiac surgery. A comprehensive search of the PubMed database was performed using a combination of Medical Subject Headings (MeSH) and free-text keywords, including “vasoplegic syndrome,” “vasoplegia,” “cardiac surgery,” “cardiopulmonary bypass,” “methylene blue,” “vasopressin,” “hydroxocobalamin,” and “angiotensin II.” Boolean operators (“AND” and “OR”) were used to refine the search strategy and improve relevance.

The search focused primarily on articles published between 2020 and 2026 to ensure the inclusion of the most recent advances in pathophysiology and management. However, seminal and landmark studies were also included when they were considered essential for understanding key mechanisms and therapeutic approaches. Both clinical and experimental studies were reviewed, including randomized controlled trials, observational studies, systematic reviews, and high-quality narrative reviews.

Articles were selected based on their relevance to the perioperative setting in adult cardiac surgery, with particular emphasis on studies addressing mechanisms of vasoplegia, diagnostic

criteria, and therapeutic strategies. Studies focusing on non-cardiac surgery or septic vasoplegia without clear applicability to the cardiac surgical population were considered less relevant and selectively excluded.

Although this is a narrative review, the literature search and selection were conducted in a structured manner to ensure comprehensiveness and to minimize selection bias. The review was conducted in accordance with the SANRA (Scale for the Assessment of Narrative Review Articles) recommendations to enhance methodological transparency and reporting quality.

Given the narrative nature of this review, no formal systematic review protocol or meta-analytic methods were applied. Instead, the objective was to provide a clinically oriented synthesis of the available literature, integrating pathophysiological insights with practical management strategies. The references cited throughout the manuscript^[1–14] were selected to reflect key and representative contributions to the field.

Pathophysiology

VS is a complex and multifactorial condition resulting from the interplay of inflammatory, endothelial, and neurohormonal mechanisms that ultimately lead to profound vasodilation and vascular hyporesponsiveness. The pathophysiology is closely linked to the use of CPB, which acts as a major trigger for systemic inflammatory activation^[2,3].

One of the primary mechanisms involved is the systemic inflammatory response induced by CPB. Exposure of blood to non-endothelial surfaces, ischemia–reperfusion injury, and surgical trauma lead to activation of leukocytes, complement pathways, and the release of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor- α ^[1]. This inflammatory cascade promotes endothelial dysfunction and contributes to the loss of vascular tone.

A key downstream effect of this inflammatory activation is the overproduction of nitric oxide (NO). Inducible NO synthase (iNOS) is upregulated in response to inflammatory mediators, leading to excessive NO generation^[6]. NO activates soluble guanylate cyclase, increasing cyclic guanosine monophosphate (cGMP) levels in vascular smooth muscle cells, resulting in vasodilation and reduced systemic vascular resistance^[7].

In parallel, vasopressin depletion plays a critical role in the development of vasoplegia. Vasopressin is an important endogenous vasoconstrictor that acts via V1 receptors on vascular smooth muscle. During prolonged shock states and after CPB, endogenous vasopressin stores become depleted and vascular responsiveness to vasopressin may also be impaired, further contributing to refractory hypotension^[7].

Another important mechanism involves dysregulation of the renin–angiotensin–aldosterone system (RAAS). Evidence suggests that patients with vasoplegia may have a relative angiotensin II deficiency, which impairs vasoconstrictive responses and contributes to persistent vasodilation^[8]. This alteration may be particularly relevant in patients who were previously treated with ACE inhibitors or angiotensin receptor blockers (ARBs).

Finally, adrenergic receptor downregulation and signaling impairment reduce the effectiveness of catecholamines. Prolonged exposure to high levels of endogenous and exogenous catecholamines leads to desensitization of α -adrenergic receptors and alterations in intracellular signaling

pathways, including G-protein uncoupling. As a result, vascular smooth muscle responsiveness to vasopressors is diminished, necessitating escalating doses to maintain adequate perfusion pressure.

Overall, these mechanisms act synergistically, leading to a state of profound vasodilation, decreased systemic vascular resistance, and hemodynamic instability characteristic of VS (Fig. 1).

Risk factors

Several perioperative factors have been identified that contribute to the development of VS following cardiac surgery. These risk factors involve patient characteristics, preoperative treatments, and intraoperative conditions, and they collectively influence susceptibility to profound vasodilation after CPB.

Among the most consistently reported risk factors is the preoperative use of angiotensin-converting enzyme inhibitors (ACE inhibitors) and ARBs. These medications interfere with the renin-angiotensin-aldosterone system and may blunt the vasoconstrictive response to endogenous catecholamines and angiotensin II, thereby predisposing patients to perioperative vasodilation and refractory hypotension^[9].

