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Case Report

Transcatheter aspiration of a thrombus and percutaneous transluminal coronary recanalization for ST-segment elevation myocardial infarction related to coronavirus disease 2019



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ABSTRACT

Although the novel coronavirus disease 2019 (COVID-19) causes severe viral pneumonia, it has also been reported, in some cases, to co-exist with ST-segment elevation myocardial infarction. Here, we describe the case of a patient with COVID-19 and coronary risk factors for hypertension, including smoking and obesity, who developed acute myocardial infarction due to primary coronary artery thrombosis and was treated with transcatheter thrombus aspiration and percutaneous transluminal coronary recanalization (PTCR) with intracoronary urokinase administration. A large volume of thrombus was collected and thrombolysis in myocardial infarction flow grade 3 was obtained after the procedures. PTCR with or without transcatheter thrombus aspiration may be a useful treatment option.

<Learning objective: ST-segment elevation myocardial infarction is a critical complication in patients with novel coronavirus disease 2019. Patients need emergent recanalization to prevent development of fatal cardiac events.>

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Introduction

The novel coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has sparked a pandemic in 2020 and 2021, and is widely known not only for severe acute viral pneumonia and inflammatory respiratory distress syndrome but also for arteriovenous thrombosis. Among them, cardiovascular disease is associated with a higher risk of mortality and needs to be addressed swiftly in addition to the usual COVID-19 treatment [1]. We report a case of ST-segment elevation myocardial infarction (STEMI) in a patient with COVID-19-related pneumonia, treated with percutaneous transluminal coronary recanalization (PTCR).

Case report

The publication of this case report was approved by the ethical committee of our hospital, and written informed consent was obtained from the patient.

A 52-year-old man with the following coronary risk factors of hypertension, a 20-pack-year smoking history and obesity (body mass index: 41), visited a primary care physician 6 days previously with a fever of 37–38°C and cough. The public health center recommended hospitalization because the SARS-CoV-2 polymerase chain reaction test result was positive. On admission, the oxygen saturation (SpO₂) level was 93% in room air. Chest X-ray and computed tomography (CT) showed scattered ground-glass opacities and hyperdensities, respectively, in both lung fields. The patient was diagnosed with COVID-19 pneumonia (severe illness), and dexamethasone 6 mg/daily was started on the same day. After the start of treatment, oxygen administration through nasal cannula was gradually tapered down from 2 L/min to 0.5 L/min.

At 11:20 on the sixth day of hospitalization, the patient developed sudden chest pain. An electrocardiogram (ECG) was per-

