

ST Elevation myocardial infarction with split LAD and bilateral acute limb ischemia in young patient with COVID-19

Abstract

We report a 31-year-old overweight patient without any other cardiovascular risk factors, who presented with a 4-hour ST-segment elevation myocardial infarction (STEMI) with chest pain. An urgent coronary angiogram observed a single coronary artery anomaly (CAA) with a duplicated left anterior descending artery (LAD) with a thrombotic occlusion of a large diagonal branch. All other coronary arteries, including the split LAD, appeared angiographically normal. A primary percutaneous coronary intervention (PCI) was performed on the diagonal branch and 3 stents were deployed. Following the procedure, TIMI 3 flow was restored, and the patient experienced immediate relief from chest pain. No further electrocardiogram (ECG) changes were observed, and a transthoracic echocardiogram (TTE) after PCI revealed only mild left ventricular dysfunction. Twenty-four hours after admission, the patient developed a fever and was diagnosed with COVID-19. On day 4 of hospitalization, he developed bilateral thrombotic occlusion of both superficial femoral arteries, resulting in severe acute limb ischemia. This was treated aggressively with a quintuple antithrombotic regimen, initially comprising prasugrel, aspirin, and enoxaparin, followed by 24-hour intra-arterial local thrombolysis with r-tPA in addition to unfractionated heparin. Subsequently, manual thrombus aspiration and the placement of self-expanding stents in both femoral and popliteal arteries allowed partial restoration of distal flow.

The patient later developed acute renal failure, shock, and multiorgan failure, ultimately succumbing to irreversible shock 13 days after hospital admission. COVID-19 can lead to multiple arterial thrombotic events, even in young patients without significant cardiovascular risk factors. In addition, we present a case of split LAD, a very rare congenital anomaly.

Keywords: Acute myocardial infarction, acute coronary syndrome, COVID-19, acute limb ischemia, peripheral arterial thrombosis,

Volume 17 Issue 2 - 2025

Mieres Juan MD,^{1,2} Rodríguez-Granillo Matías MD,¹ Fernández-Pereira Carlos MD, PhD, FACC,^{1,2} Fontana Lucia MD,¹ Lapadula Pablo MD,³ Curotto Valeria MD,¹ Sandra Swieszkowski,¹ Pérez de la Hoz Ricardo MD,¹ Rodríguez Alfredo E MD, PhD, FACC^{1,2}

¹Intensive Cardiac Unit, Otamendi Hospital, Buenos Aires School of Medicine, Argentina

²Interventional Cardiology Department, Otamendi Hospital, Argentina

³Infectious Disease Department, Otamendi Hospital, Argentina

Correspondence: Mieres Juan, Intensive Cardiac Unit, Otamendi Hospital, Buenos Aires School of Medicine, Azcuénaga 870, Capital Federal (1115), Buenos Aires Argentina

Received: February 24, 2025 | **Published:** March 14, 2025

Abbreviations: COVID-19, Coronavirus disease 2019; DAPT, Dual antiplatelet therapy; ECG, electrocardiogram; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; STEMI, ST-segment elevation myocardial infarction; ULN, Upper limit of normal value; PCI, Percutaneous Coronary Interventions; TTE, Transthoracic Echocardiogram; CAA, Coronary Artery Anomaly

Introduction

In patients with COVID-19, the prevalence of coronary heart disease and cardiovascular pathology varies, with reported rates ranging from 4.2% to 25% in most Chinese series. The mechanisms underlying cardiovascular injury remain incompletely understood.^{1,2} Several contributing factors have been identified, including direct endothelial injury,³ hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction with thrombosis, coagulation abnormalities,⁴ systemic inflammation, and cytokine storm.

Elevated fibrinogen and D-dimer levels have been observed, resembling disseminated intravascular coagulation but with a predominance of thrombotic phenomena.⁵

As a result, hospitalized COVID-19 patients face an increased risk of acute arterial thrombosis affecting various vascular territories, including coronary, cerebral, and peripheral circulation.^{6,7} This report presents an unusual case of STEMI in a young patient without major vascular risk factors, who subsequently developed bilateral acute

lower limb ischemia due to femoral and popliteal artery thrombosis during hospitalization. Patient with a rare coronary anomaly which is the duplication of the left anterior descending artery.

Case presentation

A 31-year-old man with no previous history of coronary or vascular disease was admitted to the catheterization laboratory, for STEMI. He reported experiencing chest pain for the past four hours. Additionally, he had experienced bilateral leg pain two weeks earlier, which was treated with 600 mg of ibuprofen for two days following an online medical consultation. No fever episodes were reported before admission. Upon arrival, the patient exhibited no signs of shock, heart failure, pneumonia, or fever. His physical examination was unremarkable, and all peripheral pulses were present.

