

Review Article

Prevalence of coronary thrombosis after primary angioplasty in patients with acute myocardial infarction and COVID-19: a literature review*Prevalência de trombose coronariana após angioplastia primária em pacientes com infarto agudo de miocárdio e COVID-19: uma revisão bibliográfica*

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ABSTRACT: Introduction: COVID-19 is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and individuals most likely to experience more severe symptoms are older adults with comorbidities such as diabetes, cardiovascular diseases, cancer, and a history of smoking. In COVID-19 infection, the release of markers of atherosclerosis and inflammation leads to a hypercoagulable state, which, in turn, can increase the risk for acute coronary syndrome and stent thrombosis after primary angioplasty. Objective: To assess the prevalence of coronary thrombosis after primary angioplasty in patients with acute myocardial infarction infected by COVID-19. Material and methods: This is a bibliographical research in the form of a literature review, based on studies published in the following databases: PubMed, Medline, SciELO, NCBI and Lilacs. Results: Regarding the revascularization procedure, primary angioplasty is the preferred technique for harm reduction, better outcomes, and preservation of ventricular function in acute ischemic heart. However, the incidence of thromboembolic events is extremely high in patients with ST-segment elevation acute myocardial infarction infected with

SARS-CoV-2, according to some studies. Conclusion: There is a higher prevalence of coronary thrombosis in patients with ST-segment elevation acute myocardial infarction infected with SARS-CoV-2 after undergoing primary angioplasty.

Keywords: COVID-19; SARS-CoV-2; Infarction; Thrombosis; Angioplasty.

RESUMO: Introdução: A COVID-19 é causada pelo coronavírus da síndrome respiratória aguda grave (SARS-CoV-2) e os indivíduos com maior probabilidade de apresentarem sintomas mais graves são idosos com comorbidades como: diabetes, doenças cardiovasculares, câncer e histórico de tabagismo. Esta doença cursa com liberação de mediadores inflamatórios e ateroscleróticos, gerando a instalação do estado hipercoagulável, o que levanta a possibilidade de risco aumentado para síndromes coronárias agudas e trombose coronariana pós angioplastia primária. Objetivo: Avaliar a prevalência de trombose coronariana após angioplastia primária em pacientes com infarto agudo de miocárdio infectados pela COVID-19.

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Material e métodos: Trata-se de uma pesquisa bibliográfica do tipo revisão de literatura tendo como base produções científicas publicadas nas seguintes bases de dados: PubMed, Medline, SciELO, NCBI e Lilacs. **Resultados:** Quanto a técnica de revascularização a ser escolhida temos que, a angioplastia primária é utilizada como principal estratégia para redução de danos, melhor desfecho e preservação da função ventricular, na doença cardíaca isquêmica aguda. Entretanto, a incidência de eventos tromboembólicos é extremamente alta em pacientes

INTRODUCTION

COVID-19 is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and individuals most likely to experience more severe symptoms are older adults with comorbidities such as diabetes, cardiovascular diseases, cancer, and a history of smoking. Some of the complications include acute respiratory distress syndrome (ARDS), septic shock, metabolic acidosis, coagulation disorders, and rapid progression to multiple organ dysfunction. Acute infections, such as COVID-19, are also considered predisposing factors for vascular events and, consequently, myocardial damage¹⁻⁴.

In COVID-19 infection, the release of markers of atherosclerosis and inflammation leads to a hypercoagulable state, which, in turn, can increase the risk for acute coronary syndrome and stent thrombosis⁵. A Dutch study found a 31% incidence of thromboembolic complications in patients admitted to the ICU with COVID-19⁶. Furthermore, after a viral respiratory infection, there is a greater risk of complications, mainly acute myocardial infarction (AMI)^{4,6-8}.

Primary angioplasty has been the technique used in acute ischemic heart disease to reduce ischemic damage, improve outcomes, and preserve ventricular function⁹. However, inflammatory cytokines and apoptosis of immune cells in viral infections, which are associated with the extracellular vesicles present in the cell, play an important role in the occurrence of thrombosis and worsening of the prognosis of COVID-19 patients¹⁰. The development of post-thrombotic syndrome is more common after the tenth day of illness¹¹.

OBJECTIVE

To assess the prevalence of coronary thrombosis after primary angioplasty in patients with acute myocardial infarction infected by COVID-19.

MATERIAL AND METHODS

This study is a bibliographical research in the form of a literature review. The search was conducted in Portuguese and English in the databases Medline (Medical Literature Analysis and Tetrietral System On-Line) and

com infarto agudo do miocárdio com supra de ST infectados com o SARS-Cov-2 submetidos à angioplastia primária, de acordo com alguns estudos. **Conclusão:** Há maior prevalência de trombose coronariana em pacientes com infarto agudo do miocárdio com supra de ST infectados com o SARS-Cov-2 após serem submetidos à angioplastia primária.

