Undilatable RCA CTO in a patient with NSTEMI

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Patient's profile

- Mr S.L Kuo, 82 y/o male
- Admission due to TB pleuritis
- Past history include: Smoker for decades but quitted 10 years ago, old stroke, essential hypertension

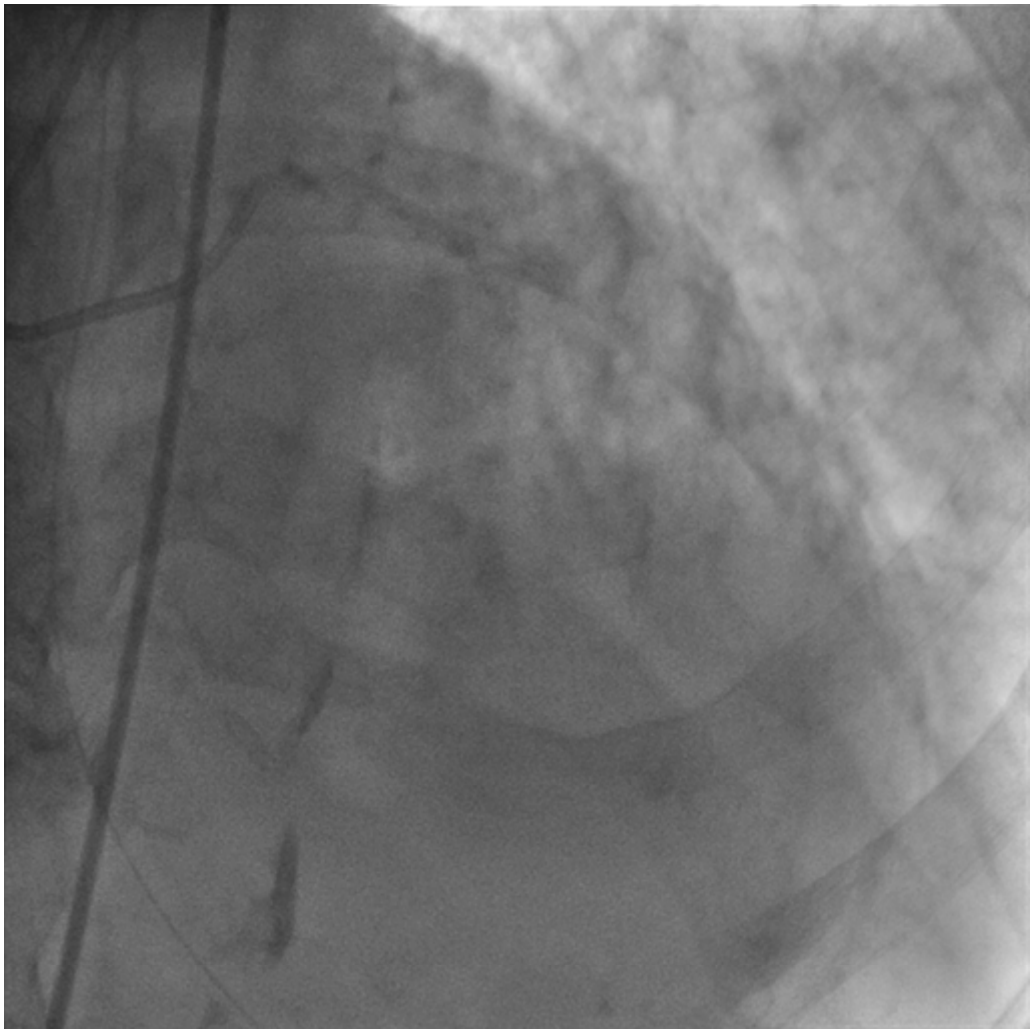
Clinical course

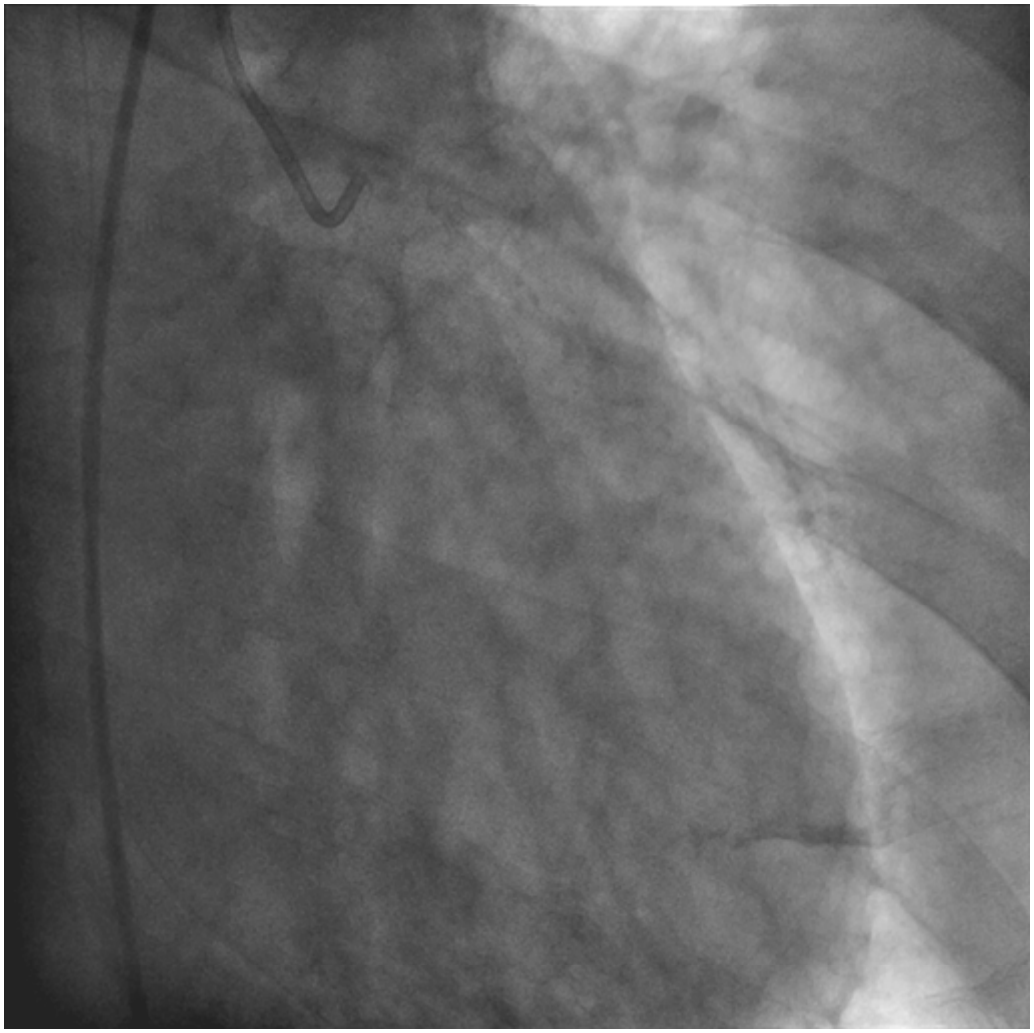
- Worsening of dyspnea 2 days after the admission due to TB pleuritis, in treatment at chest medicine ward. New onset of chest tightness also noted.