Cardiac function also plays a significant role. Patients with reduced left ventricular ejection fraction are more vulnerable to vasoplegia due to impaired cardiovascular reserve and altered neurohormonal regulation. In this context, even moderate vasodilation may result in significant hemodynamic instability^[10–14].

Intraoperative factors are equally important. Prolonged duration of CPB is strongly associated with an increased risk of vasoplegia, likely due to sustained exposure to inflammatory mediators and endothelial dysfunction. Similarly, complex cardiac procedures, including combined valve surgery, aortic surgery, or reoperations, are associated with a higher inflammatory burden and an increased likelihood of VS^[9].

In addition, several patient-related characteristics contribute to increased susceptibility. Advanced age is associated with impaired vascular responsiveness and endothelial dysfunction, while chronic kidney disease is linked to baseline inflammation, oxidative stress, and altered vascular tone regulation. These factors have been consistently associated with a higher incidence and severity of vasoplegia in the perioperative setting^[4].

Overall, the presence of multiple risk factors may have a cumulative effect, significantly increasing the likelihood of developing VS. Identification of these high-risk patients is essential for implementing preventive strategies and optimizing perioperative hemodynamic management.

Discussion

VS represents a complex interplay between inflammatory, neurohormonal, and vascular mechanisms culminating in profound vasodilation and circulatory failure. Excessive production of NO via inducible NO synthase remains a central driver of

