

Surgical and Endovascular Management of Aortic Thrombosis in COVID-19 and Vaccine-Induced Immune Thrombotic Thrombocytopenia

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Background: COVID-19 has been associated with a hypercoagulable state, leading to various thrombotic complications, including aortic thrombosis, a rare but severe manifestation requiring surgical intervention. Additionally, vaccine-induced immune thrombotic thrombocytopenia (VITT), linked to adenoviral vector vaccines, presents unique surgical challenges due to a heightened risk of thrombosis and bleeding. This review focuses on the surgical management of COVID-19-associated aortic thrombosis and VITT-related large-vessel occlusions.

Results: Surgical intervention in COVID-19-associated aortic thrombosis depends on thrombus burden, patient stability, and associated comorbidities. Open thrombectomy, aortic bypass, and hybrid endovascular techniques have been employed, with perioperative anticoagulation being critical to prevent recurrence. High thrombus burden cases often require open repair, while endovascular approaches are preferred in high-risk patients. Mortality rates remain elevated (up to 30%), with post-surgical complications including recurrent thrombosis and limb loss. In VITT cases, surgical revascularization is complicated by thrombocytopenia and a prothrombotic state, necessitating non-heparin anticoagulation and close hematologic monitoring. Delayed diagnosis and inappropriate anticoagulation significantly worsen outcomes.

Conclusion: The surgical management of aortic thrombosis in COVID-19 and VITT patients requires a multidisciplinary approach, incorporating vascular surgery, hematology, and intensive care. Early intervention with individualized surgical and anticoagulation strategies is crucial in optimizing outcomes. Further research is needed to refine surgical decision-making, improve postoperative anticoagulation protocols, and enhance patient survival in these complex thrombotic conditions.

Keywords: aortic thrombosis, COVID-19, vaccine-induced immune thrombotic thrombocytopenia, VITT, surgical management, endovascular therapy, thrombectomy, vascular surgery, thrombotic complications

Background

The outbreak of SARS-CoV-2, followed by the COVID-19 pandemic, has significantly impacted various aspects of human life. While COVID-19 is primarily recognized as a respiratory illness with symptoms that typically include fever, cough, fatigue, and dyspnea, it can also manifest in vascular, renal, cardiac, and hematologic complications.^{1,2}

COVID-19 has been increasingly associated with a hypercoagulable state, leading to an elevated risk of thromboembolic events, including deep vein thrombosis (DVT), pulmonary embolism (PE), and arterial thrombosis. This prothrombotic environment is largely attributed to endothelial injury, inflammation, and coagulopathy. Studies of critically ill COVID-19 patients have reported increased levels of hypercoagulability markers, such as D-dimer, fibrinogen, and factor VIII, alongside reduced levels of natural anticoagulants, including protein C, protein S, and antithrombin.³

One particularly severe manifestation of arterial thrombosis in COVID-19 patients is aortic thrombosis, a rare but life-threatening condition characterized by acute occlusion of the aorta. In the general population, aortic thrombosis is



exceedingly rare, with an estimated incidence of less than 1 per 100,000 person-years, often linked to underlying atherosclerosis or aneurysmal disease. However, in COVID-19 patients, the incidence rises significantly, reaching 2.7–8% in severe or critically ill cases and up to 4.4% in ICU-admitted patients, with mortality rates reaching approximately 30%.⁴ The clinical presentation varies based on thrombus location and size, with possible outcomes including acute limb ischemia, mesenteric ischemia, myocardial infarction, or stroke.^{5,6}

Another emerging thrombotic entity in the COVID-19 era is vaccine-induced immune thrombotic thrombocytopenia (VITT), a rare but severe condition primarily associated with adenoviral vector-based COVID-19 vaccines (eg, AstraZeneca and Janssen). VITT is characterized by thrombocytopenia and the development of life-threatening arterial and venous thromboses, including cerebral venous sinus thrombosis (CVST) and large-vessel occlusions.⁷

