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position giving large, sharp His potential signals on the EP system. 5 patients who had CHB with normal cardiac function were treated with RVOT pacing placing the lead in the upper posterior RVOT using standard LAO 40 and RAO 30 degrees view, getting as close to the His bundle position as possible. In the former view the lead tended to point posteriorly and upwards and in the latter the extreme right quadrant in the upper row of the “9 quadrants” was targeted. Using a peel away sheath, a standard 58 cm pacing lead was deeply screwed in with at least 17–18 turns. Lead stability was confirmed before the lead was fixed in situ. ECHO confirmed the lead position in upper posterior RVOT. Data of achieved parameters from this technique was compared with 10 patients who underwent RVOT mid-(5) septal and lower septal (5) pacing (abbreviated MS/LS) and 20 (historic) patients in RVAP groups. Immediate parameters showed a significantly narrower QRS duration (mean 96 +/- 10 msec v/s 124 ms +/- 10 msec (MS/LS) v/s 140 msec (RVAP) with deep Q waves in I and aVL leads (all $p < 0.001$), with “safe RBBB” occurring in the all patients in upper posterior RVOT group v/s only one in the mid and lower septal group. Obviously none of the RVAP group had these parameters suggesting septal pacing. Procedure time was not significantly longer in RVOT US than either RVOT MS/LS group or the RVAP group. After a mean follow-up of 9 +/- 3 months (mean 11 months), lead stability was re-confirmed in all patients. None of the RVOT leads had dislodged or any untoward event like perforation /pericardial effusion occurred. The final maximum P-wave duration; P-wave dispersion; QRS-wave complex duration, LV end-systolic and diastolic diameters and interventricular mechanical delay were significantly less in the upper posterior RVOT septal pacing group guided by the His-bundle catheter than those in the MS/LS pacing group as well as in the RVAP group ($P < 0.05$). The final LVEF of the RVOT septal pacing group remained normal, and was significantly higher than that of the RVAP group (55% to 51.5% over a year a significant drop, $P < 0.05$).

Conclusions: Compared with RVAP, upper RVOT septal pacing particularly targeting the upper posterior RVOT gives better immediate electrical parameters even than mid-septal and lower-septal pacing and in the longer term has fewer adverse effects in patients with normal cardiac function. ECG parameters almost resemble LV pacing and therefore this technique merits more investigation for its longer-term benefits.

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A CASE OF PNEUMOPERICARDIUM AND PNEUMOMEDIASTIUM IN COVID 19 PATIENT

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which originated in Wuhan China, is spreading all around the world, and the outbreak continues to escalate. Clinical features of patients with coronavirus disease 2019 (COVID-19) usually include dry cough, fever, diarrhoea, vomiting, and myalgia. However, atypical presentation and complications are reported. We are reporting a case of pneumopericardium, pneumomediastinum in a patient with COVID-19 disease.

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ST SEGMENT ELEVATION MYOCARDIAL INFARCTION IN INHERITED THROMBOPHILIA (FACTOR V LEIDEN MUTATION) - DOES FIBRINOLYSIS WITH TRIPLE THERAPY SCORE OVER PRIMARY PERCUTANEOUS CORONARY INTERVENTION?

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Factor V Leiden mutation is an autosomal dominantly inherited single gene missense mutation, leading to increased resistance of Factor V to inhibitory action by the natural anticoagulant activated protein C. This leads to a very high incidence of venous thrombosis in these patients, with homozygous patients have 80 times greater risk compared to general population, while heterozygous patients have 8-fold increased risk. However arterial thrombosis is rare and less reported. We report the case of a young male

with heterozygous factor V Leiden mutation and acute myocardial infarction.

This 28-year-old male presented with complaints of chest pain with increased sweating and palpitation. ECG showed Acute anterior wall STEMI with RBBB. His vitals were stable during admission. Transthoracic echocardiogram showed hypokinesia of anterior wall, anterosseptal and anterolateral wall with moderate left ventricular systolic dysfunction (LVEF-41%). There was a history of deep vein thrombosis in the left femoral vein, the previous year for which he was treated with anticoagulants. He had discontinued therapy after 10 months. There was a family history of death due to acute myocardial infarction in his paternal grandfather aged 55 years and his first cousin aged 39 years. Two of his first cousins had been evaluated for recurrent abortions, with a suspicion of possible anti-phospholipid antibody syndrome. He was thrombolysed with streptokinase within time window of 4 hours. Fibrinolysis was successful with full resolution of ST elevation and chest pain. He was taken up for pharmacoinvasive procedure the next day. Coronary angiogram showed right dominant coronary circulation with a large type III LAD with a Grade 4 thrombus in the proximal part astride origin of D1, with distal TIMI III flow. He was put on with oral anticoagulation with acenocoumarol along with aspirin and clopidogrel. He was diagnosed to be a diabetic during the current hospitalisation and was started on hypoglycaemic therapy. In view of his young age of coronary thrombosis with a prior history of venous thrombosis, he was evaluated for thrombophilia. He was found to be positive for heterozygous Factor V Leiden mutation. His lupus anticoagulant was also positive. He was taken up for a check coronary angiogram after 3 weeks of treatment, which showed a recanalized LAD without any residual thrombus or lesion, and good distal TIMI III flow.

It has been reported the PCI with stent implantation in a high thrombus burden, can lead to slow/no reflow, distal embolization, microvascular occlusion and increased infarct size. Protrusion through stent struts and mal-opposition can lead to stent thrombosis as well. Further in a thrombophilic patients STEMI is most often due to plaque erosion with in situ thrombosis than a plaque rupture. Hence, we postulate that triple therapy with oral anticoagulation and dual antiplatelets is a good treatment strategy for thrombus with distal TIMI III flow identified post lysis for STEMI, in patients with factor V Leiden mutation.

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THE BAILOUT LEFT MAIN STENTING AFTER DIAGNOSTIC CATHETER-INDUCED CORONARY ARTERY DISSECTION

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Background: Catheter induced dissection of the coronary arteries is a rare but potentially catastrophic complication of coronary angiography and angioplasty. We discuss a case of left main dissection induced with diagnostic catheter and subsequent life saving bailout stenting of left main and left anterior descending (LAD) artery.

Method and Results: The patient was 63 year old female, known case of CAD (Coronary artery disease) with history of PCI with stenting to LAD artery. The present admission was with Unstable Angina and the patient was planned for check coronary angiography (CAG). The patient was taken for diagnostic CAG, through right radial route using Tiger 5F catheter. The catheter was not coaxial with the left main stem and there occurred a long, linear dissection (Type 1) involving left main and LAD just after the first injection of contrast. The primary operator without knowing about the coronary dissection occurred, kept on continuing the CAG procedure and with 2 more injections patient developed complete occlusion from proximal LAD and proximal LCx (Left circumflex) arteries. With prompt response, femoral puncture was done and using JL 3.5, 6F guide catheter, both LCx and LAD were wired with RUNTHROUGH NS workhorse wires. The LAD was dilated using 2.5 × 15 mm SC (Semi compliant) balloon, and DES of size 3.5 × 28 mm was placed from left main to proximal LAD. Ostial LMCA (Left main coronary artery) was missed, so another DES of size 4.0 × 16 mm was placed from ostial left main to proximal LAD, overlapping the previous stent from LM to LAD. Another stent (size 3.5 × 21 mm) was placed in the proximal to distal LAD. Spontaneous flow was restored in LCx. POT (Proximal optimization technique) was done using 5×8 mm NC