Palavras-chaves: COVID-19; SARS-Cov-2; Infarto; Trombose; Angioplastia.

SciELO (Scientific Electronic Library OnLine), with the purpose of expanding scientific knowledge. The search allowed gathering all the findings, providing the basis and foundation for this research. Advanced search tools were used with the keywords COVID-19, SARS-CoV-2, Infarction, Thrombosis and Angioplasty in different combinations, searching for articles that addressed the presence or absence of coronary thrombosis after primary angioplasty in patients with acute myocardial infarction and COVID. A total of 46 relevant articles were found and selected, giving preference to those published after the beginning of the SARS-CoV-2 pandemic.

RESULTS

A cohort study with 346 COVID-19 positive and negative patients evaluated the presence of myocardial injury in hospitalized patients, revealing elevated cardiac enzymes (troponins) in some of them and concluding that non-ischemic myocardial lesions were present in young patients with few comorbidities and that hospital mortality and length of stay had increased¹². In addition, a French study compared 150 patients with severe acute respiratory illness syndrome (ARDS) and COVID-19 or only ARDS and found that the rate of thromboembolic complications was 11.7% against 2.1%, a 4 times higher incidence in COVID-19 ARDS patients, despite anticoagulation⁶, leading to the conclusion that the incidence of thrombotic events is higher in patients infected with SARS-CoV-2 compared to other viral pneumonias¹³.

Likewise, a preliminary study conducted in New York evaluated 18 patients with COVID-19 who had ST-segment elevation, of which half underwent coronary angiography and, of these, two thirds had obstructive disease. It was noted that all patients had elevated D-dimer levels, in contrast with the natural evolution of patients who present with ST-segment elevation myocardial infarction, of which 64% evolve with normal D-dimer levels¹⁴. Therefore, myocardial injury in patients with COVID-19 may be due to cytokine storm, plaque rupture, hypoxic injury, microthrombi, coronary spasm, or direct endothelial or vascular injury. In addition, myocardial interstitial edema has been shown on magnetic resonance in these patients¹⁵. Furthermore, a meta-analysis that looked for postmortem findings associated with COVID-19 found remarkable levels of thromboembolism,

cardiac myocyte necrosis, hemophagocytosis, and diffuse alveolar damage¹⁶.

In addition, the 70-day mortality rate after reperfusion was 4.49% in the pandemic, a value higher than in the previous year, before the COVID-19 outbreak, when it was 2.73%¹⁷. Similarly, an observational study of 115 patients admitted with ST-segment elevation acute myocardial infarction (STEMI) treated with primary percutaneous coronary intervention at Barts Heart Center in London found that patients with STEMI and concurrent COVID-19 infection may have a higher thrombus burden and poorer outcome¹⁸. Likewise, a case report of a 68-year-old diabetic patient with STEMI and COVID-19 described that, due to flaws in the hospital system, the patient did not receive primary percutaneous coronary intervention and was instead submitted to fibrinolytic therapy and referred for rescue angioplasty. At the end of the case, the patient died due to massive thrombotic burden. Despite the need for further research, it is evident that this type of situation can occur in other cardiology centers¹⁹.

Fibrinolytic therapy may be related, as the use of low molecular weight heparin (LMWH) seems to be associated with improved prognosis in severe disease²⁰. Acute coronary syndrome, acute limb ischemia, abdominal and thoracic aortic thrombosis, mesenteric ischemia, venous thromboembolism, acute cerebrovascular accident and disseminated intravascular coagulation are also complications associated with SARS-CoV-2²¹.

Furthermore, a study evaluated 37 patients with a confirmed COVID-19 diagnosis by RT-PCR admitted to a hospital in Turkey due to chest pain with ST-segment elevation acute myocardial infarction. Coagulation tests were collected and an echocardiography was performed to evaluate cardiac wall abnormalities. All patients had high levels of myocardial enzyme release. Coronary artery disorders requiring revascularization were detected in 25 patients, and percutaneous coronary interventions (PCI) were performed in patients with culprit lesions. Success rate of PCI was 87.5% and PCI failed in four patients, who had a coronary artery bypass surgery. In addition, 30% of patients required re-intervention owing to early stent thrombosis, and 33.3% died after PCI²². Following the same reasoning, a cohort study conducted in Spain including 16 hospitals found that the main comorbidity in patients who required hospitalization due to COVID-19 was arterial hypertension, and of those who had a thromboembolic event, 76.6% had venous thromboembolism²³.