- Sinus tachycardia with new onset of PAf noted
- Physical examination revealed new bilateral basal rales on chest auscultation.
- ECG showed new ST depression from V2-5
- Cardiac enzymes were elevated: CPK 621 IU/L, CK-MB 108 ng/ml, troponin-I 17.3 ng/ml.

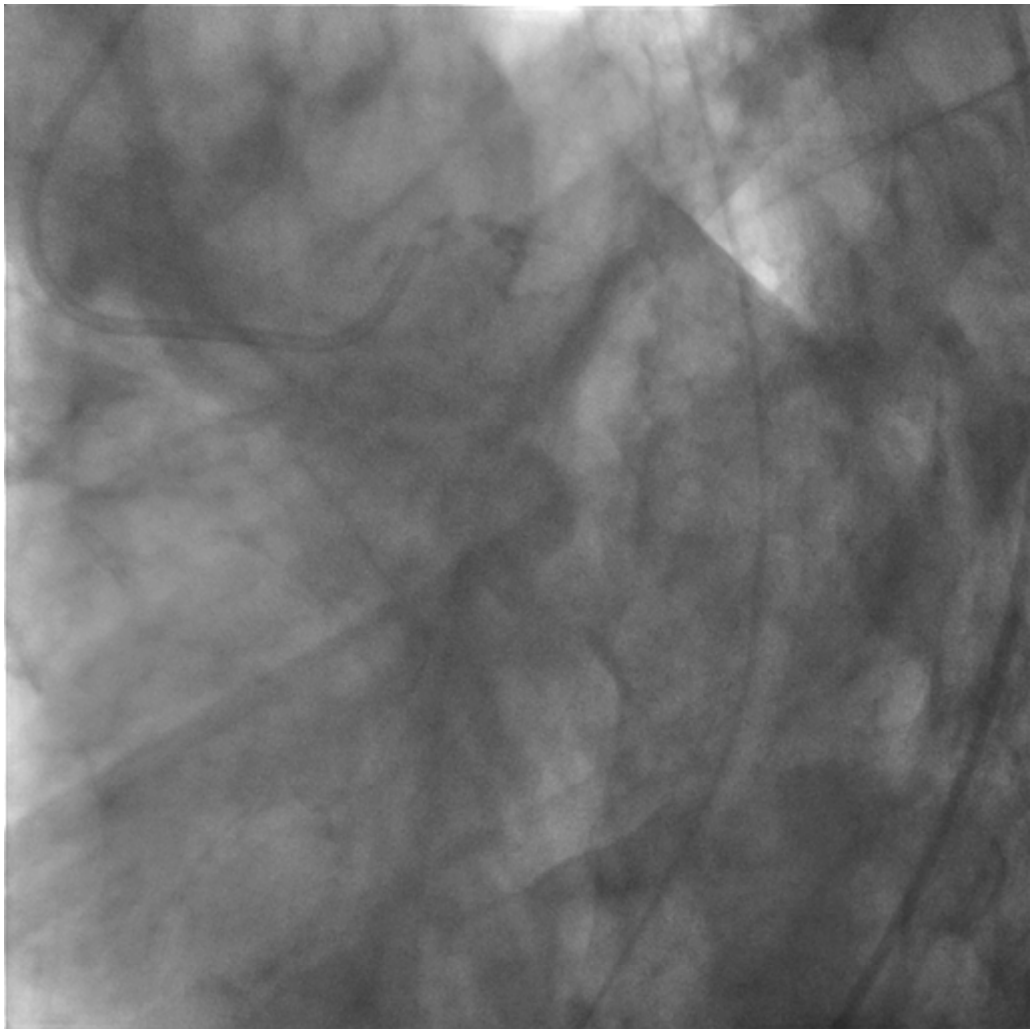
- Echocardiography showed impaired systolic function with EF 42%, regional hypokinesia on mid-apical areas of LV anterior-lateral wall. Moderate MR and mild TR were impressed.