While VITT has been predominantly linked to COVID-19 vaccines, similar immune-mediated thrombotic syndromes (eg, akin to heparin-induced thrombocytopenia) have not been widely reported with non-COVID vaccines, though theoretical risks exist in other adenoviral vector platforms.^{8,9}

The rising incidence of aortic thromboembolism and acute aortic occlusion further underscores the indispensable role of vascular surgical intervention in optimizing patient outcomes. Early recognition and prompt surgical management remain key in mitigating mortality and long-term disability associated with COVID-19-associated thrombotic events.¹⁰

Timely surgical intervention in these cases offers several benefits: it helps prevent complications such as limb ischemia, renal infarction, and mesenteric ischemia. Delays in surgical intervention can lead to irreversible damage to affected organs and increase the need for additional secondary surgeries, ultimately reducing survival rates.¹¹

Methods

A systematic literature search was conducted using PubMed, Scopus, and Google Scholar databases from January 2020 to October 2025. Search terms included “aortic thrombosis” AND (“COVID-19” OR “SARS-CoV-2” OR “VITT” OR “vaccine-induced immune thrombotic thrombocytopenia”), combined with “surgical management”, “endovascular therapy”, “case reports”, “guidelines”, and “outcomes”. Inclusion criteria encompassed English-language articles, case reports, reviews, and guidelines on diagnosis, mechanisms, and treatment. Exclusion criteria included non-relevant thrombotic conditions or non-human studies. Data were extracted on clinical manifestations, diagnostic criteria, treatment strategies, and mortality. A narrative synthesis was performed due to the heterogeneity of case reports.

COVID-19 and Coagulopathy

COVID-19-Associated Coagulopathy (CAC)

COVID-19-associated coagulopathy (CAC) is a severe hematological complication of SARS-CoV-2 infection, characterized by hypercoagulability, endothelial dysfunction, and systemic inflammation, all of which contribute to an increased risk of thrombotic events.^{12,13} Studies indicate that COVID-19 promotes a prothrombotic state through endothelial injury, platelet activation, and an exaggerated immune response, leading to both venous and arterial thrombosis.¹⁴ Among arterial complications, aortic thrombosis has emerged as a rare but critical manifestation, often resulting in life-threatening ischemic, including limb loss and death even in young individuals.^{12,15}

Aortic thrombosis has also been implicated in cerebrovascular events. Histopathological analysis revealed that the thrombus was platelet-rich and formed on an eroded atherosclerotic plaque, rather than an ulcerated lesion, suggesting that SARS-CoV-2 may directly trigger thrombus formation through endothelial injury.¹⁶ This aligns with broader evidence that COVID-19 exacerbates pre-existing vascular conditions, increasing the risk of acute aortic syndromes.¹³ To differentiate COVID-19-associated aortic thrombosis from thrombosis due to pre-existing plaques, key features include: COVID-19 thrombi often occur in vessels with minimal atherosclerosis, are platelet-rich with endothelial dysfunction, and are associated with elevated inflammatory markers (eg, CRP, IL-6) and D-dimer >1000 ng/mL. In contrast, plaque rupture thrombosis involves ulcerated lesions with lipid-rich cores, typically in patients with established atherosclerosis, diagnosed via CT angiography showing plaque disruption.¹⁷

The implications of CAC extend significantly into surgical practice, where patients undergoing procedures face heightened risks of both thrombosis and bleeding.¹⁴