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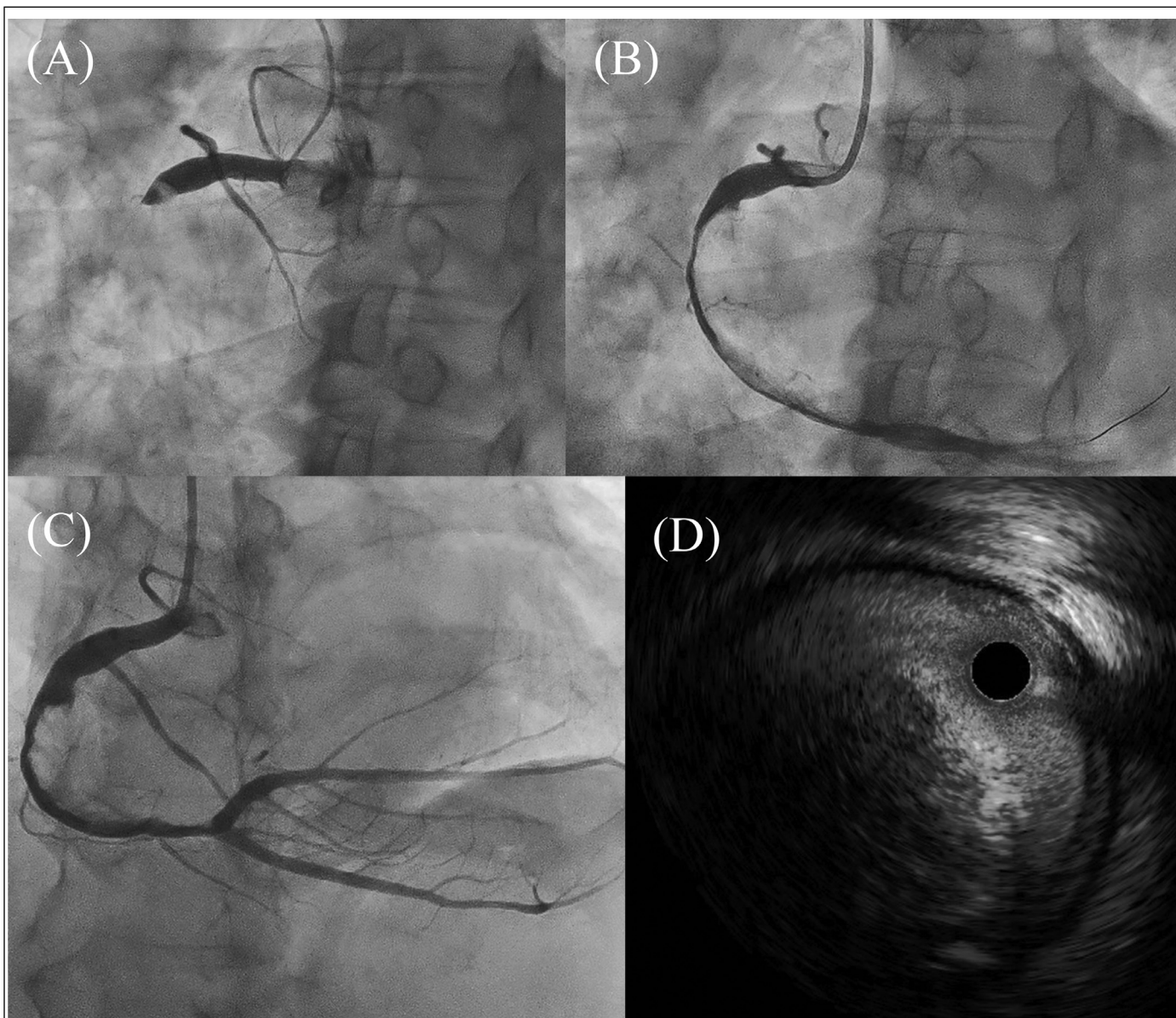


Fig. 1. Coronary angiography and coronary intervention for ST-segment elevation myocardial infarction. (A) Coronary angiography shows total occlusion of the right coronary artery with thrombus. (B) After transcatheter thrombus aspiration, reperfusion was established. (C) After intracoronary administration of urokinase, thrombolysis in myocardial infarction flow grade 3 was obtained. (D) Intravascular ultrasound showed massive thrombus.

formed, which showed a complete atrioventricular block and ST-T elevation in II, III, aVF, and V3-V6 leads, leading to the diagnosis of STEMI. Transthoracic echocardiography (TTE) showed hypokinetic wall motion in the posterior and inferior walls [left ventricular ejection fraction (LVEF), 45%]. At 13:05, an emergency coronary angiography (CAG) was performed with an infection prevention strategy using N95 masks, goggles, sterile gowns, and gloves during the procedures in the catheterization laboratory, which revealed thrombotic occlusion of the proximal right coronary artery (RCA). We subsequently performed an ad hoc percutaneous coronary intervention (PCI). A guidewire was passed easily, and the thrombus was aspirated using a thrombus aspiration catheter. A large volume of mixed thrombus, mainly white thrombus, was collected, and thrombolysis in myocardial infarction (TIMI) flow grade 2 was obtained (onset to reperfusion time: 148 min). After reperfusion, the chest pain resolved. Intravascular ultrasound (IVUS) revealed a massive thrombus in the mid to proximal RCA. To reduce the

thrombus volume, a total of 960,000 units of urokinase were administered into the coronary artery via a microcatheter. Although the thrombus remained soon after PTCR, we completed the procedures without balloon dilation or stent implantation because the vessel diameters were large, and TIMI flow grade 3 was obtained (Fig. 1). Since no atrial fibrillation was documented on ECG monitoring during hospitalization and no intracardiac thrombus was observed on TTE before and after emergent CAG, we suspected STEMI due to primary coronary thrombosis.