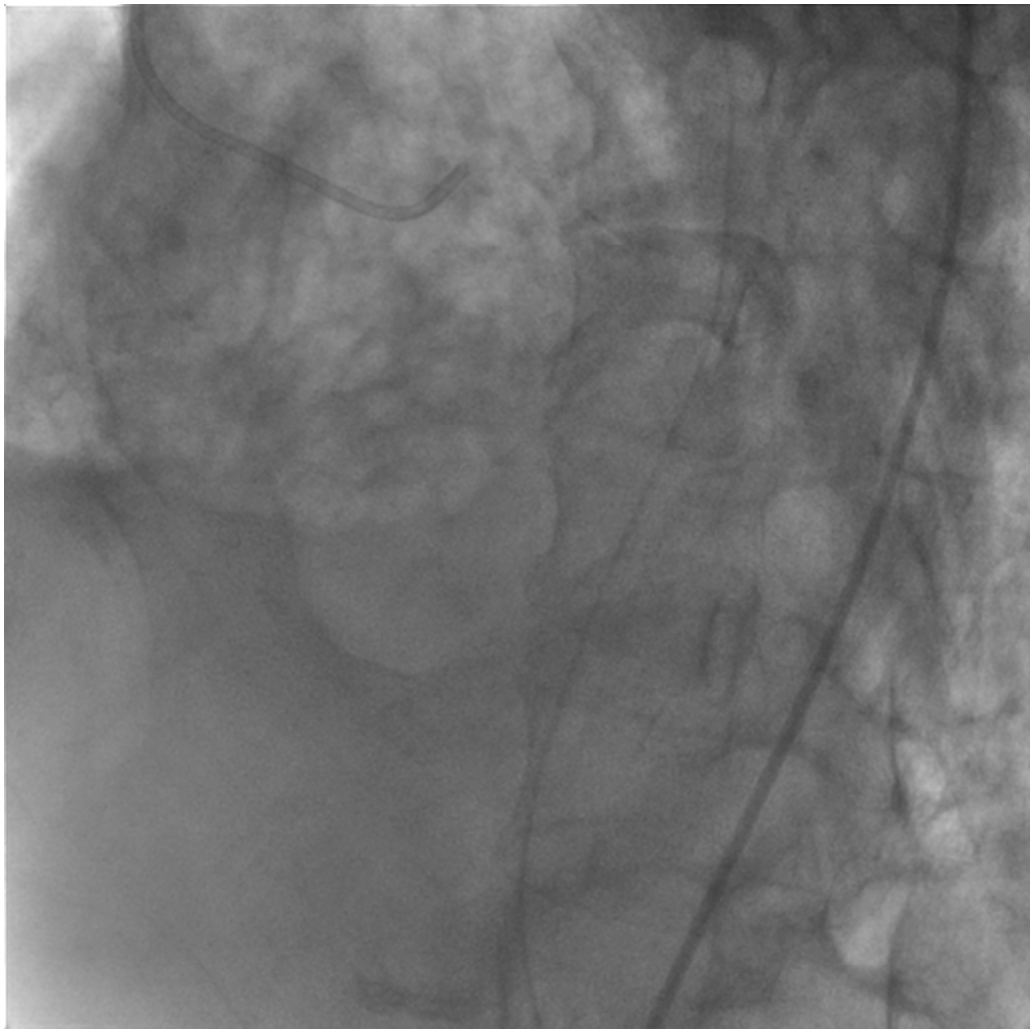
- NSTEMI was impressed .
- Because of old age, his family members requested conservative medical treatment first. Dual antiplatelet drugs (aspirin and clopidogrel.), enoxaparin, captopril, NTG iv infusion, diuretics, were given
- Recurrent short-run VT noted on the hospital day 3. Early coronary angiography and eventually PCI was recommended again.
- Cardiac catheterization was arranged.

Diagnostic coronary angiography.