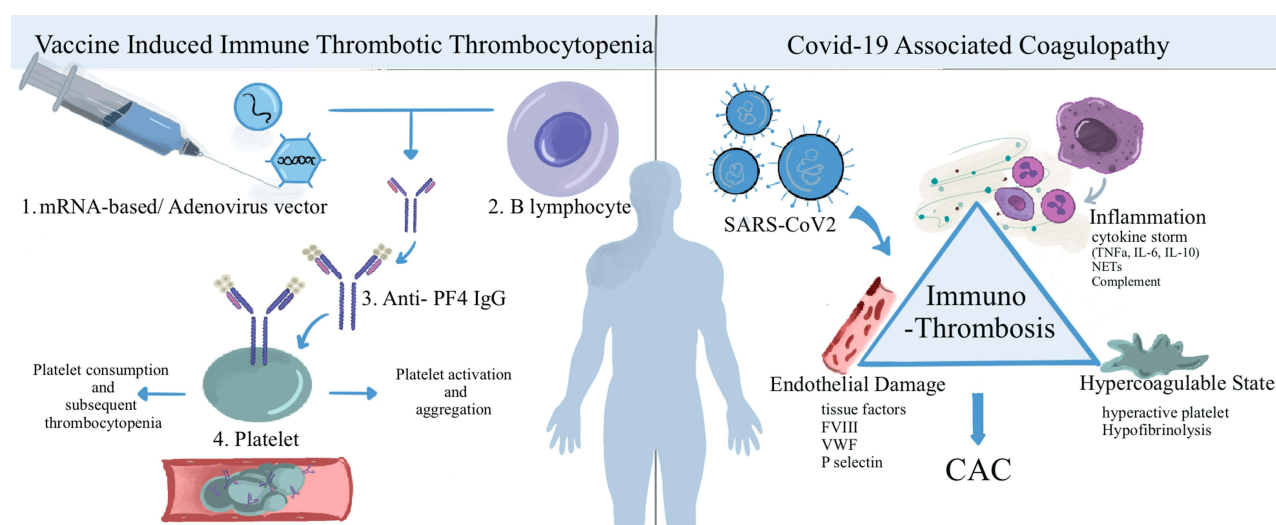


Figure 1 Schematic representation of vascular complications associated with aortic thrombosis in COVID-19. The figure illustrates thrombus formation, vascular obstruction, and potential sequelae, including embolization and aortic dissection. Differences between CAC and VITT are highlighted, including the complete mechanistic pathway for VITT: vaccine-induced PF4-antibody complexes activate platelets via FcγRIIa, leading to aggregation; inflammatory cells (neutrophils) release NETs, and cytokines (IL-1β, TNF-α, IL-6) amplify endothelial activation and coagulopathy via NLRP3 inflammasome. In CAC, endothelial injury from SARS-CoV-2 triggers cytokine storm (IL-6, TNF-α) and complement activation, promoting platelet-rich thrombi on eroded plaques.

Vaccine-Induced Immune Thrombotic Thrombocytopenia (VITT)

Vaccine-induced immune thrombotic thrombocytopenia (VITT) is a rare, serious side effect. It occurs in a small number of people after some COVID-19 vaccines. VITT features a puzzling mix of low platelet count, known as thrombocytopenia, and thrombosis.^{6,18} Although most attention has been on the venous aspects, new evidence suggests that events secondary to can also lead to arterial thrombotic events in unusual locations, such as the aorta and extracranial vessels, potentially causing strokes.⁶ The immune response triggers platelet activation, which, in rare cases, leads to unusual arterial clots. This mechanism differs from the typical inflammation and coagulation seen in COVID-19.¹⁸

The mechanisms underlying VITT-related large-vessel occlusions involve immune-mediated platelet activation, where vaccine components bind to PF4, forming complexes that trigger IgG antibodies. This leads to platelet aggregation and thrombus formation. Additionally, inflammatory cells (eg, neutrophils via NETosis) and cytokines (eg, IL-1β, TNF-α, IL-6) amplify the process, promoting endothelial activation and coagulopathy. The NLRP3 inflammasome plays a role in cytokine release, exacerbating thrombosis.^{19–21} Diagnostic criteria for VITT include: vaccination 4–30 days prior, thrombocytopenia ($<150,000/\text{mm}^3$), thrombosis (arterial/venous), elevated D-dimer ($>4\times$ upper limit), and positive anti-PF4 antibodies.^{22,23} Differentiation from plaque-related thrombosis: VITT thrombi are immune-driven, often in atypical sites without atherosclerosis, with profound thrombocytopenia absent in plaque rupture.^{17,24}