After PTCR, 81 mg of oral aspirin orally and continuous infusion of unfractionated heparin in addition to standard medical therapy for STEMI were also administered. On the seventh day of admission, an oral anticoagulant (warfarin, 5 mg per day) was administered alongside the continuous intravenous infusion of unfractionated heparin, and heparin was discontinued after confirming that prothrombin time-international normalized ratio was >2.0 . Two weeks later, follow-up CAG showed coronary wall irregularities

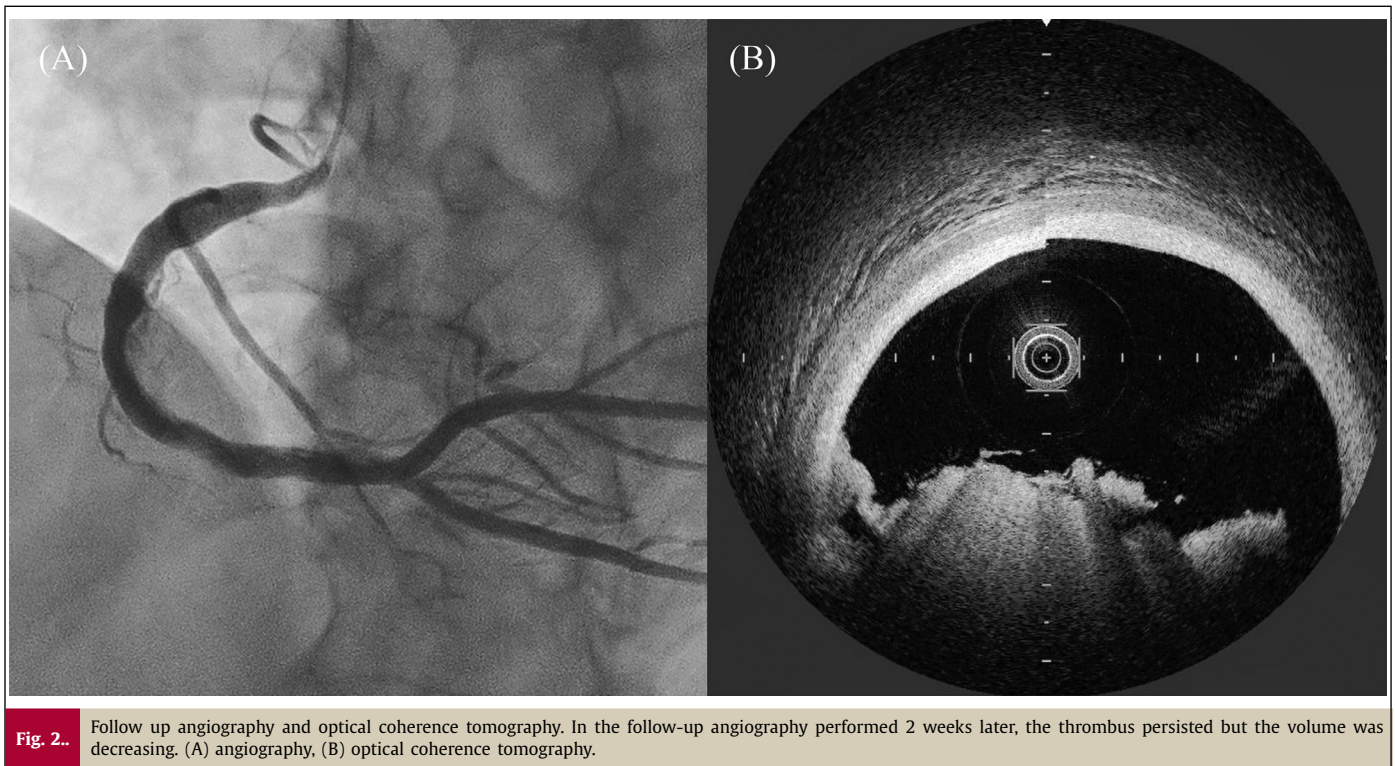


Fig. 2. Follow up angiography and optical coherence tomography. In the follow-up angiography performed 2 weeks later, the thrombus persisted but the volume was decreasing. (A) angiography, (B) optical coherence tomography.

that may have been residual thrombus; however, the volume of the thrombus was greatly reduced. We also performed optical coherence tomography (OCT) to evaluate the condition of the coronary artery wall. There were no ruptured plaques or calcified nodules within the observation range except for mild intimal thickening and residual thrombus; however, we could not observe the entire circumference because the vessel diameter was too large (Fig. 2). We assessed the infarct size by TTE at 10 days after the procedure, which revealed mild hypokinesia of the inferior and posterior walls (LVEF, 55.6%). The peak levels of creatinine kinase (CK) and CK-MB were 12,900 U/L and 762 U/L.