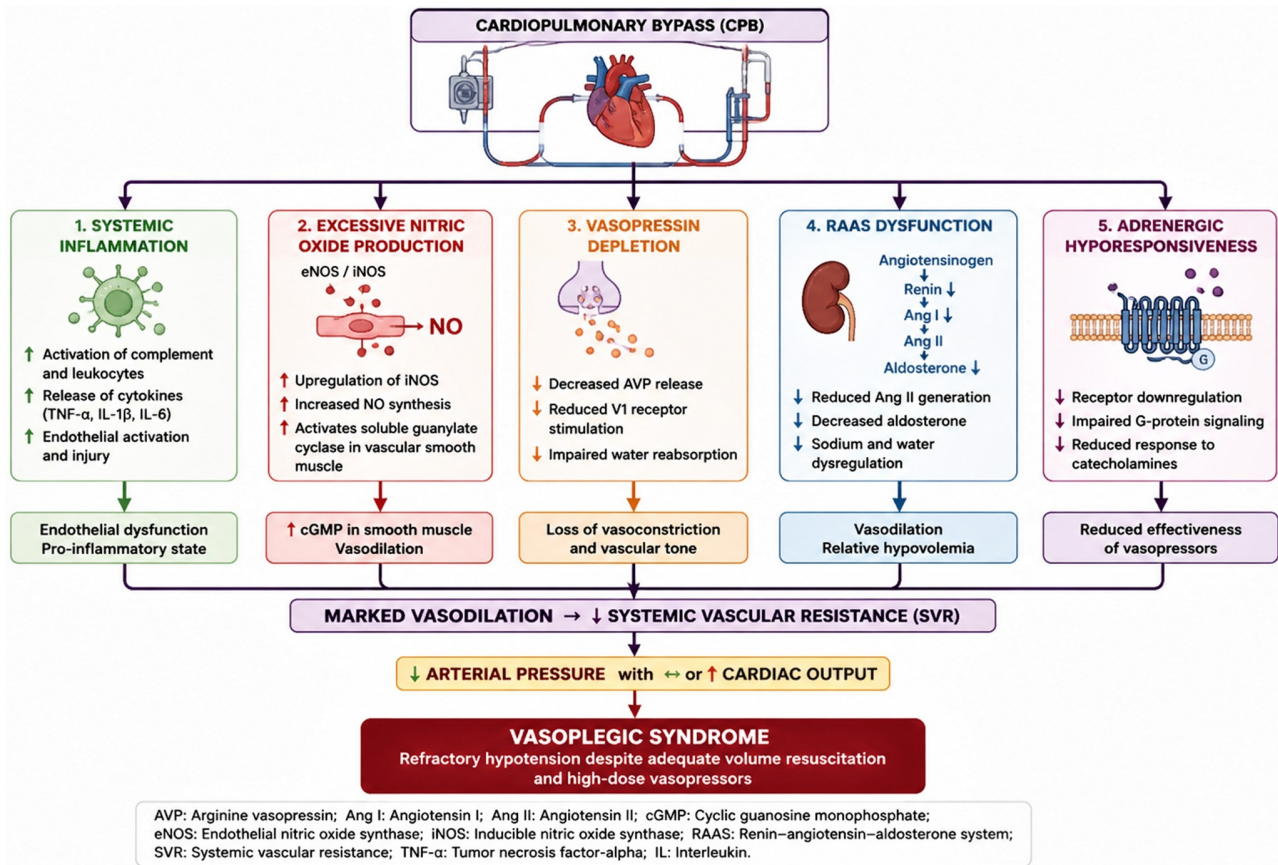


Figure 1. Pathophysiology of vasoplegic syndrome after cardiopulmonary bypass. This schematic represents the pathophysiological mechanisms underlying vasoplegic syndrome following cardiopulmonary bypass – including inflammation, nitric oxide overproduction, vasopressin depletion, RAAS dysfunction, and adrenergic hypo-responsiveness – that lead to systemic vasodilation and reduced SVR, and was created by the authors based on current literature.

vasoplegia, leading to decreased vascular tone and impaired responsiveness to vasopressors^[6]. This mechanism explains the limited efficacy of catecholamines in severe cases, where receptor desensitization and altered intracellular signaling further exacerbate vascular hyporesponsiveness. Recent high-quality reviews have reinforced these mechanisms while emphasizing persistent gaps in therapeutic standardization and clinical implementation^[15,16].

In parallel, relative vasopressin deficiency plays a key role in vasodilatory shock, including post-cardiac surgery vasoplegia^[7,17]. Depletion of endogenous vasopressin and impaired V1 receptor signaling contribute to refractory hypotension. Clinically, early initiation of vasopressin restores vascular tone and reduces catecholamine requirements, supporting its role as a cornerstone of multimodal management. Contemporary consensus recommendations advocate early integration of vasopressin within vasopressor strategies to enhance vascular responsiveness and limit catecholamine-related toxicity^[18,19].

The renin–angiotensin–aldosterone system has emerged as another critical pathway. Elevated plasma renin levels following CPB suggest a compensatory response to relative angiotensin II deficiency, contributing to persistent vasodilation and vasopressor resistance^[14]. This provides a strong physiological rationale for exogenous angiotensin II administration in refractory states^[8]. Emerging evidence indicates that angiotensin II improves hemodynamic stability, particularly when introduced early, although its impact on long-term outcomes remains uncertain^[17,18,20]. Notably, angiotensin II may improve systemic vascular tone without significantly increasing pulmonary vascular resistance, suggesting a favorable hemodynamic profile in patients with right ventricular vulnerability^[20]. Furthermore, early initiation appears to enhance hemodynamic stabilization and reduce catecholamine exposure, supporting a shift away from its use as a late rescue therapy^[18]. However, despite consistent improvements in mean arterial pressure and catecholamine-sparing effects, recent meta-analytic data have not demonstrated a clear mortality benefit^[20].

Adjunctive therapies targeting the NO–cGMP pathway, including methylene blue and hydroxocobalamin, have gained increasing attention. Methylene blue inhibits soluble guanylate cyclase, reducing cyclic GMP-mediated vasodilation, whereas hydroxocobalamin acts as a NO scavenger^[11,12]. Although both therapies have shown efficacy in observational studies and small trials, the absence of robust randomized evidence limits the strength of recommendations. Their safety profiles – including the risk of serotonin syndrome and laboratory interference – must be carefully considered. A comparative overview of pharmacologic therapies is provided in Table 1, while their corresponding levels of evidence are summarized in Table 2.

From a clinical standpoint, management should rely on an early, multimodal, and physiology-guided strategy rather than sequential escalation of single agents. Prompt identification of vasoplegia is essential and may be guided by key hemodynamic thresholds, including MAP ≥ 65 mmHg, SVR < 800 dyn·s/cm⁵, and cardiac index ≥ 2.2 L/min/m². Initial management includes optimization of preload, exclusion of reversible causes, and early initiation of norepinephrine. Rapid addition of vasopressin is recommended in catecholamine-resistant states, followed by consideration of adjunctive therapies such as methylene blue, hydroxocobalamin, or angiotensin II in refractory cases. Early combination therapy

Table 1

Comparative overview of pharmacologic therapies for vasoplegic syndrome in cardiac surgery.