COVID-19 and VITT both induce thrombosis, but their underlying mechanisms differ. COVID-19 induces a prothrombotic state through inflammation, endothelial injury, and cytokine storm, which complicates multi-organ involvement and surgery. In contrast, VITT is an immune-mediated phenomenon triggered by vaccination, where platelet activation leads to clot formation. Consequently, management strategies must be adapted accordingly.²⁵ The treatment of both illnesses depends on anticoagulation. However, the type and level of therapy may vary. Clinicians may try to avoid heparin in VITT.²⁶ Both conditions require anticoagulation therapy, but the choice of anticoagulants differs. For VITT, clinicians may avoid heparin due to concerns about heparin-induced thrombocytopenia (HIT), preferring non-heparin anticoagulants such as direct thrombin inhibitors or fondaparinux.²⁶ These distinctions necessitate tailored surgical planning and perioperative management strategies for each condition.²⁷ Figure 1 presents and explains both pathways, and Figure 2 serves as a complement, providing further clarification and context.

Recent guidelines from the International Society on Thrombosis and Haemostasis (ISTH) recommend therapeutic anticoagulation for COVID-19-related thrombosis in hospitalized patients and non-heparin options for VITT.^{28,29}

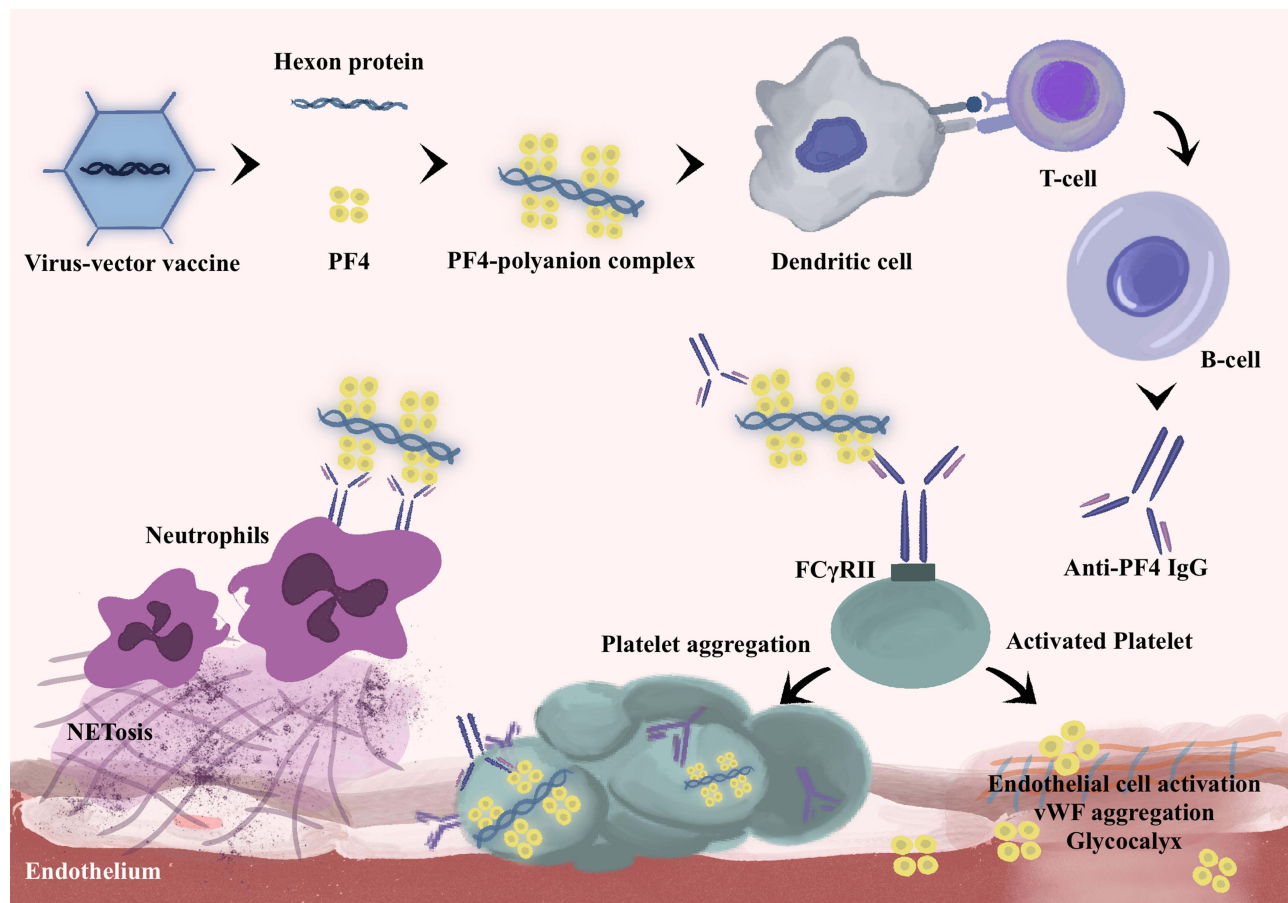


Figure 2 Proposed mechanism of vaccine-induced immune thrombotic thrombocytopenia (VITT). Viral vector components interact with platelet factor 4 (PF4) to form PF4-polyanion complexes, leading to anti-PF4 antibody production. The immune complexes activate platelets via FcγRIIa, triggering platelet aggregation, NETosis, and endothelial activation, resulting in thrombosis and thrombocytopenia.

Pre-Operative Considerations and Risks

In COVID-19 patients, aortic thrombosis often presents with acute limb ischemia or is incidentally detected during diagnostic imaging.³ COVID-19-associated aortic thrombosis carries high morbidity and mortality, with 30-day mortality rates of up to 30% and amputation rates reaching 50%.³⁰

