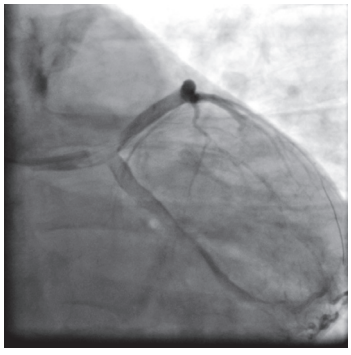
CHALLENGING CASES – A JOURNEY THROUGH THE CARDIOLOGY UNIVERSE

25
A complex left main PCI in the setting of disastrous intrapartum STEMI. thrombus , dissection, no-reflow and perforation. multiple complications in disguise

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Clinical history: A 31-year-old female 38 weeks Primigravida, with no risk factors for CAD except +ve family history. Developed sudden onset severe central chest pain while in labor. ECG: ST elevation in V1-V6, ST depression in I II AVF. A diagnosis of anterolateral myocardial infarction was made. After successful delivery, arrangements were made for interhospital transfer for urgent primary percutaneous coronary intervention (PCI).
On physical examination: ABP 90/50 mmhg Pulse 110 Beats per minute regular equal on both arms Chest examination:Harsh vesicular breathing The first and second heart sounds were normal RBS 130 mg per deciliter Hg 11 g per deciliter International Normalized Ratio (INR) 1.2 Creatinine level: 1.1 mg per deciliter Mild thrombocytopenia.
Angiography: LM: Normal stem bifurcating into LAD & LCX. LAD: Shows an osteal thrombus filling (+/– Coronary dissection) 80% lesion with fair distal flow. LCX: Normal vessel. RCA:Normal vessel.
Procedure step: A radial approach using a left EBU 3.5, 6f guiding catheter used to cannulate the right and left coronary artery. introduction of devices into the coronary artery, led to the dissection of the Left Main and the LCX with retrograde extension into the aortic root and ascending aorta Then TAP technique to LM-LAD and LCX, followed by final simultaneous kissing balloon. Distal LAD wire perforation, Immediate Tamponade and cardiac arrest. CPR, Pericadiocentesis, and prolonged balloon inflation to seal the perforation. Followed by LAD No- reflow, multiple intracoronary injections, with final result of LAD TIMI II flow. Patient admitted to CCU and unfortunately 3 days developed Ventricular fibrillation and died.

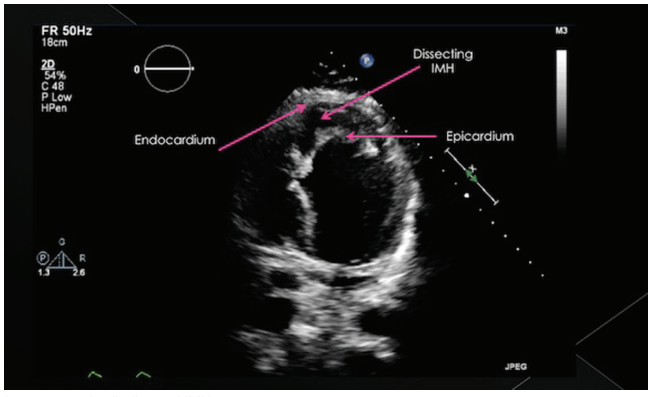


Conclusion: Pregnancy associated MI (PAMI) is rare and different from AMI in nonpregnant patients in several important aspects that need to be taken into strong considerations in the management of women with this condition. There is frequent involvement of the LAD and LM, and the location of PAMI is commonly the anterior wall, resulting in a high incidence of LV dysfunction, congestive heart failure, cardiogenic shock, and mortality. large number of cases have Coronary dissection or normal coronary anatomy, the risk of thrombolytic therapy may outweigh a potential benefit. The high incidence of iatrogenic coronary dissection secondary to intracoronary contrast injection and mechanical interventions suggests that an invasive approach to PAMI should be limited to high-risk patients and that mechanical coronary manipulations should be limited to cases in which potential benefits clearly outweigh the risk. More information is needed on the mechanisms of PAMI, the efficacy and safety of standard therapy, and the applicability of guideline recommendations designed for the general AMI population, to women with PAMI. Management of complications associated with PCI requires rapid recognition, reversal and arrest of the underlying pathogenic mechanisms when possible.

