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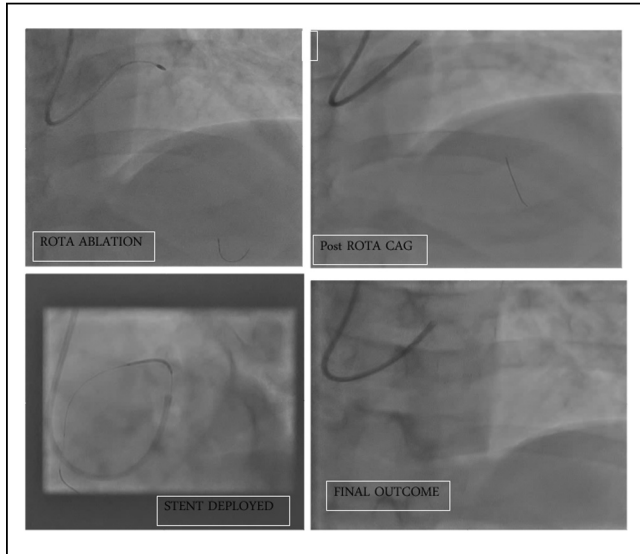
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CLINICAL INFORMATION

Patient Initials or Identifier Number. PE

Relevant Clinical History and Physical Exam. A 71-year-old lady, with a background history of diabetes mellitus, hypertension and atrial fibrillation, presented with progressive heart failure symptoms for the past 1 month with occasional chest pain. She was previously under medical clinic follow up and the last echocardiogram 4 months prior was normal with an EF >55%. The working diagnosis was that she may have had a silent myocardial infarction. She was started on Tab Frusemide, Tab Spironolactone, Tab Aspirin, and Tab Bisoprolol.

Relevant Test Results Prior to Catheterization. A repeat echocardiogram showed an EF 25% with global hypokinesia. A cardiac MRI was arranged to assess for myocardial viability. Interestingly the MRI showed no areas of infarction but there was a mid-wall fibrosis at the posterior LV and at the insertion point of the RV.

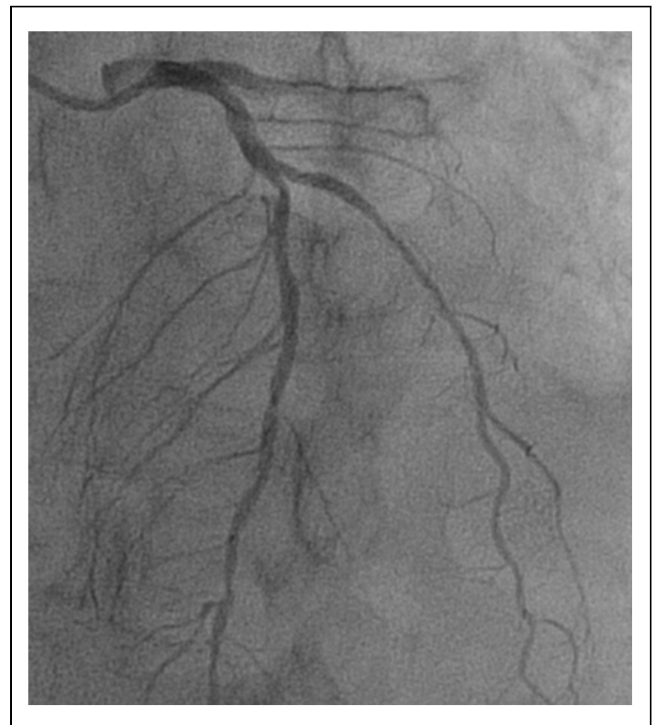
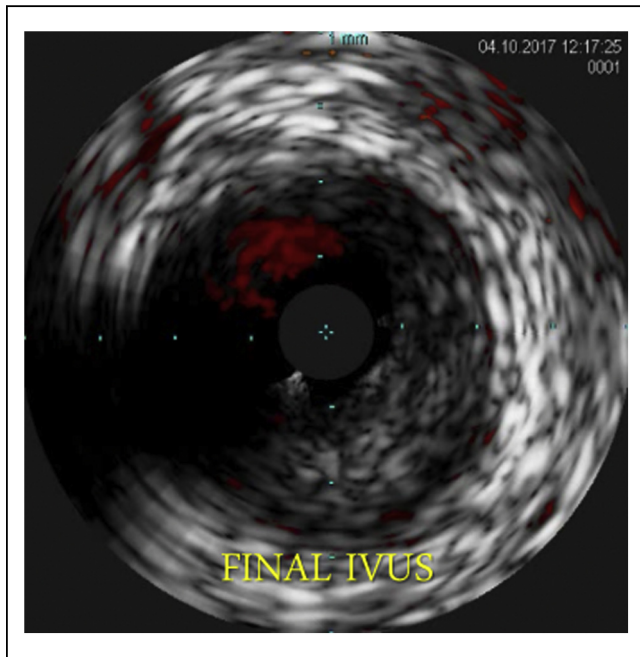
Relevant Catheterization Findings. As she had had chest pain, a diagnostic angiogram was also done which showed an intermediate lesion in the LAD 50% stenosis and a diseased RCA with 90% proximal and a long diffuse disease from mid to distal of about 80-90%.