A study conducted by Bagheri et al identified key factors influencing thromboembolic events in COVID-19 patients. ICU admission in terminal-stage disease was a major predictor of arterial thrombosis and pulmonary embolism, while male sex was associated with a lower risk.¹⁶ Other studies have supported these findings, identifying D-dimer as a critical prognostic marker in COVID-19-associated thrombosis.^{31–33} D-dimer levels showed a borderline association with total thromboembolic events and the occurrence of these events is related to the D-dimer value.^{16,22}

Thrombosis has been reported even in arterial regions with minimal atherosclerotic plaque burden, such as the aortic arch. SARS-CoV-2 appears to trigger arterial thrombosis primarily in individuals with pre-existing atherosclerosis rather than solely through hypercoagulability.³³

COVID-19 patients undergoing surgery require special considerations to minimize transmission risks and complications. Elective surgeries performed during the incubation period of COVID-19 have been linked to high rates of postoperative pneumonia, ICU admission (44.1%), and mortality (20.5%).²⁸ Surgical management in COVID-19 patients demands enhanced safety protocols to reduce contagion between patients and healthcare personnel.²⁹ Non-essential surgeries should be postponed, and perioperative management should prioritize a multidisciplinary approach.³⁴ Severe thrombotic complications, such as intestinal ischemia and acute limb ischemia, may necessitate surgical intervention, but these procedures carry significant mortality risks.³⁵

Surgical Challenges

Open and Endovascular Options

The management of aortic thrombosis in COVID-19 depends on thrombus burden, location, and associated complications. The surgical approach varied based on the severity and type of aortic pathology. Open surgery was the most commonly employed method, performed in 58.8% of cases reported by Silvestri et al. Procedures included ascending aorta and hemiarch replacement, and aortic graft revisions.¹³ Endovascular techniques are selectively used, particularly for thrombotic complications and aneurysmal disease. However, 17.6% of patients succumbed before surgery due to acute thrombosis or aortic dissection, highlighting the urgency of early intervention.¹⁵