DISCUSSION

Blood stasis, endothelial damage and hypercoagulable state, together, are closely related to the occurrence of thromboembolic events²⁴ and the hyper-inflammatory

state is an important risk factor for its occurrence²⁵. Thus, in the case of an unbalanced response to SARS-CoV-2 infection, an exacerbated immune response to the viral infection can be produced, leading to a hyper-inflammatory state. This response to infection is inadequate in individuals with endothelial dysfunction and chronic inflammatory processes and can further increase the risk of severe COVID-19 and thrombosis in diabetic patients and patients with cardiovascular diseases^{26,27}.

In SARS-CoV-2 infection, atherosclerotic progression is associated with a specific immune response that generates Th1-type cytokines, such as IL-12 and interferon gamma^{28,29}. IL-12 is produced by monocytes in response to increased LDH due to cell destruction, and IFN-gamma is a pro-atherogenic cytokine responsible for activating macrophages and releasing more pro-inflammatory cytokines such as IL-2, IL-1, IL-6, IL-7, IL-12, IL-18. All these interactions play a critical role in the pro-thrombotic effect of viral infections³⁰. Platelets will act together with white blood cells to fight the infection³¹. This deregulation of homeostatic mechanisms generates an over activation of coagulation cascade and platelets and leads to thrombocytopenia and elevated D-dimer³². The whole process of infection control leads to an increase in cytokines and prothrombotic factors, further favoring hypercoagulability³³.

It is also known that the inflammatory and coagulation processes are related to levels of platelet activation, fibrin formation, and physiological anticoagulant pathways, which confirms the theory of simultaneous modulation of coagulation and inflammation, such as therapies aimed at tissue factor or at physiological regulatory pathways, such as the protein C system³⁴.

The high incidence of thromboembolic events within 24 hours of admission of patients with COVID-19 confirms the high inflammatory burden of the disease, and the high levels of D-dimers in patients who died also confirms cardiac damage. In addition, patients who develop myocardial injury after six months of the acute phase of COVID-19 may have significant diastolic dysfunction³⁵. This may be associated with the virulence mechanism; according to Burak Erdinc et al., in an analysis of several studies including a total of more than 8000 patients, lymphopenia, exhaustion of CD8+ T cells, and the increase in acute thrombotic events seem to be related to the cytokine storm caused by the SARS-CoV-2 virus³⁶. Thus, patients with SARS-CoV-2 display signs of myocardial injury, which is more evident in patients with severe disease³⁷. Predictors of a fatal outcome in COVID-9 include age, the presence of underlying diseases, the presence of secondary infection and elevated inflammatory indicators in the blood, as seen in a study with 150 patients infected with SARS-CoV-2 in China³⁸.

However, even though it is the gold standard treatment for patients with STEMI, percutaneous

coronary intervention has been replaced by fibrinolytic therapy in the pandemic caused by SARS-CoV-2³⁹, either due to the distance from medical centers, the increase in the mortality of the medical staff, or the redirection of hospital flows to care for patients with COVID-19^{40,41}. Therefore, this demonstrates the change of flows in the cardiology sector and the consequent increase in the number of complications and mortality of these patients, especially of those with higher morbidity.

As for the revascularization procedure, primary angioplasty is the preferred technique for harm reduction, better outcomes, and preservation of ventricular function in acute ischemic heart disease¹¹. Despite the benefits of fibrinolytic therapy, the first choice treatment in patients with acute myocardial infarction is primary angioplasty. However, if fibrinolytic therapy has already been performed, the patient should be transferred to a specialized center so that the rescue angioplasty can be performed if there is no restoration of epicardial coronary blood flow⁴², which may occur in 50 to 70% of patients treated with streptokinase and 20 to 50% of those treated with fibrinolytic drugs⁴³. The main causes of failure are intimal tears or thrombus resistance⁴². However, the use of

pharmacological prophylaxis in patients with COVID-19 admitted to the ICU is strongly recommended, even in the absence of randomized evidence⁶, given that the incidence of thromboembolic events is extremely high in patients with STEMI, according to the largest case series to date¹³.

Therefore, STEMI may represent the first clinical manifestation of COVID-19 and approximately 40% of patients with COVID-19 and STEMI have a culprit lesion not identifiable by coronary angiography. In this context, a dedicated diagnostic pathway should be delineated for these patients, with the objective of minimizing procedural risks and healthcare providers' risk of infection⁴⁴. Furthermore, a study with 900 patients treated with percutaneous coronary intervention for STEMI found that large thrombus (≥ 2 times the vessel diameter) is a significant independent predictor for mortality⁴⁵.

CONCLUSION

There is a higher prevalence of coronary thrombosis in patients with ST-segment elevation acute myocardial infarction infected with SARS-CoV-2 after undergoing primary angioplasty.

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