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A hit that was a miss- a STEMI with a twist

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A 35 year old gentleman presented after a fall from a motorcycle leading to traumatic injury on the left side of the chest, followed by sudden syncope which lasted for 10–15 minutes. A day later he began to experience severe retrosternal chest pain associated with profuse sweating, vomiting and weakness. He was rushed to the nearby hospital where the ECG showed acute ST elevation indicating an anterior wall myocardial infarction after which he was transferred to our hospital for further management. On arrival, he appeared sick, sweating with cold clammy extremities, in shock with BP 80/60 mmHg, tachycardia 142/minute normal sinus rhythm. Auscultation revealed bilateral basal crepitations up to mid-scapulae with LV S3 suggesting pulmonary edema secondary to left ventricular failure (LVF) with cardiogenic shock. He was planned for Primary PCI. Emergency echocardiographic screening was done which is a routine practice. This revealed a very large variably echogenic structure involving the apex, anterior septum and mid anterior wall. The ejection fraction was 25% with severe mitral regurgitation (MR). There was severe pulmonary hypertension with PASP 60 mmHg., restrictive grade 3 diastolic dysfunction and central venous pressure of 15 mmHg or more. Diagnosis of Intra-Myocardial dissecting hematoma (IMH) was based on the presence of an expansile mass bound by intact endocardium and epicardium which expands into the LV cavity during systole. In view of his chest trauma, CT chest was done which revealed lung contusions and confirmed the findings of IMH.
Due to his unstable hemodynamic condition intra aortic balloon pump (IABP) was deployed to stabilize the patient in the ICU. PCI was deferred due to the fact that post PCI compulsory aggressive use of anti-thrombotics (heparin, dual antiplatelet agents) could further increase the IMH and may lead to catastrophic pseudoaneurysm, cardiac rupture, infarct extension, arrhythmias, aggravation of LVF and mortality. The patient was kept on medical management with low dose aspirin. Clopidogrel and heparin were withdrawn. High dose statins were added. He subsequently underwent coronary angiography, which revealed 100% occluded Left Anterior descending artery (LAD) but intervention was deferred due to above mentioned reasons and the fact that the presence of IMH denotes a transmural infarct and not much benefit would have been achieved by PCI at this stage.
Gradually he was weaned off the IABP support and optimized on medical therapy and discharged a month later with good functional recovery but with persistent severe LV dysfunction and moderate MR. He came for subsequent follow up 2 months post factum, remains in NYHA class II on medical management. The echocardiogram showed a gradual regression of the hematoma and scar formation in the affected region. The literature on treatment of IMH remains controversial and scant.
The unique features of this case are: 1) Suspected traumatic occlusion of LAD masquerading as STEMI anterior wall. The delayed presentation of severe anginal syndrome after a day of trauma supports this etiology.
2) Value of echocardiography in emergency primary PCI setting which helped in diagnosis of IMH and altered the treatment approach completely to medical management over the usual primary PCI.
3) The role of IABP as a bail out treatment modality, prolonged ICU stay and poor LV recovery of function with potentially poor long term outcomes.



Important echo findings of IMH

4) Need for recognizing this rare entity of IMH and differentiate it from the intraventricular clot. Hallmark of IMH being the presence of an expansile echolucent mass bound by intact endo and epicardium with systolic expansion into LV cavity.

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Treatment of an iatrogenic left subclavian artery dissection by the left radial artery approach in a patient with coronary artery by-pass graft