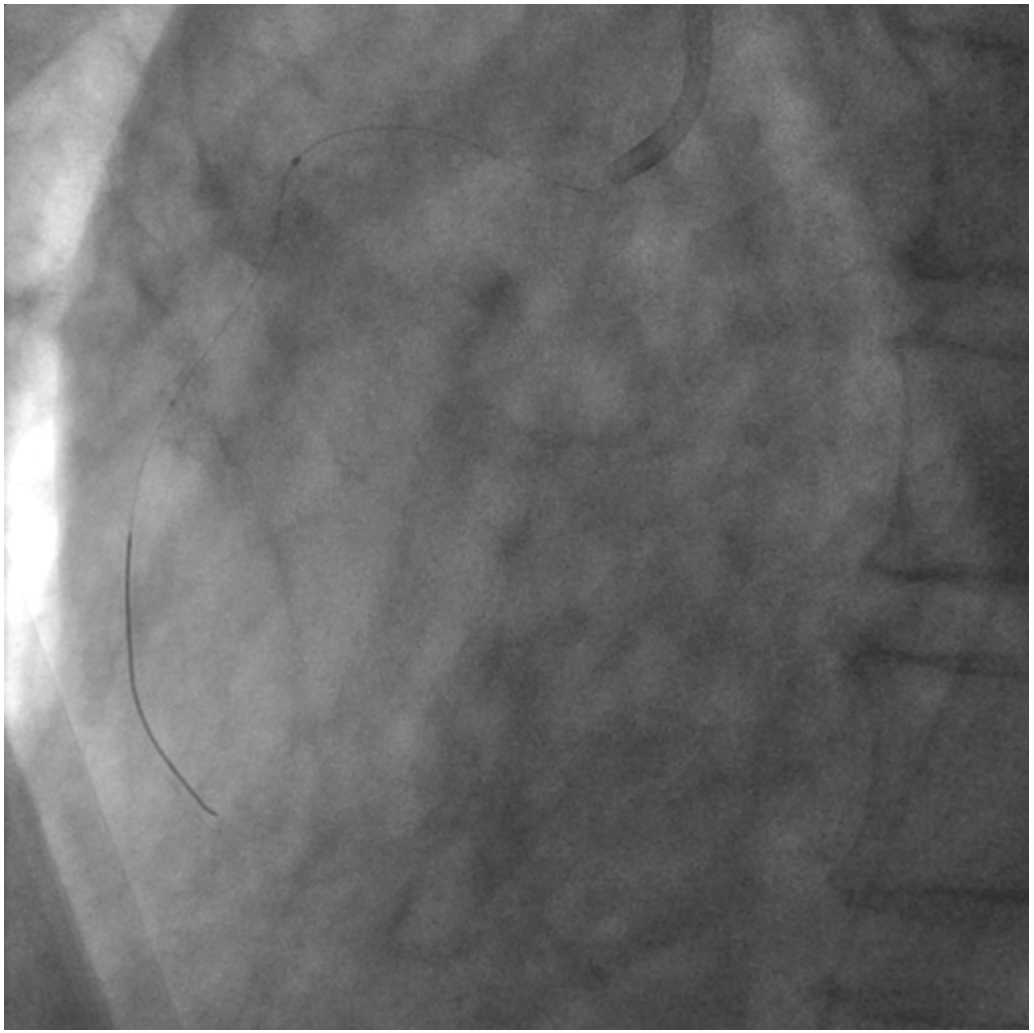
Decision making…

- Why and how the NSTEMI occurred?
- Infection related stress causing overload of the heart?
- Was the LAD the target vessel?
- Was RCA with acute thrombosis on the already existent lesion as cause of NSTEMI?
- Or the LAD already with atherosclerotic lesions was unable to supply enough flow to collaterals to RCA due to stress secondary to infection?

