and 24.0 (interquartile range 33) in the poor LDL group, with no significant difference (p=0.88). Furthermore, the clinical severity of ACS was worse in the good LDL group, as evidenced by a higher mean GRACE score (149.7 ± 41.1 vs. 137.1 ± 38.4, p=0.02). Among those with good LDC control, logistic regression analysis identified age (OR: 1.06, 95% CI: 1.01–1.11) and chronic kidney disease (OR: 9.14, 95% CI: 1.08–77.70) as potential predictors of severe ACS.

Discussion These findings suggest that low LDL cholesterol levels may not always correlate with reduced ASCVD risk, possibly due to underlying chronic diseases, increased inflammation, and the presence of highly atherogenic cholesterol particles. Crucially, however, the paradoxical association between good LDL control and poor clinical outcomes in ACS patients warrants further investigation.

Conclusion This study highlights the need for a deeper understanding of the mechanisms linking low LDL cholesterol to severe ACS. Furthermore, the findings raise concerns about the sufficiency of LDL as a sole target for ASCVD prevention, emphasizing the potential role of inflammation and other lipid parameters in patient risk stratification.

APCU 39 ORBITAL ATHERECTOMY IN A CALCIFIED RIGHT CORONARY ARTERY: RETRIEVAL OF THE ENTRAPPED CROWN

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Introduction The orbital atherectomy system (OAS) is a device used to ablate calcified coronary lesions during percutaneous coronary interventions (PCI). Optimal calcified plaque modification facilitates optimal stent placement and expansion. Nevertheless, its utility carries an uncommon risk of crown entrapment which may require a snare or an invasive surgery for its retrieval. This report describes a successful case of retrieving an entrapped diamond crown, which is detached from its wire, using a microcatheter.

Case Presentation A 60-year-old gentleman with a non-ST elevation myocardial infarction underwent coronary angioplasty for a severely calcified right coronary artery with diffuse stenosis. Amplatz Left 1 (AL 1) was engaged and the lesion was prepared using the Diamondback 360 Orbital Atherectomy System. The atherectomy was performed at 80,000 rpm with progressive escalation up to 120,000 rpm, at which point the system experienced an abrupt halt. Withdrawal of the crown results in a breakage that separates the crown from the atherectomy system leading to crown entrapment. Angiography revealed a sealed perforation in the target lesion. Due to the unavailability of a snare, a FineCross microcatheter was advanced over the OAS traction wire toward the trapped crown. The crown was successfully captured and retrieved as it adhered to the microcatheter. Final angiography showed a concealed perforation with TIMI 2 flow.

Discussion Coronary artery calcification emerges as a sequela in the genesis of atherosclerotic plaque. Its emergence is contributed by several factors such as advanced age, diabetes mellitus, hypertension, and chronic kidney disease. The two distinct types of calcifications are vascular intimal and medial calcifications, with the former being more commonly found. Owing to the risk of major adverse cardiovascular events contributed by the presence of moderate to severe coronary artery calcifications, an orbital atherectomy has been introduced as a calcified plaque modifying device before coronary stent implantation. Equipped with a diamond coated crown, it rotates over its guidewire in a centrifugal pattern, crushing the calcified plaque. Breakage of the device component accounts for 40% of the complications, entrapment of device occurs in 8% of cases, and breakage with subsequent entrapment of the device pieces represents 0.4% of its complications.

Conclusion Crown entrapment is a serious consequence of atherectomy that requires a suitable device for its retrieval.

APCU 40 DE WINTER SYNDROME: A RARE BUT FATAL ENTITY OFTEN MISSED

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Introduction The de Winter Syndrome is an electrocardiogram pattern that is highly suggestive of acute occlusion of left anterior descending artery (LAD). It often presents a diagnostic challenge due to its uncommon electrocardiogram pattern which may lead to a catastrophic complication such as cardiovascular mortality. Hence, we present a case of de Winter syndrome, in which angiogram confirmed an acute occlusion of left anterior descending artery.

Case Presentation A 41-year-old gentleman, active smoker with no known medical illness previously, presented to us with sudden onset central chest pain which occurred while resting. It was described as heaviness in nature and non-radiating, associated with giddiness and nausea during the event. His ECG showed upsloping ST segment depression at the J point at V2-V6 with peaked T wave. There is also a 1 mm ST elevation in lead aVR. Subsequent ECG in ED showed no evolvement into ST elevation pattern. His troponin level was raised. We diagnosed him as de Winter Syndrome and planned for primary percutaneous intervention (PCI). Unfortunately, we were unable to proceed with PCI due to service unavailability. Subsequently, ECG in the ward showed evolvement into Wellens pattern. Angiogram on day 5 of admission confirmed 90% occlusion of the mid LAD and a stent was successfully inserted into the mid LAD. Subsequently the patient was discharge well after cardiac rehabilitation in our cardiac care unit.

Discussion De Winter pattern accounts for about 2% of patient with LAD occlusion. The ECG patterns in de Winter syndrome are upsloping ST segment depression at the J point in lead V1–V6, peaked T waves and 1–2 mm ST elevation in lead aVR. A few theories proposed as underlying aetiology such as anatomical variant, existing collateral blood supply and lack of sarcolemma ATP-sensitive potassium (KATP) channels.

Conclusion De Winter syndrome is a rare ECG pattern that is equivalent to anterior ST elevation myocardial infarction. Though fatal, it is often missed. Early recognition is crucial to ensure successful early reperfusion strategy.