The patient led a normal lifestyle, with obesity (body mass index: 35 kg/m²) being the only reported cardiovascular risk factor. There was no family history of coronary or vascular disease, and both parents were alive at the time of admission. He was not taking any medications or drugs, except for ibuprofen in the days preceding admission. The initial ECG showed sinus rhythm with a lateral STEMI (D1, avL, and V6) and ST segment depression in leads D3 and avF (Figure 1).

The time from symptom onset to hospital admission was four hours, and door-to-catheterization laboratory time was 30 minutes. Coronary angiography via the radial approach revealed a CAA with a duplicated LAD and a large first septal artery arising from

the right coronary sinus, adjacent to the origin of the right coronary artery (RCA) (Figure 2). In a very high posterior portion of the left ostium, the left main coronary artery (LMCA) with the left circumflex artery (LCX) and a duplicated medium-sized left anterior descending artery with a large diagonal branch is located. This second LAD was identified by the presence of septal branches arising from it (Figure 3). While the RCA, LMCA, LAD, and LCX were angiographically normal, the proximal diagonal branch exhibited complete thrombotic occlusion with Thrombolysis in Myocardial Infarction (TIMI) grade 0 flow, identifying it as the culprit lesion in STEMI.



Figure 1 ECG at admission and immediately post PCI.

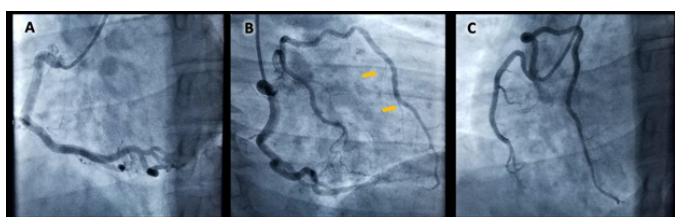


Figure 2 Coronary angiogram CAA of duplicate LAD.

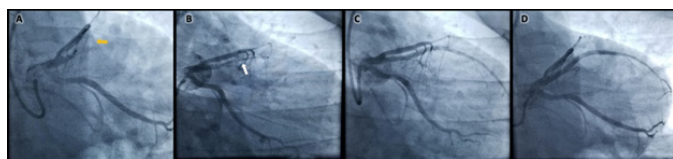


Figure 3 PCI and stents implantation of diagonal branch.

Following the deployment of three stents in the diagonal branch, TIMI 3 flow was restored, and a widely patent diagonal branch was observed. The optimal PCI outcome resulted in complete resolution of chest pain (Figure 3). A transthoracic echocardiogram (TTE) revealed mild left ventricular dysfunction with lateral hypokinesia. Dual antiplatelet therapy (DAPT) with aspirin and prasugrel was initiated at the time of PCI, along with enoxaparin (200 mg/day).

Laboratory tests showed elevated troponin (27,000 ng/L, ULN < 14 ng/L), CK-MB (300 ng/mL, ULN < 5), and CPK (2,425 IU/L, ULN < 171 IU/L), while creatinine levels (0.7 mg/dL), blood cell counts and coagulation test were normal.

Twenty-four hours post-PCI, the patient developed a fever. Given the severe presentation in a low-risk young adult, a complete virologic screening, including COVID-19 testing, was performed, yielding positive results. Inflammatory markers were elevated, including white blood cell count (18.9 G/L), lymphopenia (8.9%), ferritin (591 ng/mL, ULN < 336), D-dimer (1.25 µg/mL, ULN < 0.5), and C-reactive protein (119.7 mg/L, ULN < 5). No symptoms or signs of congestive heart failure were noted and the patient remained free of chest pain or dyspnea with no new ECG changes.

On day 4 of hospitalization, the patient developed pain and signs of hypoperfusion in both legs. Peripheral pulses below the knees were absent, and pallor was noted. Peripheral Doppler ultrasound showed monophasic flow in both legs. Consequently, the enoxaparin dose was increased to 240 mg/day.

On day 6, given the worsening distal involvement of both legs, a peripheral angiogram was performed, revealing complete thrombotic occlusion of both femoral arteries at Hunter's canal. A large thrombus burden was observed in the femoral and popliteal arteries, with severely diminished antegrade and collateral flow (Figure 4). Local intra-arterial r-tPA infusion was initiated and maintained for 24 hours, while enoxaparin was replaced with intravenous unfractionated heparin.