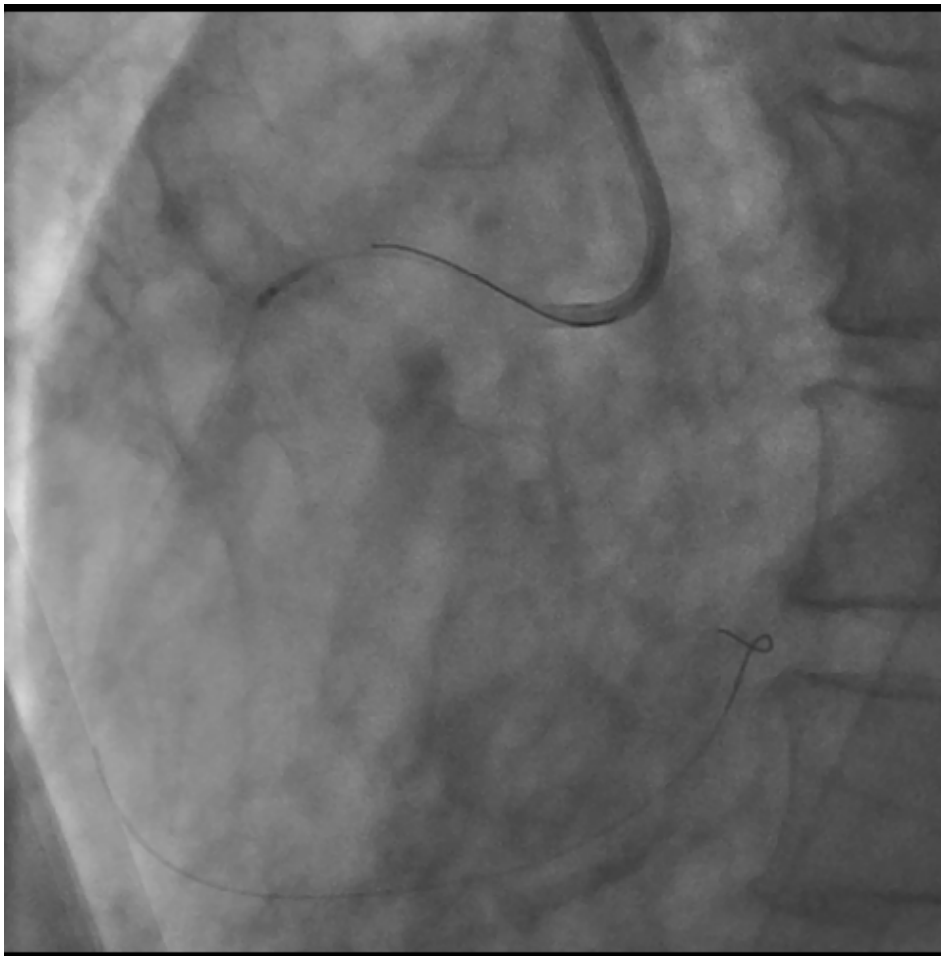
We decided …

- As LAD and LCX had TIMI III flow, we decided to attempt revascularization on RCA, trying to lower the loading of the LAD, which supplies collaterals to RCA.
- Femoral approach.
- Guiding catheter: 6Fr Medtronic SCR 3.5
- 5Fr JL 4.0 for left coronary angiography, for contralateral enhancement of left coronary arteries to see its collaterals to RCA.
- Support the Asahi Neo's Fielder FC wire with Terumo Ryujin OTW 1.25x10 balloon

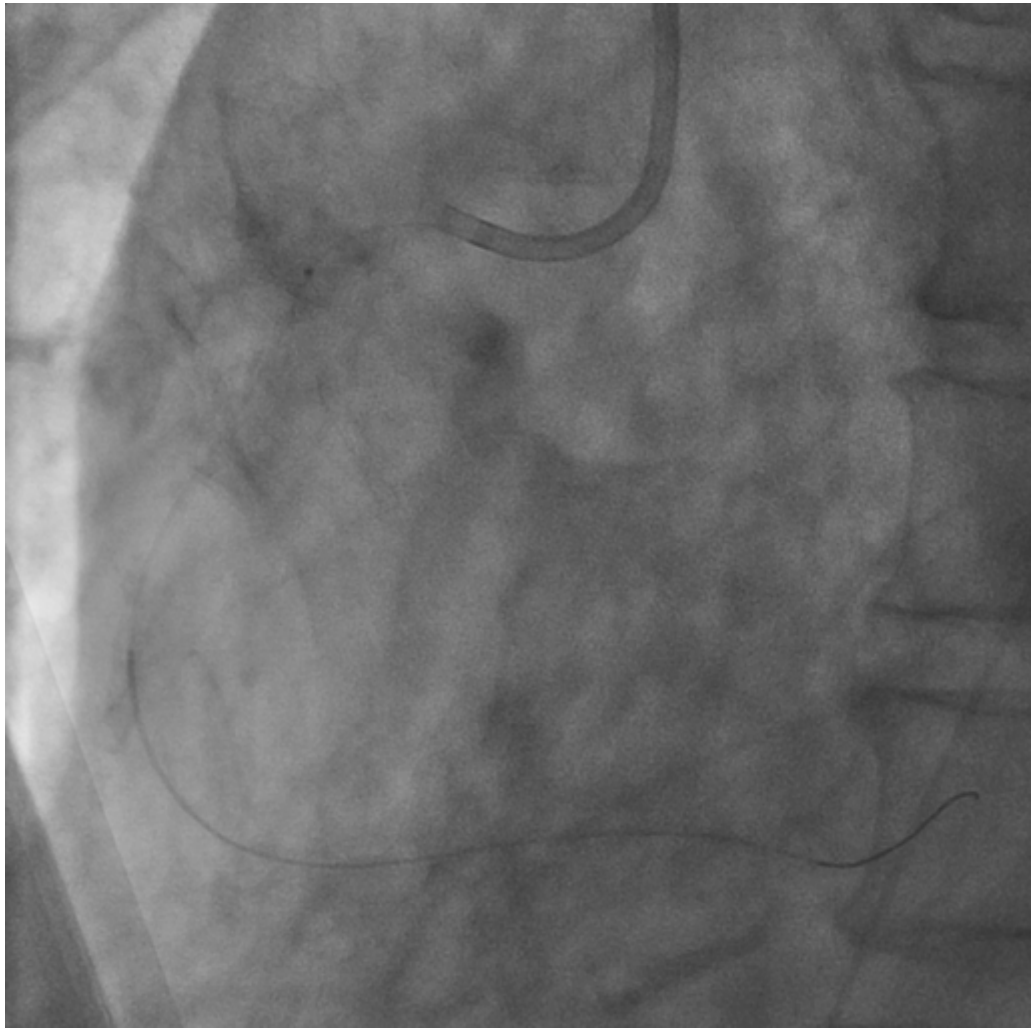
Two difficult curves: proximal RCA and then distal RCA. OTW balloon helped the wire to cross the first curve, but the balloon was unable to cross the lesion to support the wire in the second curve



