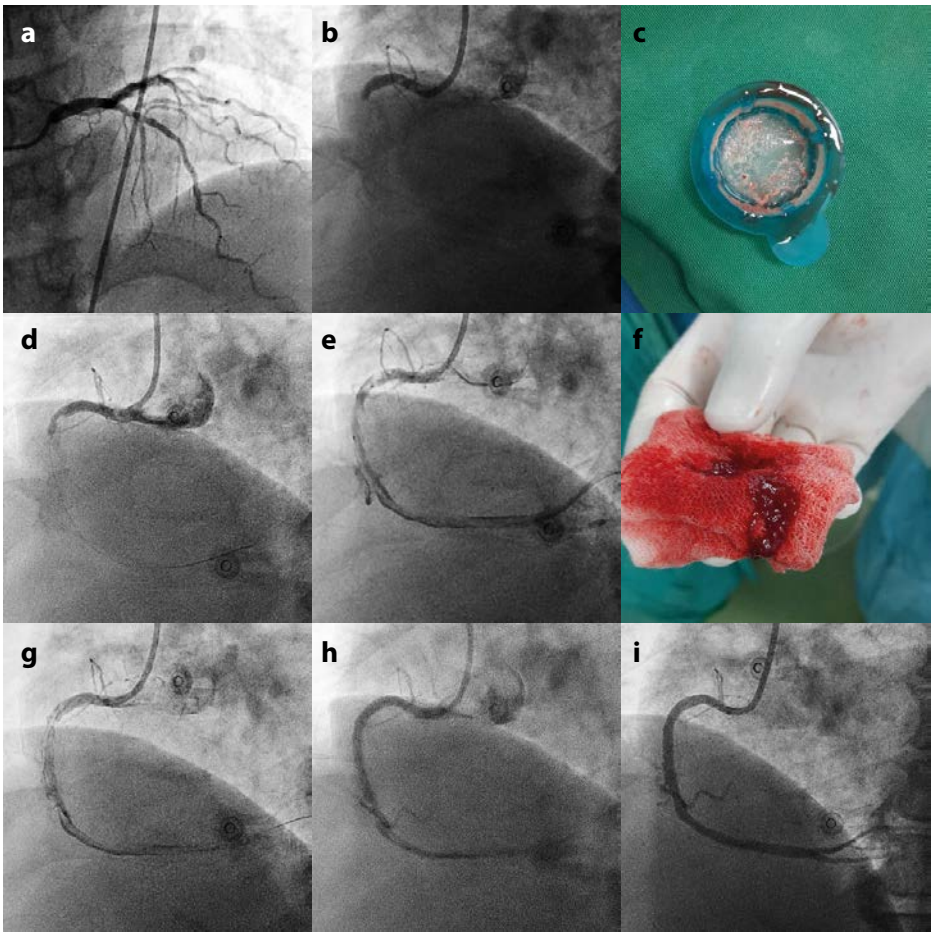
Fig 2. *Coronary Angiography. (a) LMCA, LAD, and LCx appear normal. (b) Total occlusion of the proximal part of the RCA with TIMI flow grade 0. (c) MAT succeeded in obtaining a red and white thrombus. (d) Evaluation after the first MAT. (e) Evaluation after the first balloon angioplasty. (f) There is a clot blocking the aspiration catheter after the second MAT. (g) Evaluation after intracoronary thrombolysis and balloon angioplasty. (h) Post-stent evaluation proximal to mid-RCA. (i) Angiography evaluation 4 days after primary PCI*



a 0.014" high-torque hydrophilic guidewire (Whisper MS, Abbot, Minneapolis, USA) and successfully penetrated the lesion distal to the RCA; however, antegrade flow was obtained on angiography, with grade 0 TIMI flow and grade 5 TIMI thrombus quantification.

MAT was performed using a 6-Fr aspiration catheter (Capturer, iVascular, Barcelona, Italy), and a red and white thrombus was obtained (Fig. 2c); however, there was no antegrade flow from the proximal RCA (Fig. 2d). To compress the lesion, a 1.5 × 15 mm semi-compliant balloon (Sapphire II, OrbusNeich, Netherlands) was inflated to 16 atmospheres (atm) proximal to distal to the RCA, followed by administration of 0.5 mg atropine sulfate as the patient developed bradycardia and hypotension. The procedure was continued by dilating the lesion using a 2.25 × 12 mm semi-compliant balloon (Sapphire II, OrbusNeich, Netherlands) to 15 atm midway to the proximal RCA. Angiography after balloon angioplasty showed an increase in coronary blood flow from TIMI flow grade 0 to 2 with a diffuse thrombus along the RCA (Fig. 2e). A second MAT was performed in an attempt to remove the remaining thrombus, only to find the aspiration catheter blocked by a clot (Fig. 2f).

Due to a diffuse thrombus in the RCA, we decided to perform intracoronary thrombolysis. Streptokinase 250,000 units (Fibrion, Lyocontract, Lisenburg, Germany) diluted in 16.7 mL normal saline was administered slowly over 5 minutes using a 4.0 6-Fr Judkin Right guiding catheter (Launcher, Medtronic, Minneapolis, USA), followed by dilatation with a 3.0 × 20 mm semi-compliant balloon (Sapphire II, OrbusNeich, Hoevelaken, Netherlands) up to 15 atm for manipulating the partially lysed thrombus; however, angiography showed disappointing results. We decided to continue intracoronary thrombolysis using the same amount of streptokinase, but there was almost no difference in antegrade flow in the distal portion of the RCA. After repeated intracoronary thrombolysis five times with a total of 1,500,000 units of streptokinase over 30 minutes, an antegrade flow improvement to TIMI flow grade 3 was seen with the thrombus still in place (Fig. 2g). The proximal to the middle of the RCA was then stented using a 3.5 × 36 mm sirolimus drug-eluting stent (DES) (X-limus, Cardionovum, Bonn, Germany) developed to 16 atm (Fig. 2h). After undergoing the procedure, there were no complaints of chest pain anymore, but the patient began to experience hypotension; therefore, he was given dopamine (5 mcg/kg/min) and norepinephrine (0.1 mcg/kg/min) with a target systolic blood pressure of > 140 mmHg.

With a residual thrombus after stent placement, the risk of stent thrombosis is very high. After considering the low bleeding risk (PRECISE-DAPT score = 10), the patient was given triple therapy: aspirin 100 mg every 24 hours and ticagrelor 90 mg every 12 hours, followed by warfarin 2 mg every 24 hours one day after PCI. During the three days of intensive care after primary PCI, there were no complaints of chest pain or signs of bleeding; this may be related to the absence of an increase in the INR value in the lab results. On evaluation angiography performed four days after primary PCI, complete dissolution of the thrombus was seen with TIMI flow grade 3 on the RCA. Subsequently, the patient was discharged six days post-primary PCI with dual antiplatelet therapy (aspirin and ticagrelor) for more than 12 months. There were no complaints of chest pain or bleeding during the patient's follow-up visits on days 3 and 30 after being discharged from hospital.

Discussion

Intracoronary Thrombus

The pathophysiology underlying the development of intracoronary thrombus is Virchow's triad, triggered by rupture or erosion of an atherosclerotic plaque resulting in thrombus formation in the epicardial coronary arteries. In approximately 70% of cases, acute thrombus formation occurs after atherosclero-