APCU 41 SPONTANEOUS HEMOPERICARDIUM POST THROMBOLYSIS IN ACUTE MYOCARDIAL INFARCTION: A RARE YET FATAL HEMORRHAGIC COMPLICATION

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Introduction Thrombolytic therapy using streptokinase has been widely used for acute myocardial infarction to achieve recanalization in infarct related artery. Bleeding into critical organ and space in a known complication. Inflammation of the pericardium in acute myocardial infarction increases the risk of bleeding after administration of thrombolytic agent.

Case Presentation A 68 year old gentleman with history of inferior myocardial infarction in 2014, dyslipidaemia who defaulted antiplatelet presented with severe central chest pain associated with palpitations, dyspnoea and diaphoresis of sudden onset. He denied having fever, chest trauma, anticoagulant use, or failure symptoms. Clinically, he was alert and hemodynamically stable. He saturated well under room air with clear heart sounds and bibasal crepitations. He had no other signs of heart failure. Initial ECG showed ST segment elevation at lead I, aVL and V2-V6 with pathological Q waves at inferior leads. Bedside echocardiography showed impaired left ventricular ejection fraction ~40% with akinetic anterolateral and inferior wall, no pericardial effusion seen. He was treated as acute anterolateral myocardial infarction KILLIP II and was given IVI Streptokinase 1.5mU. Post thrombolysis, his chest pain improved and ECG post thrombolysis showed >50% reduction of ST segment elevation. 22 hours post thrombolysis, he complained of recurrent chest pain, dyspnoea with desaturation episode. Clinically, he was alert, restless with moderate pulse volume and coolish peripheries. He was tachycardic and hypotensive requiring IVI Noradrenaline 1.2mcg/kg/min. Serial ECG and cardiac biomarkers did not show signs of reinfarction. Bedside echo showed global pericardial effusion with collapsed RV and distended IVC. Pericardial tapping done via apical approach aspirated 250cc blood. Post tapping, his heart rate normalise and inotropic support reduced 0.5mcg/kg/min. He developed left haemothorax which required left intrapleural catheter drainage. CT angiography showed no dissection or arterial bleed. He was hemodynamically stable on day two onwards with well expanded lung on day five. Repeated echo showed residual pericardial effusion of 0.7cm with LV clot. He was started on warfarin therapy and discharged well. Coronary angiography revealed triple vessel disease with LAD being the IRA.

Discussion Pericardial effusion is commonly seen in myocardial infarction and are generally asymptomatic. Expansion of effusion is self-limiting and no specific therapy is needed. Progressive accumulation of fluid within pericardial space will impede diastolic filling of the ventricle which causes tamponade.

Conclusion Hemopericardium in a rare yet major haemorrhagic and potentially fatal complication post thrombolysis. Unexplained hemodynamic instability post thrombolysis following large territory myocardial infarction warrants urgent echocardiography to exclude treatable condition. Timely bedside needle pericardiocentesis followed by drainage will render favourable outcome.

APCU 42 A CASE OF GIANT LEFT ATRIUM IN A PATIENT WITH POST-VALVE REPLACEMENT

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Introduction The giant left atrium (GLA) is a rare, yet significant clinical finding often associated with long-standing rheumatic mitral valve disease. It is characterized by a left atrium sized above 6 cm in diameter. GLA may lead to complications such as atrial fibrillation, thromboembolism, and compression of adjacent structures making it challenging to manage both medically and surgically.

Case Presentation A 62-year-old male with a history of chronic rheumatic heart disease and atrial fibrillation(AF) underwent mitral and aortic valve replacement in 1993 and had been on warfarin therapy. He presented with worsening heart failure symptoms which further deteriorated despite on optimal treatment. An echocardiographic revealed a markedly dilated left atrium, with an estimated left atrium volume index of 746 ml/m^2, left atrium diameter of 7.1 cm, and left ventricular ejection fraction (LVEF) of 30%. Meanwhile, the prosthetic mitral valve mean PG, DVi, and MV EOA were 6 mmHg, 1.44, and 3.69 cm^2, respectively. On the other hand, the prosthetic aortic valve mean PG, DVi, and EOA were 18 mmHg, 0.41, and 2.65 cm^2, respectively. Despite having a therapeutic INR, the patient developed severe respiratory distress and reduced consciousness, requiring intubation. A CT scan of the brain revealed a large stroke in the territories of the middle cerebral artery (MCA) and posterior cerebral artery (PCA), with haemorrhagic transformation. However, he succumbed to complications of a stroke.

Discussion Patients who undergone valve replacement expected to have negative left atrial remodelling. However, the giant left atrium remain unchanged in this patient, which were attributed to poor LVEF, atrial fibrillation and also undiagnosed low flow low gradient prosthetic mismatch. Low flow low gradient prosthetic valve mismatch is difficult to evaluate due to atrial fibrillation and reduced LVEF which affect the accurate measurements of prosthetic valve functions. Therefore, its difficult to assess the severity of valve dysfunction. This likely contributed to ongoing left atrial remodelling and heart failure. Advanced imaging techniques needed to diagnose and address the valve dysfunction and prevent progression of heart failure and complications.

Conclusion This case highlights the challenges of managing giant left atrium in patients with rheumatic heart disease who have undergone dual valve replacement. It emphasizes the