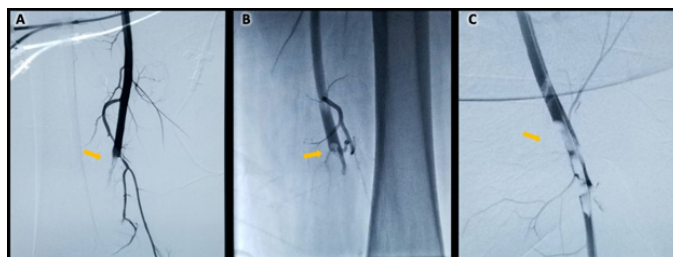


Figure 4 Bilateral femoral and popliteal arteries occlusion. Large thrombus burden.

Due to persistent poor peripheral perfusion, including cyanosis of the feet, percutaneous peripheral angioplasty was attempted 24 hours after angiography on both femoral and popliteal arteries. The procedure included manual aspiration thrombectomy and the deployment of self-expanding stents, leading to partial restoration of flow in the right posterior tibial artery (Figure 5) and left peroneal artery (Figure 6).



Figure 5 Peripheral angioplasty and stents implantation of right femoral and popliteal arteries.

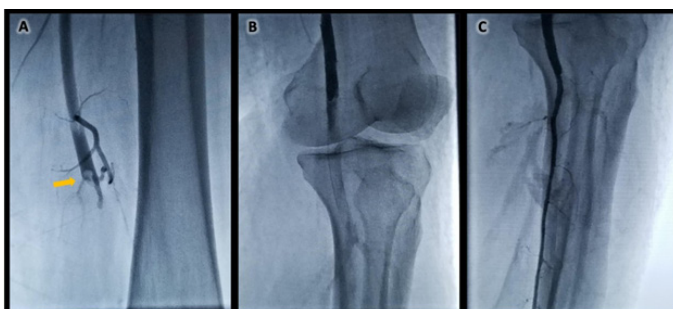


Figure 6 Peripheral angioplasty and stents implantation of left femoral and popliteal arteries.

Following peripheral angioplasty, the patient developed severe acute renal failure, with creatinine levels rising to 6 mg/dL (ULN < 1.2 mg/dL), necessitating dialysis and hemodynamic support with norepinephrine. Despite these interventions, the patient experienced metabolic acidosis and distributive shock, requiring mechanical ventilation. Intravenous hydrocortisone (300 mg/day) was administered, and vasopressin was added to maintain hemodynamic stability.

Despite aggressive therapeutic measures, the patient dies of multiorgan failure 13 days after hospital admission (Figure 7).

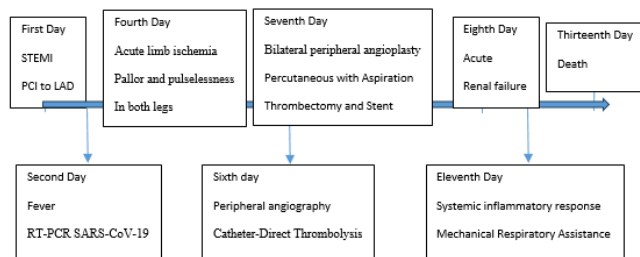


Figure 7 In hospital outcome and procedures since hospital admission.

Discussion

Myocardial infarction (MI) in young adults has been increasingly observed in recent years, typically affecting individuals with traditional cardiovascular risk factors, such as smoking, elevated LDL cholesterol, diabetes mellitus, significant family history of coronary artery disease, or drug abuse.⁸ In the case presented here, obesity was the only identifiable risk factor for coronary artery disease (CAD). The patient's angiographic findings revealed single-vessel disease with thrombotic occlusion in a large diagonal branch, while all other coronary arteries appeared angiographically normal. Given the absence of severe comorbidities and the presence of normal angiographic findings in the RCA, LMCA, LAD, and LCX, it is likely that this thrombotic event was triggered by COVID-19. The virus is now well established as a prothrombotic condition that promotes an exaggerated local immune response, increasing the likelihood of plaque erosion or rupture.^{9,10}

The patient had no prior symptoms suggestive of COVID-19, and the infection was only diagnosed 24 hours after PCI. Of particular concern, the patient developed severe acute limb ischemia, with complete thrombotic occlusion of both superficial femoral arteries despite being on dual antiplatelet therapy (DAPT) with aspirin and prasugrel,¹¹ as well as enoxaparin at 200 mg/day. The absence of atrial fibrillation, only mild left ventricular dysfunction, and no evidence of mural thrombus on TTE strongly suggested that the peripheral thrombotic events were secondary to the hyperinflammatory and prothrombotic state induced by COVID-19.