After cardiac rehabilitation, the patient was discharged with a prescription for warfarin and aspirin on the 17th day after admission when the isolation period, as set by the Japanese government, ended.

Five months later, CT coronary angiography (CTCA) was performed, which showed no thrombus in the RCA and no significant coronary stenosis or calcified lesion (Fig. 3). Based on these findings, we discontinued the oral anticoagulation therapy.

Discussion

The causes of STEMI include thrombosis secondary to plaque rupture, coronary spasm, thromboembolism, and thrombosis associated with intimal erosion and calcified nodules. One-third of STEMI cases in patients with COVID-19 do not have any obvious stenosis on CAG, and its mechanisms are thought to include cases of angiographically unidentified plaque rupture or intimal erosion, spasm, microthrombus, and hypoxia [2].

Recent studies have shown that the level of serum angiotensin II increases in proportion to the viral load of SARS-CoV-2 and that angiotensin II and the renin-angiotensin-aldosterone system enhance the systemic inflammatory response and have a significant effect on platelet aggregation and the coagulation-fibrinolysis system in patients with COVID-19 [3]. Neutrophil extracellular traps (NETs) reportedly play an important role as a cause of STEMI in

patients with COVID-19 and that NETs and thrombi composed of fibrin and polymorphonuclear cells were detected in this group of patients [4]. Elevated serum levels of the highly specific markers of NETs such as cell-free DNA, MPO-DNA complexes, and citrullinated histone H3 may support a diagnosis of coronary thrombosis associated with COVID-19 [5].

In our case, first, we were unable to detect plaque rupture, calcified nodules, or organic stenosis in the culprit vessel by IVUS and OCT; however, the observational range was limited because of the large vessel diameter and the backscattering projections of the thrombus. Therefore, we performed CTCA after 6 months of anticoagulation therapy, which revealed no organic coronary lesion or residual thrombus. Second, we also performed an acetylcholine provocation test (50 µg in the RCA) 7 months later, and coronary spasm was not induced. In addition, the patient had been using calcium channel antagonists for hypertension and did not have a prior episode of chest pain at rest. We considered the possibility of STEMI associated with coronary spasm to be negative. Third, during hospitalization, the serological predisposition to abnormalities of the coagulation-fibrinolysis system was investigated. Lupus anticoagulant and antiphospholipid antibodies (anticardiolipin antibody and anti-β₂-glycoprotein I antibody) were negative, and homocysteine, protein C activity, and protein S activity were all within normal limits. Fourth, although the patient was on oxygen, administered via nasal cannula at 0.5 L/min at the time of STEMI, the patient was not hypoxic with an SpO₂ level of 98%. Fifth, atrial fibrillation in ECG monitoring during hospitalization and intracardiac thrombus on TTE before using heparin or anticoagulant were not observed. Based on these points, the most likely cause of STEMI, in this case, was primary coronary thrombosis associated with SARS-CoV-2 infection. However, one limitation of this study was that we could not perform pathological examinations for NETs because they are not common in general medical practice in Japan.

The treatment of STEMI includes primary PCI and transvenous thrombolysis in global guidelines. During the COVID-19 pandemic, primary PCI was recommended as the standard treatment for hos-