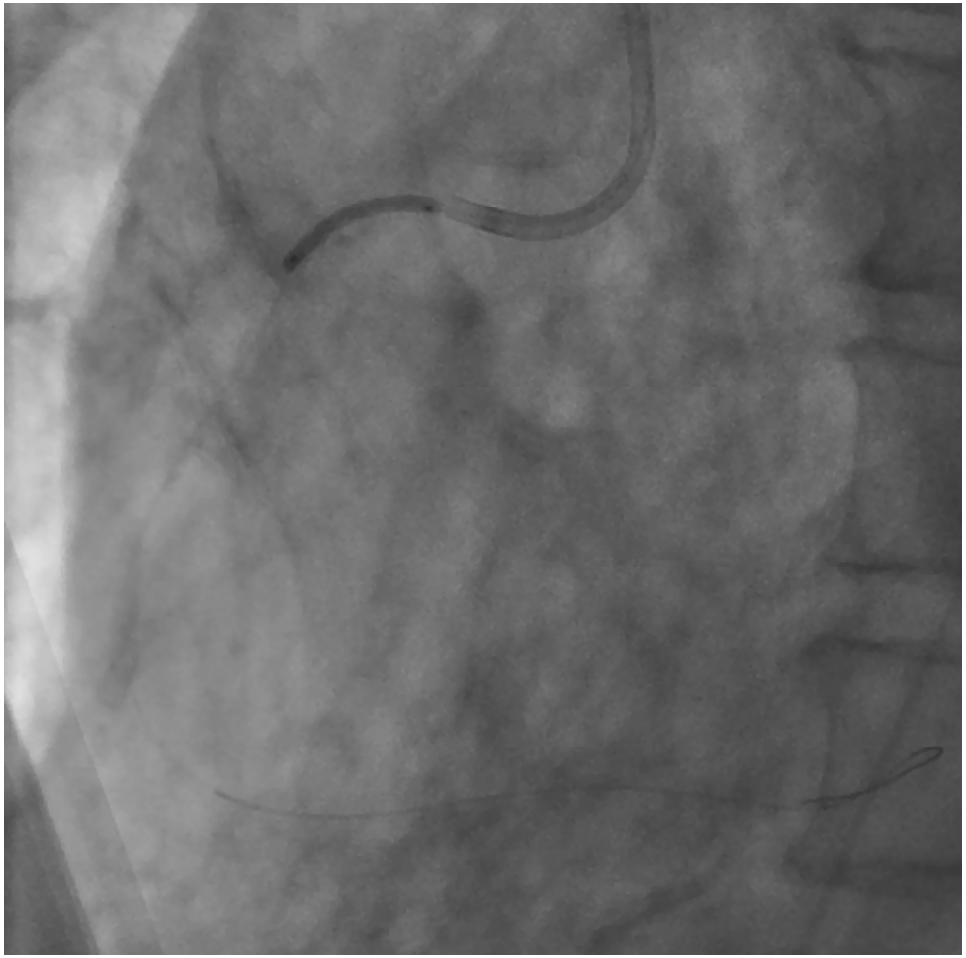
The Fielder FC wire achieved to reach distal RCA.
But the 1.25 OTW balloon was still unable the cross the proximal RCA lesion. We tried with additional Runthrough NS wire. Failure to perform buddy wire technique. Wire anchoring technique failed to enhance support and delivery of the OTW balloon distally. We tried to dilate the lesion on RCA-p