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Introduction: Iatrogenic dissection of the subclavian artery during cardiac catheterization is a rare complication that is occasionally referred in literature, because of low incidence. We present a case of a left subclavian artery dissection caused by catheter and wire in a patient with a left internal mammary artery (LIMA) graft and angina symptoms. Treatment was performed by balloon angioplasty and stent placement, using a retrograde left radial approach.
Case presentation: A 67-year-old man who had coronary artery bypass graft surgery that included a left internal mammary artery graft to the left anterior descending artery, 13 years ago. He had exertional chest pain, dyspnea and palpitations since 2 years. He was hospitalized and left heart catheterization was performed from right femoral aproach. For the imaging of the LIMA, diagnostic JR4 catheter and 0.035-inch hydrophilic wire were pushed forward from the left subclavian artery togather but wire was not progressed. Contrast media injection was showed us the dissection of the subclavian artery at the proximal part before the LIMA. The dissection was rapidly progresse until total occlusion (Figure 1A). The patient had serious angina pectoris with hypotension. The left radial artery was prepared and a 6-French sheath was introduced into the nonpulsatile artery with

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Doppler technique. A 0.014 inch guidewire (300 cm) was passed through the dissected area of the left proximal subclavian artery to the descending aorta (Figure 1B). Guidewire was captured with a endovascular snare system (12–20 mm) from the 6-French JR4 guiding catheter and radio femoral wire loop has been occured. From the femoral sheath, dissected segment of the subclavian artery was passed through with a 5 -Fr diagnostic catheter and 0.035 inch hydrophilic guidewire was placed to brachial artery. Then the diagnostic catheter was replaced with a right guiding catheter. Percutaneous angioplasty was performed with a 5x40 mm balloon (Figure 2A). Following balloon deflation, a balloon expandable stent 8x59 mm was positioned in the proximal subclavian artery. The stent was deployed and angiography revealed that the stented area to be satisfactory with a good antegrade flow and absence of residual dissection. Normal flow was observed into the subclavian artery, vertebral artery, and LIMA (Figure 2B). Simultaneous pressure measurements were equal in the thoracic descending aorta and left brachial artery. After stent placement his angina was diminished and blood pressure was normal on the follow up. He was discharged without any complication 4 days later.

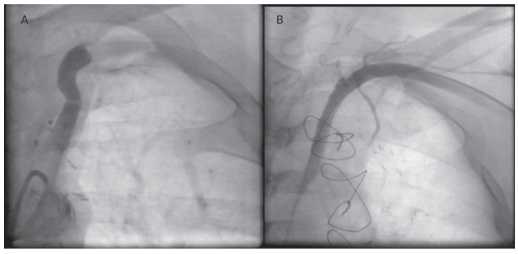


Figure. Dissection and treatment.

Discussion: The complication of iatrogenic subclavian artery dissection during diagnostic and interventional cardiac procedures is described as an infrequently condition and usually occurs when attempting to cannulate internal mammary artery grafts. There is no standart treatment protocol for its management. The management of the dissection can range from either a conservative or interventional approach according to the side branch vessels involved and distal flow. The importance of this complication and the urgency for immediate treatment was determined by the the degree of dependence of the coronary circulation on the grafted LIMA. Long term results of this procedure are not known because of the rare occurrence and limited number of reports of this complication of catheterization.

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An unusual case of stroke and atrial fibrillation in a 23 year-old

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Introduction and case report description: A 23-year-old man presented with difficulty speaking and right-sided weakness. He had a reported history of spinal muscle atrophy (SMA) type III diagnosed by muscle biopsy and clinical manifestations at an outside institution at age 4. He was not on any medication. He smoked marijuana occasionally, but denied other drug or alcohol use.
Description of the problem, procedures, techniques and/or equipment used: Head CT angiography showed a left middle cerebral artery (MCA) stroke. His physical exam was significant for truncal weakness, significant contractures at the elbows, and he was noted to walk on his toes which was lifelong (Figure 1, panel A and B). Heart exam was notable for a regular bradycardia. His electrocardiogram (ECG) demonstrated atrial fibrillation with a slow ventricular response (Figure 1, panel C), at times with regularization consistent with complete heart block and a junctional escape (Figure 1, panel D). Transthoracic echocardiogram showed a normal ejection fraction with no valvular disease. The MCA stroke was felt to be a thromboembolic complication of atrial fibrillation, eventhough CHA2DS2-VASc score was 0. He was treated upon presentation with thrombolytic therapy (tPA) with complete resolution of symptoms.
Questions, problems or possible differential diagnosis: The above mentioned findings on physical exam and cardiac manifestations of atrial fibrillation, stroke and conduction disease were more congruent with a neuromuscular disorder (NMD), specfically Emery Dreifuss muscular dystrophy (EDMD) rather than his reported history of SMA. EDMD is typically characterized by a classic triad of early joint contractures, slowly progressive muscle weakness and wasting, and cardiac disease, typically AV conduction abnormalities and atrial arrhythmias.
Answers and discussion: His ongoing evaluation and management included a cardiac MRI which showed biatrial enlargement and preserved left and right ventricular function without late gadolinium enhancement. He was anticoagulated with rivaroxaban and a TEE was initially performed to guide cardioversion. After we excluded left atrial appendage thrombus, two successive cardioversions were performed in the electrophysiology laboratory and were each accompanied by 5 second pauses with early recurrence of atrial fibrillation. While briefly in sinus rhythm, the AH interval was measured at 230 ms and the HV interval at 55 ms. Atrial electrograms were small and the atrial tissue in multiple sites was unable to be paced consistent with an atriopathy. Genetic testing showed mutations in

