Cardiovascular Medicine

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Abstract Session: Clinical Cases I

O01–O05

Joint Annual Meeting 2019 of the Swiss Society of Cardiology and the Swiss Society of Cardiac Surgery

O01

Ineffective shock delivery during swimming in salt water

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Case: A 17-year old competitive swimmer with arrhythmogenic cardiomyopathy and successful implantation of a subcutaneous implantable cardioverter-defibrillator (S-ICD) 2 months ago experienced two appropriate shocks during swimming in salt water. Clinical examination showed a localized reversible skin erythema following the S-ICD lead (Figures O01-1 and 2, arrows).

Electrogram review revealed a sustained ventricular tachycardia, treated with a very low impedance shock (24Ω), which was ineffective and induced ventricular fibrillation. A second shock (24Ω) fortunately converted him to sinus rhythm.

Subsequent in-hospital defibrillation testing confirmed appropriate device function with a normal shock impedance (57Ω). No malfunction was detected, but it was concluded that salt water can significantly lower shock impedance, especially in patients with low amounts of body fat, since parts of the shock current will flow into surrounding salt water through the skin, explaining the irritation observed in that area. Due to this shunting-effect, shocks can be ineffective and potentially lethal.

Figure: O01-1. Localized thoracic erythema following the S-ICD lead (arrow).

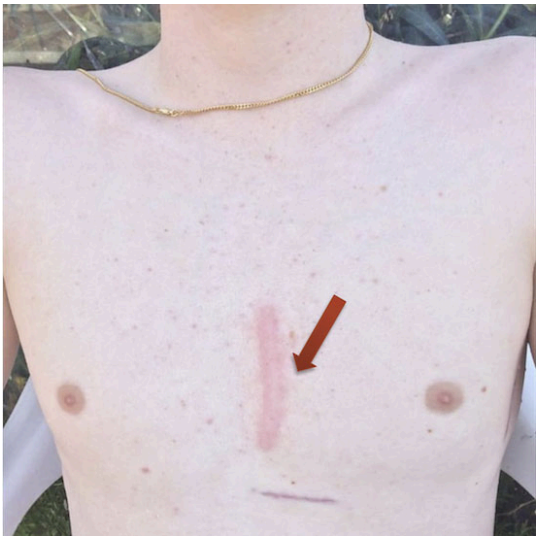
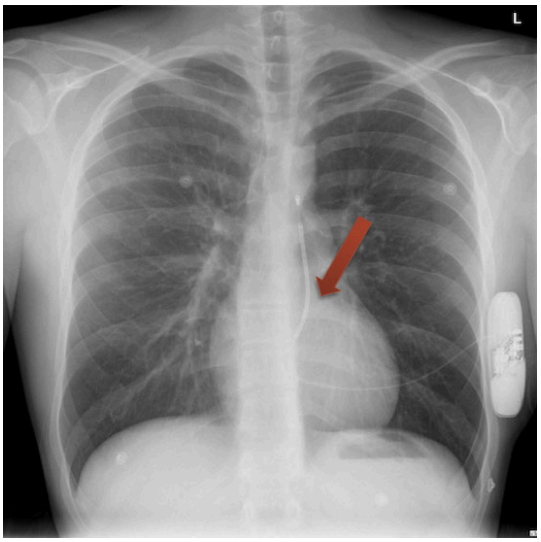


Figure: O01-2. Chest x-ray (anterior-posterior) showing the position of the subcutaneous ICD and ICD lead



O02

"SCN5A Overlap Syndromes": an unsolved Rubik's cube

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Introduction: SCN5A Overlap Syndromes represent a novel clinical entity characterised by the expression of either mixed phenotypes resulting from the simultaneous manifestation of different SCN5A-related arrhythmic syndromes or of isolated phenotypes differing among the mutation carriers (MCs) of the same SCN5A mutation. We report a clinical case of SCN5A Overlap Syndrome with a new variable phenotypic expression associated to the mutation SCN5A-G1408R.

Methods: A 20-years old female patient was addressed at our institution to investigate recurring vertigo and

Figure: O02-1. Uncle's ECG showing spontaneous type I ST-segment elevation with negative T waves

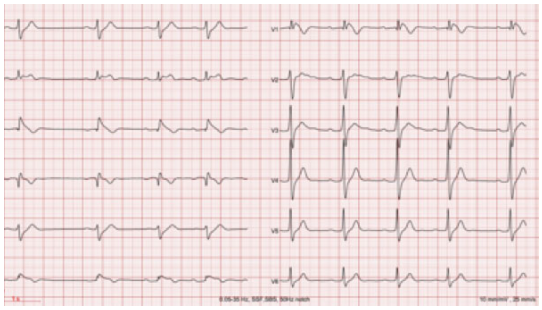
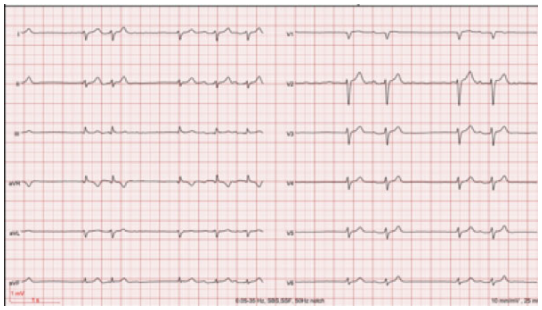