INTERVENTIONAL MANAGEMENT

Procedural Step. We decided to do further assessment of the LAD lesion.

A 6Fr EBU 3.5 guiding catheter was used to engage the left coronary artery. The LAD was wired distally with a St Jude FFR floppy wire. Resting FFR was 0.89. IC Adenosine 120 mcg bolus was given however the post adenosine FFR was still not significant at 0.86. Therefore, the lesion was left for medical therapy.

As she had had history of chest pain, we decided to intervene for the RCA lesion. We proceeded to engage the right coronary artery with a 6F JR4 guiding catheter. A BMW coronary wire was passed down to the distal RCA. Pre-dilatation was done sequentially to the proximal, mid, and distal lesion with a semi-compliant balloon 2.0 x 12 mm up to 14 ATM. The mid to distal lesion was treated with drug coated balloon Sequent Please 2.25 x 40 mm at 8 ATM for 60seconds. The proximal lesion was treated with a bare metal stent Azure 2.5 x 15 mm and inflated at 10 ATM for 30 seconds. The stent was then post dilated with a non-compliant Sapphire II balloon 3.0 x 10 mm at 16 ATM for 20 seconds. The final angiogram showed a well expanded stent in the proximal segment and minimal recoil in the distal segment treated with drug coated balloon. There was TIMI 3 flow to the distal RCA. Post procedure was uneventful.



Conclusions. 1. Difficult strategy to adapt
 2. Post procedure not visualizing in IVUS as well as in ANGIO. The aneurysm could not be explained.
 The strategy which we followed and did it in our place Right or Wrong...?

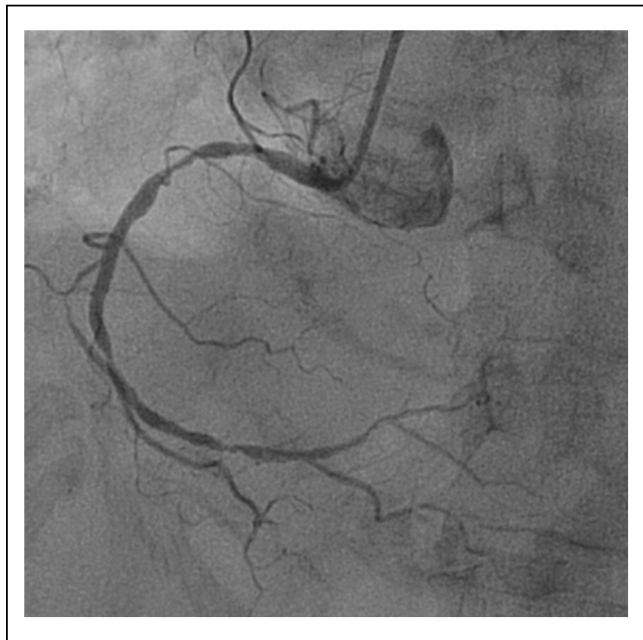
PHYSIOLOGIC LESION ASSESSMENT (TCTAP C-206)

TCTAP C-206
Bystander Coronary Artery Lesion in Non-ischemic Cardiomyopathy: Should We Treat It?