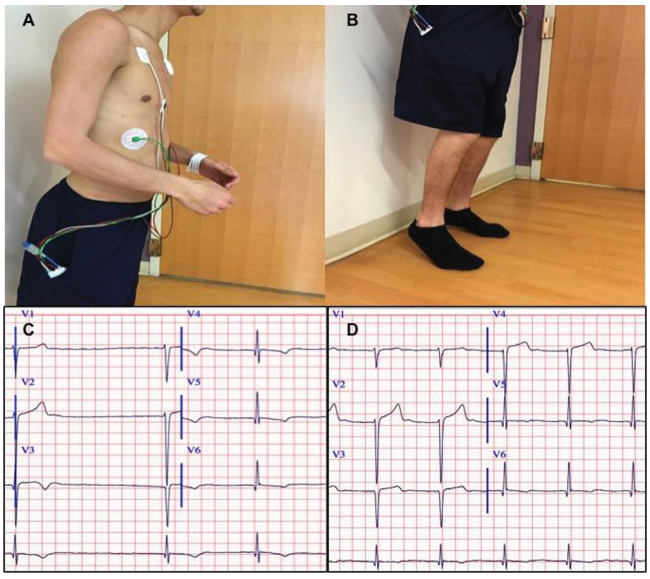


Figure 1

LMNA (c. 122G>A p. Arg41His), known to be mutants in EDMD and Lamin A/C. A single chamber ICD was implanted given the risk of ventricular arrhythmias in addition to conduction disease associated with EDMD with Lamin A/C mutation.
Conclusions and implications for clinical practice: This case highlights the electrophysiological manifestations in one of the neuromuscular disorders, specifically EDMD. A multidisciplinary approach for early diagnosis of these neuromuscular disorders, given the risk of sudden cardiac death and other findings like embolic stroke, is of paramount importance. Genetic testing is also important in cases of NMD. If testing reveals a LMNA mutation, there is a role for routine cardiac monitoring including ECG and echocardiogram given the high rate of cardiac manifestations and sudden cardiac death in EDMD.

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Double acute compartment syndrome in an extracorporeal membrane oxygenation (ECMO) assisted patient

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A 68-year-old man was transferred to our hospital with chest pain and diagnosis of established anterior myocardial infarction. Catheterism confirmed proximal anterior descendent artery occlusion and no-reflow phenomenon was observed in spite of implantation of a drug-eluting stent. Immediately after revascularization the patient suffered a cardiac arrest requiring advanced cardiopulmonary resuscitation and, after recovery of spontaneous circulation, vasoactive drugs and circulatory support with an intraaortic balloon pump. Despite of increasing dose of ionotropes, his haemodynamic worsened and a venoarterial extracorporeal membrane oxygenation (ECMO) was finally implanted. Left femoral artery was cannulated by arterial dissection with a 21 F cannula and a 16 F cannula was inserted in left femoral vein. A reperfusion distal cannula was added to optimize distal perfusion. Some hours after ECMO implantation a massive bleeding of the cannulation point required reintervention and countless sutures on the bled were necessary. Marked hemodinamical improvement was achieved with circulatory support but



Figure 1

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