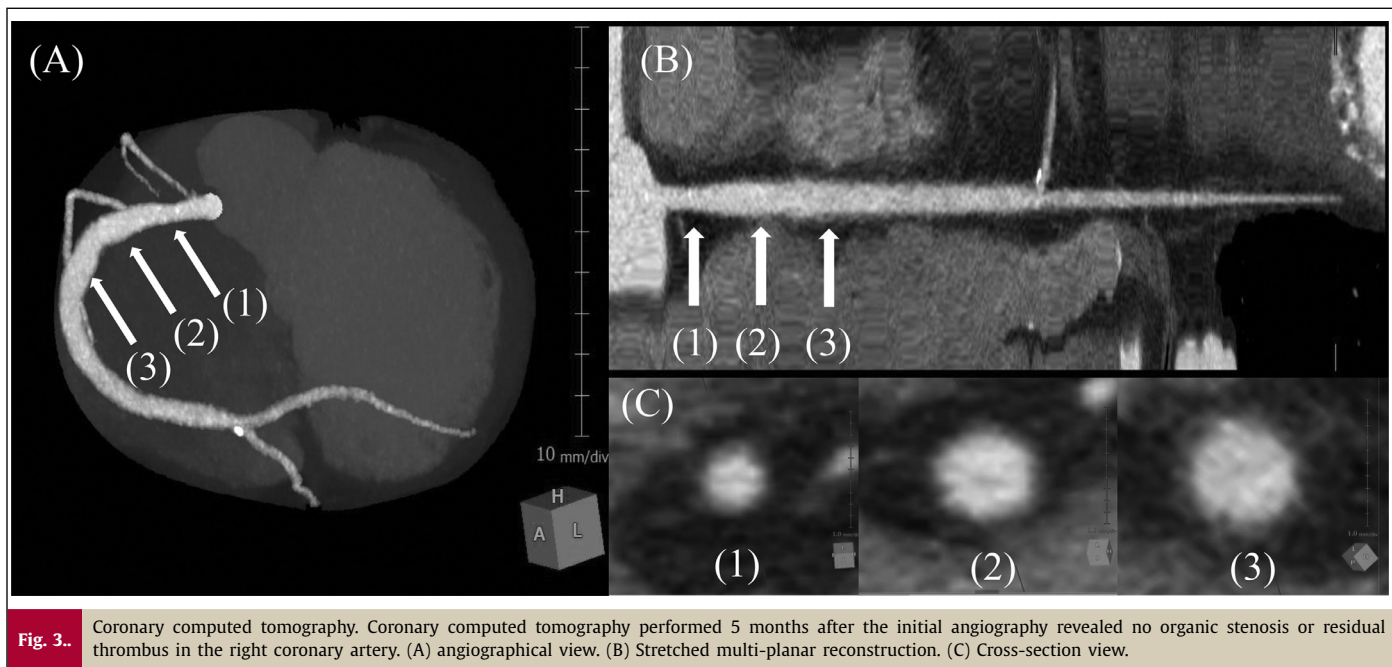


Fig. 3..

Coronary computed tomography. Coronary computed tomography performed 5 months after the initial angiography revealed no organic stenosis or residual thrombus in the right coronary artery. (A) angiographical view. (B) Stretched multi-planar reconstruction. (C) Cross-section view.

pitals with the ability to perform PCI [6]. With the spread of COVID-19 in Japan, it has been reported that the rate of primary PCI for STEMI was maintained at a high level in Japan [7]. The reason for this is not clear, but it may be because the number of patients with COVID-19 was relatively low. Conversely, as primary PCI has become more widespread and its outcomes have improved, PTCR has become less common in recent years, except in limited cases. Although primary PCI with stents is undoubtedly the gold standard, a stent-less strategy is also used for stenosis of dilated vessels and thrombosed lesion. PTCR have been reported as a method to reduce the volume of thrombus, especially in patients with STEMI who also have a large thrombus burden [8]. The previous study demonstrated that a higher burden of thrombus was observed in patients with acute myocardial infarction combined with COVID-19 than in patients without COVID-19 [9]. There has also been a reported case where it was difficult to treat a patient with primary PCI due to massive thrombosis [10]. The advantage of PTCR over transvenous thrombolysis is that it allows selective and direct administration of anticoagulant agents after identification of the lesion, and recanalization can be confirmed soon after the procedure. PTCR can be performed before conventional PCI as an extension of the standard procedure and can be switched to balloon dilatation and stent implantation, as required. In patients with COVID-19, we consider that the etiology of STEMI may be primary thrombosis due to immuno-thrombogenicity. In these cases, PTCR with or without transcatheter thrombus aspiration may be a useful treatment option.

In patients with COVID-19, the possibility of arteriovenous thrombosis, especially STEMI, resulting in a fatal cardiac event should always be considered. Therefore, PTCR may be a useful treatment option.

Declaration of Competing Interest

The authors declare that there is no conflict of interest.

Acknowledgments

None

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