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INNOVATIVE DEVICES AND FUTURISTIC THERAPIES (TCTAP C-207)

TCTAP C-207

Atrial Septum Fenestration for Patient with Idiopathic Pulmonary Artery Hypertension

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CLINICAL INFORMATION

Patient Initials or Identifier Number. A.B.

Relevant Clinical History and Physical Exam. A 21 y.o. M admitted to Cardiac Center with low tolerance to physical exercise, exertional dyspnea.

Anamnesmorbi: exertional dyspnea x 10 y. First presyncope in 06.2014. Cardiac catheterization in 10.2014 - Pulmonary Hypertension.

Meds: bosentan 250mg/day, diltiazem 180mg/day, iloprost 10 mg x 6/day, furosemide 80mg, eplerenone 50mg, hydrocortisone 10-10-5mg, levothyroxine 75mcg.

SH: 2011 endosupracellarcranopharyngioma resection. Secondary hypothyroidism, hypogonadism, hypocorticism.

Relevant Test Results Prior to Catheterization. EchoCG before admission (24.01.17.)

Left ventricle - 3.9 cm, Left atrium - 3.7 cm, Right ventricle - 4.7 cm, wall - 0.7 cm, Right atrium - 8.5 x 6.4 cm, EF(Lv) - 70%, EF(Rv) - 40% Sim, S(Ra) - 59 cm².

Dilatation of the right chambers with signs of RV overload and a moderate decrease in its contractile function. Doppler: mitral regurgitation (+), tricuspid regurgitation (+++) with gradient 60 mmHg. Right ventricle pressure systolic 80 mmHg, diastolic 40 mmHg

Relevant Catheterization Findings. Atrioseptostomia with implantation of the "modified" occluder MemoPart under the control of the Ultra ICE probe and TTE. A conventional ASD occluder (Memopart, LePu Medical etc, 14 mm) was modified by keeping out all the polyester patches (left disc, right disc, and waist) and fenestrated by balloon dilation before implantation.

INTERVENTIONAL MANAGEMENT

Procedural Step. Atrioseptostomia with implantation of the "modified" occlude Memo Part under the control of the Ultra ICE probe and TTE. Trans septal puncture of the atrial septum was performed under TTE and Ultra ICE control in the cath lab. Consecutively, pre-dilatation of atrial puncture site by balloons EMPIRA 1.5x20, EMPIRA NC3.75x15 and TYSHAK 12 mm and 15 mm advanced over the guide wire in the left atrium were performed. A conventional ASD occluder (Memopart, LePu Medical etc, 14 mm) was modified by keeping out all the polyester patches (left disc, right disc, and waist) and fenestrated by balloon dilation before implantation. The Nitinol wires at the border of this fenestration were kept opened by stitches with a final 7 mm hole. So, the home-made "fenestrated" occluder was finally implanted under the control of the Ultra ICE probe and TTE. Control of the stability of the occluder's position by "push & pull test" on fluoroscopy, intracardiac examination, and TTE was accomplished and we finally deployed the modified occluder appropriately.

Postoperative period with no complications.

Conclusions. Bystander coronary artery lesion can occur in non-ischemic cardiomyopathy. Functional assessment is required, especially in cases where the patient also had had angina. On follow up, the patient did very well and was out of heart failure. Her repeated echocardiogram showed remarkable improvement with EF >55%, and she no longer had chest pain.

This case illustrated that selective PCI and optimal medical therapy can improve the outcome of non-ischemic cardiomyopathy.