Therapy	Mechanism	Advantages	Limitations
Norepinephrine ^[10]	Alpha-adrenergic vasoconstriction	First-line, widely available	High doses → adverse effects
Vasopressin ^[7]	V1 receptor stimulation	Catecholamine-sparing	Risk of ischemia
Methylene Blue ^[11]	Inhibits NO–cGMP pathway	Effective in refractory VS	Serotonin syndrome risk
Hydroxocobalamin ^[12]	NO scavenger	Rapid effect	Expensive, lab interference
Angiotensin II ^[8,13]	RAAS activation	Effective in refractory shock	Limited cardiac surgery data
Corticosteroids ^[10]	Anti-inflammatory	May reduce vasopressor need	Limited evidence

Overview of pharmacologic therapies for vasoplegic syndrome, highlighting mechanisms of action, clinical benefits, and limitations. First-line and adjunctive therapies target complementary vasoregulatory pathways to restore vascular tone in refractory vasodilatory states. Created by the authors based on synthesized data from the literature.

may improve hemodynamic control, reduce vasopressor exposure, and enhance organ perfusion^[15,19], as illustrated in Figure 2.

Overall, this review highlights a paradigm shift toward multimodal, mechanism-based hemodynamic management. Nevertheless, important limitations persist. A narrative design introduces potential selection bias, and heterogeneity in diagnostic criteria limits comparability across studies. Furthermore, evidence supporting adjunctive therapies remains limited, emphasizing the need for well-designed randomized controlled trials. The absence of validated biomarkers and standardized treatment algorithms continues to hinder the implementation of precision medicine approaches.

Future research should focus on refining diagnostic definitions, identifying reliable biomarkers – such as renin levels or vasopressor responsiveness – and conducting randomized trials

Table 2

Level of evidence for pharmacologic therapies in vasoplegic syndrome.

Therapy	Level of evidence ^a	Quality of evidence	Strength of recommendation
Norepinephrine	Level I–II	Moderate–High	Strong
Vasopressin	Level I–II	Moderate	Strong
Methylene Blue	Level II–III	Low–Moderate	Conditional
Hydroxocobalamin	Level III	Low	Conditional
Angiotensin II	Level II (emerging)	Moderate	Conditional
Corticosteroids	Level II–III	Low–Moderate	Conditional

Level of evidence for pharmacologic therapies used in vasoplegic syndrome following cardiac surgery. Evidence levels are based on randomized controlled trials, observational studies, and meta-analyses. Norepinephrine and vasopressin are supported by moderate-to-high-quality evidence and remain first-line therapies, whereas adjunctive treatments are supported by lower levels of evidence and are typically reserved for refractory cases. Created by the authors according to published evidence hierarchies.

^aLevel of evidence:

Level I: Randomized controlled trials/meta-analyses

Level II: Observational studies/cohort studies

Level III: Case series/expert opinion

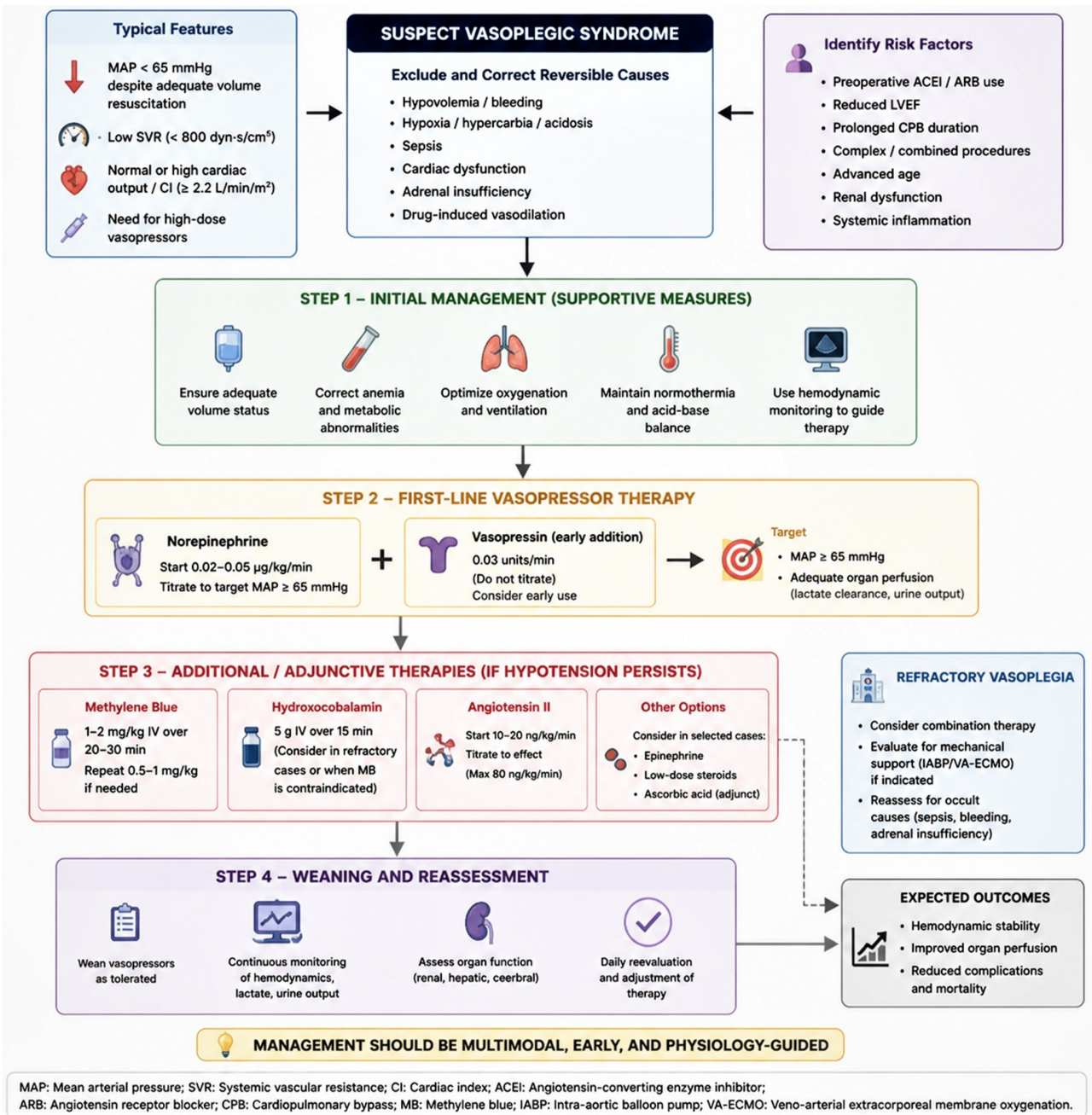


Figure 2. Perioperative management algorithm for vasoplegic syndrome after cardiac surgery. Stepwise perioperative management algorithm for vasoplegic syndrome after cardiac surgery, emphasizing early vasopressor therapy, exclusion of reversible causes, and escalation to adjunctive treatments in refractory cases. This algorithm was created by the authors based on current evidence and expert recommendations.

to optimize therapeutic sequencing. The development of individualized, algorithm-based strategies integrating advanced hemodynamic monitoring and targeted therapies represents a promising avenue to improve outcomes in VS.

Conclusion

VS remains a significant and potentially life-threatening complication in cardiac surgery, particularly in the context of CPB. Its multifactorial pathophysiology, involving inflammatory

activation, NO overproduction, and neurohormonal dysregulation, contributes to profound vasodilation and resistance to conventional vasopressor therapy.

Early recognition and prompt differentiation from other causes of perioperative shock are essential for initiating appropriate treatment and preventing progression to multi-organ dysfunction. Current management strategies emphasize a multimodal, physiology-guided approach, combining catecholamines with non-adrenergic agents such as vasopressin, methylene blue, hydroxocobalamin, and angiotensin II.

Despite advances in understanding and treatment, important gaps remain concerning optimal therapeutic sequencing, patient selection, and long-term outcomes. Future research should focus on standardized definitions, biomarker-guided strategies, and well-designed randomized controlled trials to refine management algorithms and improve patient outcomes.

Ethical approval

Not applicable.

Consent

Not applicable.

Sources of funding

No funding was received for this work.

Author contributions

S.K.: conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, supervision, validation, manuscript drafting, critical revision, and final approval of the manuscript; M.D.: conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, supervision, validation, and critical revision of the manuscript; H.E.B.: conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, supervision, validation, and critical revision of the manuscript; H.A.E.G.: investigation, methodology, project administration, resources, supervision, validation, and manuscript review; M.D.: investigation, methodology, project administration, resources, supervision, validation, and manuscript review. All authors read and approved the final version of the manuscript.

Conflicts of interest disclosure

The authors declare no conflicts of interest.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

Said Khallikane is the guarantor of this work and accepts full responsibility for the integrity of the study, the accuracy of the manuscript, and the decision to submit it for publication.

Provenance and peer review

Not commissioned; externally peer reviewed.

Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Acknowledgements

Not applicable.

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