Janula et al described a successful trans-aortic thromboembolectomy, demonstrating that surgical intervention can lead to thrombus resolution and functional recovery.³⁶ Although endovascular techniques such as thrombectomy and stenting have not been widely reported in COVID-19 cases, they may be preferable for high-risk surgical patients. However, further research is needed in this area.²⁰

While systemic anticoagulation alone may be considered in select cases, large thrombi carry a high risk of embolization and often necessitate surgical or endovascular intervention. Early anticoagulation and endovascular treatments play critical roles in managing vascular emergencies in COVID-19 patients, despite potential complications such as late endoleaks.¹⁹

A comprehensive summary of published case reports (n=50+ from global literature, 2020–2025) shows: Open surgery advantages include complete thrombus removal in extensive cases but disadvantages are higher invasiveness and perioperative risks (eg, infection in COVID-19). Endovascular therapy offers minimally invasive benefits, faster recovery, but risks include incomplete resolution and endoleaks. Anticoagulation (eg, LMWH) and thrombolysis are adjuncts, with immunomodulation (IVIG, steroids) for VITT. Overall mortality ~30% for COVID-19 aortic thrombosis, higher in untreated cases.^{13,15,16,18,25–27,30,37}

Exeptions for VITT Patients

Surgically managing aortic thrombosis in the context of VITT presents distinct challenges, particularly in balancing bleeding and thrombotic risks. The concurrent presence of low platelet levels increases the risk of hemorrhage, while the tendency for new clot formation further complicates surgical decision-making and perioperative management.⁵ Treatment typically includes intravenous immunoglobulins, corticosteroids, and anticoagulation, avoiding heparin.³⁸ Vascular interventions may be necessary, with argatroban as an alternative anticoagulant.³⁹ Endovascular treatment in VITT cases has shown promising outcomes, with a 90-day modified Rankin Scale score of 2 reported in treated patients.²⁰ Early diagnosis for favorable outcomes is crucial, with VITT suspected when platelet count is below 100,000/mm³ and D-dimer is elevated.⁴⁰ For VITT, pharmacological options (eg, IVIG + non-heparin anticoagulants) are preferred initially due to high surgical risks; surgery is reserved for life-threatening occlusions.

Endovascular treatment (EVT) has emerged as a potential intervention for VITT patients, particularly in cases with extensive clot burden or clinical deterioration. While EVT can improve local blood flow, mortality remains high at 50%.⁴⁰ For VITT, pharmacological options (eg, IVIG + non-heparin anticoagulants) are preferred initially due to high surgical risks; surgery is reserved for life-threatening occlusions.^{22,29}

Elective surgeries should take place once the patient has recovered from vaccine side effects. They should also be done when the immune response is steady. This reduces the risk of complications from immune activation or coagulopathy. For patients who have received vaccinations, the probability of developing severe COVID-19 complications is reduced. This decrease in risk minimizes the need to postpone necessary surgical interventions.⁴¹

Despite advancements in surgical interventions, prognosis remains guarded, particularly in critically ill patients. Jokšić-Mazinjanin et al highlighted the necessity of a multidisciplinary approach, combining surgical, endovascular, and intensive care strategies for optimal outcomes.²¹ Table 1 summarizes the surgical and endovascular management strategies for aortic thrombosis in COVID-19 and VITT patients, as shown.