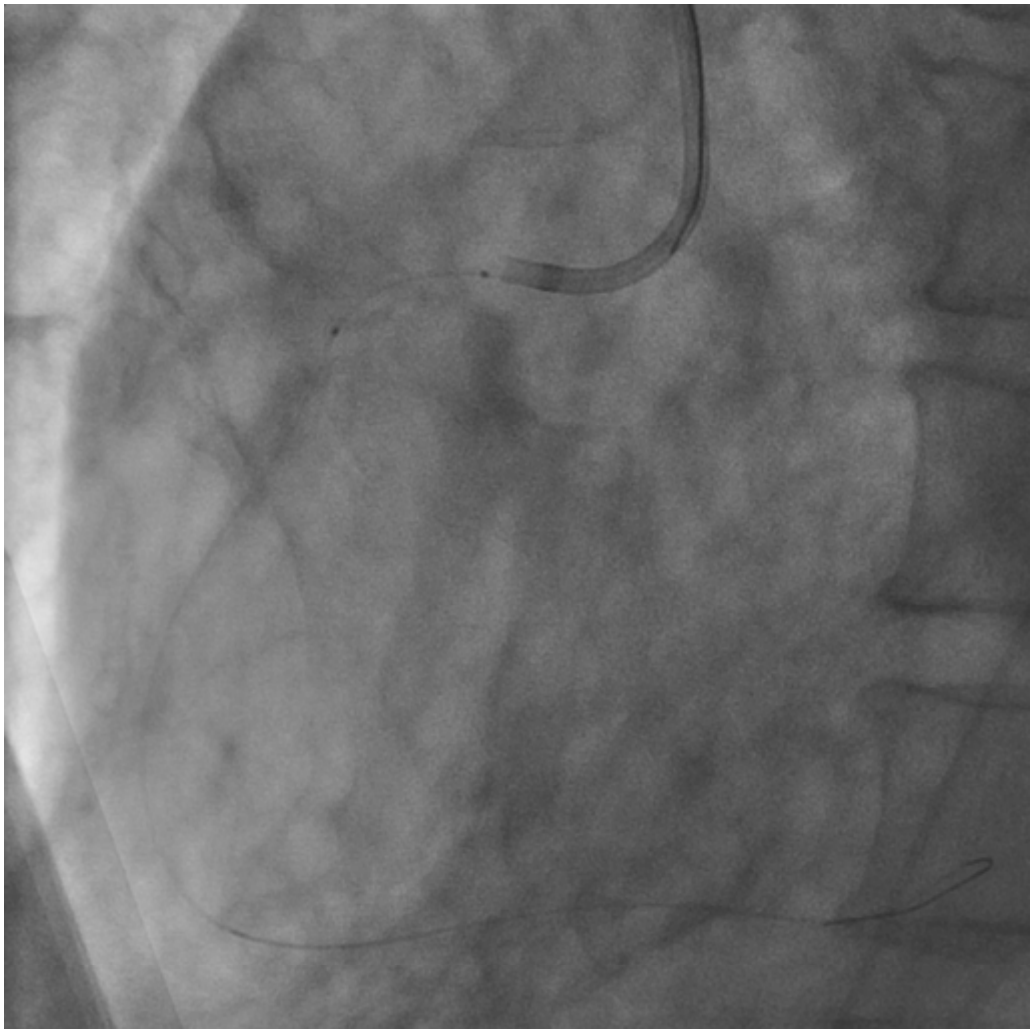


After the OTW 1.25 balloon dilatation, 10 atm

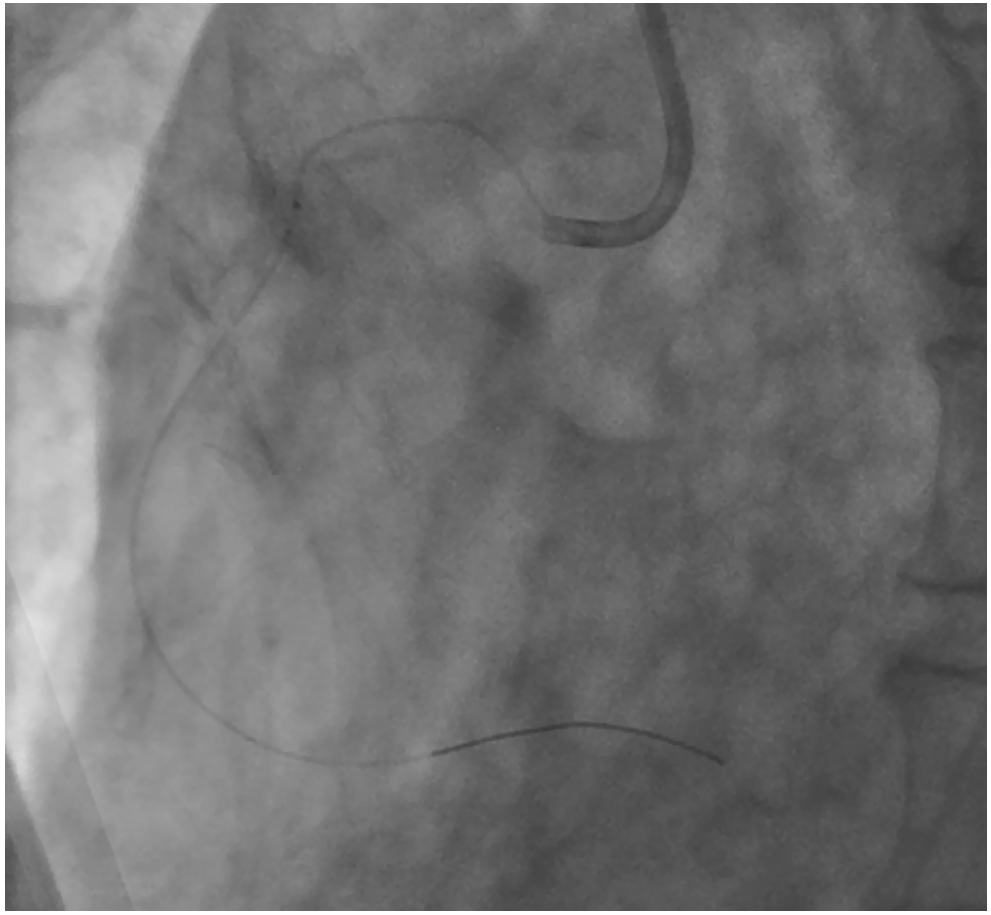


We tried with Medtronic Sprinter 2.0x20 balloon, 6 atm, 2 times.