A previously published case report¹² described ischemia in two vascular territories similar to ours. However, a significant distinction is that in our patient, angiographic imaging revealed no signs of atherosclerotic disease, whereas the previously reported case demonstrated left common iliac artery occlusion on CT angiography with calcified plaques as evidence of atherosclerosis.

Inflammatory markers were markedly elevated in our patient, with high levels of D-dimer, ferritin, and C-reactive protein, all of which have been associated with coagulation activation and consumption in severe COVID-19 cases.¹³ Following the episode of limb ischemia, enoxaparin was escalated to therapeutic doses alongside DAPT. Interestingly, as reported in the literature, this patient exhibited no symptoms of COVID-19 before STEMI, reinforcing the notion that ischemic events may be the first clinical manifestation of COVID-19, even in asymptomatic individuals with low cardiovascular risk. Our case contributes to the growing evidence on the role of multiple arterial thromboses in COVID-19, particularly in the simultaneous involvement of both peripheral and coronary arteries, which has been rarely reported.¹⁴

A unique aspect of this case is the presence of a rare coronary anomaly. CAA occurs in 0.2–1.2% of the population, with the most common being right coronary artery anomalies. The left coronary artery arising from the right coronary sinus occurs in approximately 2.3% of cases. Reports of duplicated left anterior descending arteries, as seen in this case, are exceedingly rare.¹⁵

Limitations

Due to the severity of complications in this patient, a 3D CT angiography could not be performed to further characterize the coronary anomaly. However, detailed imaging of the anomaly was not the primary focus of this report.

Conclusion

The prothrombotic and inflammatory state induced by COVID-19 increases the risk of arterial thrombosis, even in young patients with STEMI and minimal or no traditional cardiovascular risk factors. Given the increased thrombotic burden observed in these patients, therapeutic anticoagulation should be considered alongside dual antiplatelet therapy. Additionally, early and aggressive reperfusion strategies may be crucial in preventing fatal complications. Coronary anomalies are rare and require a high level of suspicion for accurate identification and management.

References

- Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *JAMA*. 2020;323(11):1061–1069.
- Chen T, Wu D, Chen H, et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. *BMJ*. 2020;368:m1091.
- Teuwen LA, Geldhof V, Pasut A, et al. COVID-19: the vasculature unleashed. *Nat Rev Immunol*. 2020;20(7):389–391.
- Panigada M, Bottino N, Tagliabue P, et al. Hypercoagulability of COVID-19 patients in intensive care unit: a report of thromboelastography findings and other parameters of hemostasis. *J Thromb Haemost*. 2020;18(7):1738–1742.
- Klok FA, Kruip MJHA, van der Meer NJM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. *Thromb Res*. 2020;191:145–147.
- Bilaloglu S, Aphinyanaphongs Y, Jones S, et al. Thrombosis in hospitalized patients with COVID-19 in a New York City health system. *JAMA*. 2020;324(8):799–801.
- Rey JR, Caro-Codón J, Poveda Pineda D, et al. Arterial thrombotic complications in hospitalized patients with COVID-19. *Rev Esp Cardiol (Engl Ed)*. 2020;S1885–5857(20)30205–X.
- Collet JP, Zeitouni M, Procopi N, et al. Long-term evolution of premature coronary artery disease. *J Am Coll Cardiol*. 2019;74(15):1868–1878.
- Bangalore S, Sharma A, Slotwimer A, et al. ST-segment elevation in patients with Covid-19—a case series. *N Engl J Med*. 2020;382(25):2478–2480.
- Rodríguez AE, Rodríguez-Granillo AM, Ascarrunz SD, et al. Did prasugrel and ticagrelor offer the same benefit in patients with acute coronary syndromes after percutaneous coronary interventions compared to clopidogrel? Insights from randomized clinical trials, registries and meta-analysis. *Curr Pharm Des*. 2018;24(4):465–477.
- Aissaoui O, Husam S, Mounir A, et al. Concomitant acute limb ischemia and myocardial infarction: another challenge of COVID-19's hypercoagulability. *Am J Cardiovasc Dis*. 2022;12(3):149–152.

12. Mondal R, Lahiri D, Deb S, et al. COVID-19: are we dealing with a multisystem vasculopathy in disguise of a viral infection? *J Thromb Thrombolysis*. 2020;1–13.
13. Kau P, Posimreaddy S, Singh B, et al. COVID-19 presenting as acute limb ischaemia. *Eur J Case Rep Intern Med*. 2020;7(6):001724.
14. Schanzenbächer P, Strotmann J. Images in cardiology: bilateral origin of a split left anterior descending coronary artery combined with atypical branching of the left circumflex coronary artery. *Heart*. 2002;88(1):70.