Table 1 Surgical and Endovascular Treatment Approaches for Aortic Thrombosis in COVID-19 and VITT Patients

Factor	Open Surgery	Endovascular Techniques (EVT)	Considerations in VITT Patients
Indications	Large thrombi, extensive aortic pathology, failed anticoagulation	High-risk surgical patients, aneurysmal disease, localized thrombus	High thrombotic risk with concurrent bleeding concerns
Common Procedures	Ascending aorta and hemiarch replacement, thromboembolectomy, aortic graft revisions	Thrombectomy, stenting, EVT for clot burden reduction	EVT preferred if clot burden is high and surgery is risky
Reported Success	58.8% cases reported by Silvestri et al underwent open surgery	Limited reports in COVID-19; EVT improves local blood flow	90-day functional recovery in EVT-treated patients
Mortality Risks	17.6% succumbed before surgery due to acute thrombosis/dissection	Potential late complications (eg, endoleaks)	50% mortality despite EVT in severe cases
Anticoagulation Approach	Early systemic anticoagulation to prevent embolization	Often combined with anticoagulation for clot stabilization	Avoid heparin; use argatroban, IVIG, corticosteroids
Timing of Intervention	Urgent in acute cases, elective if stabilized	Preferable in patients at high surgical risk	Performed after stabilization of immune response
Multidisciplinary Considerations	Requires ICU support, cardiac surgery, and vascular expertise	Endovascular specialists needed for optimal outcomes	Combined hematology, vascular surgery, and ICU care recommended

Notes: This table outlines the surgical and endovascular treatment approaches for aortic thrombosis in COVID-19 and VITT patients, including procedure types, associated risks, and outcomes.

Post-Operative Care and Outcomes

The postoperative period is a crucial determinant of aortic thrombosis surgery success, particularly in COVID-19 patients who face both the systemic effects of the virus and the inherent risks of vascular surgery.⁴² Ensuring hemodynamic stability is the top priority, necessitating continuous monitoring of blood pressure, heart rate, and oxygen saturation to detect early signs of compromised perfusion. Bleeding control is also essential, especially in patients receiving anticoagulation therapy, as they have an increased risk of hemorrhage.⁴³

Postoperative outcomes vary significantly, with some patients experiencing complications such as renal failure requiring dialysis or prolonged viral positivity. These findings underscore the challenges of managing acute aortic conditions in COVID-19 patients, necessitating an individualized approach based on disease progression and comorbidities.¹³

For patients with VITT, postoperative monitoring requires a specialized approach, including platelet count assessments, tracking inflammatory markers, and evaluating coagulation status. The primary objective is to identify early signs of bleeding or recurrent thrombosis and facilitate prompt medical intervention.⁴⁴

Respiratory support is often required, as lung damage caused by COVID-19 can result in respiratory insufficiency, necessitating supplemental oxygen or mechanical ventilation. Early mobilization is key to reducing the risk of thrombotic events and expediting recovery, encouraging patients to begin ambulation as soon as physically feasible.⁸

Prevention of Recurrence and Long-Term Anticoagulation

Given the hypercoagulable state associated with COVID-19, preventing recurrent thrombosis is a key concern. Systemic anticoagulation remains the cornerstone of prevention, with treatment plans tailored to each patient's individual risk factors, including renal function and bleeding propensity.⁸ Regular monitoring of coagulation markers such as D-dimer, fibrinogen, and platelet levels is essential for assessing anticoagulation efficacy and detecting potential complications early.³²

Patients with extensive thrombotic burden or severe COVID-19 may require extended anticoagulation therapy. Additionally, addressing systemic inflammation is crucial, as it contributes to the hypercoagulable state. A combined approach using anticoagulation and anti-inflammatory therapies has been shown to improve clinical outcomes.⁴⁵

Complications and Outcomes

Postoperative complications in this high-risk population can be classified into two major categories: surgical complications and systemic complications exacerbated by COVID-19. Maintaining graft patency is critical for restoring blood flow, necessitating routine imaging and clinical assessments to detect early signs of graft failure. If failure occurs, re-intervention via endovascular techniques or additional surgery may be required.⁴⁶ The hypercoagulable state also elevates the risk of embolic events, particularly strokes. Early neuroimaging (CT or MRI) and continuous neurological monitoring are essential for rapid detection and timely intervention.^{14,47} However long term patency of grafts in this patients is not fully investigated yet.