We tried with smaller balloon: Medtronic Sprinter 1.25x6 , to see if can cross the lesion and reach the RCA-m. Attempt with deep seating guiding catheter using balloon anchoring.
All these manuever failed.
As we don’t have Tornus catheter or Rotablator …



Final angiography of RCA

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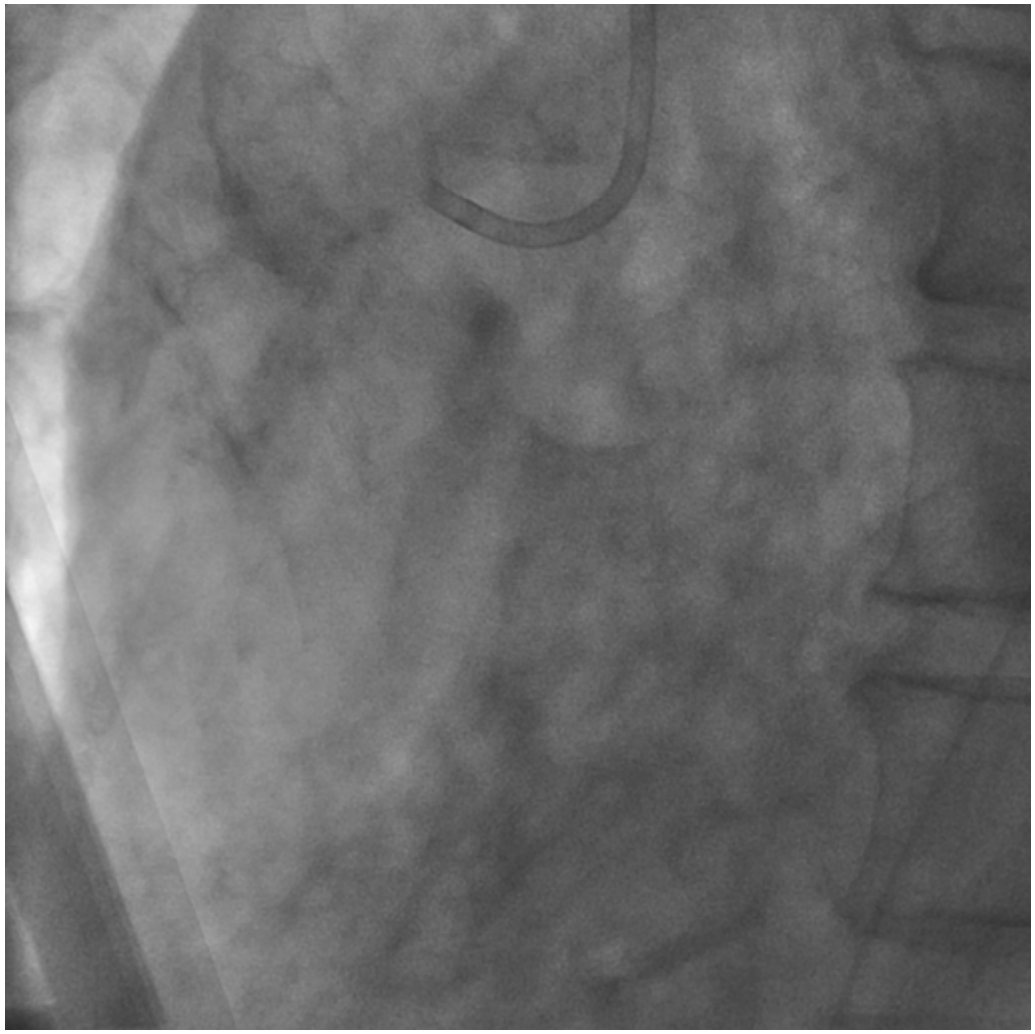

- After the procedure, no further short-run VTs, recovered to sinus rhythm, symptoms and signs of heart failure had resolved within 2 days post PCI.
- We continued with dual antiplatelets, enoxaparin, captopril, loop diuretics and added bisoprolol after resolution of pulmonary congestion. Patient remained hemodynamically stable

discussion

- Would be better start the intervention on RCA with microcathter? Which microcathter would be most suitable to these lesion?
- Further intervention for RCA heavy calcified lesion:Tornus catheter? Rotablator?
- Does LAD need intervention? FFR ? IVUS?
- The LM is short and seem to have a stenosis of nearly 50%. Further evaluation with IVUS ? If it have to be intervened, which strategy?