Several case reports illustrate the diverse clinical presentations and outcomes of COVID-19-associated aortic thrombosis. Burley et al (2021) described a case of aortic thrombosis leading to acute limb ischemia, necessitating emergency surgical intervention.³² Despite aggressive management—including thrombectomy, bypass, and anticoagulation—the limb was non-salvageable, emphasizing the severe and often irreversible nature of thrombotic complications in COVID-19. Similarly, Andriamizanaka et al (2021) documented a case of extensive arterial thrombosis requiring limb amputation, further demonstrating the devastating consequences of COVID-19-induced hypercoagulability.²²

For vascular surgeries, particularly those addressing aortic thrombosis, minimally invasive techniques such as endovascular approaches are preferred to reduce perioperative morbidity, especially in critically ill COVID-19 patients.¹⁰

Long-Term Prognosis and Follow-Up

Long-term outcomes following aortic thrombosis surgery remain an area of active research. While surgery is often life-saving, many patients continue to experience persistent COVID-19-related symptoms and chronic vascular complications. Residual organ dysfunction, particularly affecting the lungs and cardiovascular system, may significantly impact quality of life and overall prognosis.⁴⁸ Chronic anticoagulation is often necessary to prevent recurrent thrombosis, requiring regular adjustments based on individual risk assessments.⁴¹ Routine follow-up imaging is essential for detecting late complications, such as graft deterioration or re-thrombosis, which may develop months or even years after the initial surgery.⁴⁹

Long-term follow-up care, involving primary care physicians, cardiologists, and vascular surgeons, is essential to ensure ongoing monitoring, comorbidity management, and early intervention in case of complications.⁵⁰ Post-discharge thrombotic events in COVID-19 patients remain a concern, though they are relatively uncommon. A meta-analysis by Rungjirajittranon et al reported an overall thrombosis incidence of 1.3%, with venous thrombosis at 0.7% and arterial thrombosis at 0.6%, while mortality reached 2.8%.⁵¹

Critically ill COVID-19 patients in intensive care units face an even greater risk. Literature suggests that arterial thrombosis incidence in ICU-admitted patients is 4.4%, with symptomatic multi-arterial involvement and an associated mortality rate of approximately 20%.⁹

Conclusion

The surgical management of aortic thrombosis in COVID-19 and VITT patients presents significant challenges due to the complex interplay of hypercoagulability, endothelial dysfunction, and perioperative bleeding risks. Open thrombectomy and aortic bypass remain essential for patients with extensive thrombus burden, while endovascular approaches offer a less invasive alternative in high-risk individuals. In VITT cases, surgical intervention must be accompanied by careful anticoagulation strategies, avoiding heparin-based therapies to mitigate further thrombotic events. Optimal surgical outcomes depend on early diagnosis, a tailored operative approach, and multidisciplinary perioperative management. Outcome and mortality scores indicate high risks: overall mortality for COVID-19 aortic thrombosis is ~30%, with 6-month post-procedure mortality at 32.1% in confirmed cases; for VITT-related thrombosis, mortality is 32–40%, with FAPIC scores predicting risks from 2% (score 0) to 90% (score 5).^{47,52–56} Further research is needed to refine surgical techniques, improve patient selection criteria, and establish standardized protocols for postoperative anticoagulation in this unique patient population.

Abbreviations

SARS-CoV-2, Severe Acute Respiratory Syndrome Coronavirus 2; COVID-19, Coronavirus Disease 2019; DVT, Deep Vein Thrombosis; PE, Pulmonary Embolism; VITT, Vaccine-Induced Immune Thrombotic Thrombocytopenia; CVST, Cerebral Venous Sinus Thrombosis; CAC, COVID-19-Associated Coagulopathy; HIT, Heparin-Induced Thrombocytopenia; EVT, Endovascular Treatment.

Ethics Approval and Consent to Participate

This study did not involve human participants, human data, or human tissue. Therefore, ethics approval and consent to participate are not applicable.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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