Figure: O02-2. IP's ECG showing alternating junctional and sinus rhythm with first degree AV block



lipothymic episodes. The patient had no previous medical history. The family background included 15 members among whom a maternal uncle has been recently diagnosed with Brugada Syndrome (BS) due to the serendipitous observation of spontaneous type I ST-segment elevation on ECG during a routine medical screening. Family members underwent a cardiac work-up including ECG, exercise stress test, 24-h ECG, electrophysiological (EEP) study and/or sodium channel blockage challenge. A genetic test has been performed (till present) in the index patient (IP) and in her maternal uncle.

Results: We identified in the IP and in her maternal uncle the missense mutation SCN5A-G1408R associated to a variable phenotypic expression. The maternal uncle showed isolated BS with spontaneous type I ST-segment elevation on ECG (Figure O02-1). Due to the absence of cardiovascular symptoms, the patient is clinically followed-up on a yearly basis. The IP expressed a severe juvenile form of sick sinus syndrome (SSS) with inappropriate sinus bradycardia and first degree atrio-ventricular block (Figure O02-2) associated to BS with typical type I ECG appearing after drug challenge. Due to symptom persistence during efforts and to the further development of nocturnal palpitations, despite the absence of inducible ventricular arrhythmias at the EEP study, the IP opted for the implantation of a double chamber defibrillator.

Conclusions: We report, for the first time, a new variable phenotypic expression among MCs of the same SCN5A-G1408R mutation manifesting as either isolated BS or as a severe juvenile form of SSS associated to BS. Our case represents an example of SCN5A Overlap Syndrome and raises further unanswered questions about the causative role of SCN5A mutations in determining phenotypic expression, suggesting rather the contribution of « modifying risk factors» in modulating the susceptibility to the same SCN5A mutation effects.

O03

Successful ablation of incessant premature ventricular complex through retrograde transvenous ethanol infusion

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Background: Ablation of intramyocardial or epicardial premature ventricular complexes (PVC) remains challenging. Recently, ethanol infusion, using a retrograde coronary sinus (CS) venous approach, was reported as a method for ventricular tachycardia (VT) ablation. Herein, we report a case of refractory epicardial PVC successfully treated using a retrograde CS venous ethanol infusion.

Case description: A 24-year old male patient with monomorphic PVCs since 2012 (30% burden) was referred to our center for ablation because of progressive left ventricular (LV) dysfunction after an unsuccessful retro-aortic attempt. The ECG (Fig. O03-1A) suggested an epicardial location (QRS duration 200 ms, pseudo-delta wave in D1 and V1) from the medio-infero-lateral LV. Using a transseptal access, the PVC was localized between the mid-ventricular and basal third of the infero-lateral LV. Despite a local electrogram (EGM) timing at 0 ms from QRS onset, endocardial ablation only suppressed transiently the PVC. The CS anatomy and its side branches were reconstructed using an electroanatomical mapping system (Carto3). A high lateral CS vein directed towards the PVC focus was identified and occluded with the ablation catheter (AC, SmartTouch SF). Proper localization was confirmed by infusion of 5°C saline that briefly suppressed the PVC. Following the injection of 1 ml of 96% ethanol through the AC, a non-sustained VT occurred followed by PVC suppression (Fig. O03-1B) with recurrence 10-min later. A coronary guiding catheter was then advanced into the target vein with guidewires for subselective catheterization closer to the arrhythmic focus. A 1.5-mm over-the-wire balloon was inflated to prevent backflow (Fig. O03-1C) and 1 ml of ethanol was infused with a 5-min venous occlusion, followed by sustained disappearance of the PVC.

Discussion: Retrograde venous ethanol injection is a promising technique to target VT and PVC of intramyocardial and epicardial origin. Unlike intracoronary ethanol infusion, retrograde venous ethanol infusion preferentially damages the epicardium. Cold saline injection is a useful method to confirm adequate localization before ethanol injection. The ideal ethanol volume needs, however, to be further clarified to optimize balance between efficacy and myocardial damage. In our case, follow-up CMR is planned at 3 months after the procedure to characterize myocardial injury distribution.

Figure: O03-1, A: 12 leads ECG with PVC morphology; B: VT followed by sinus rythm after ethanol infusion.

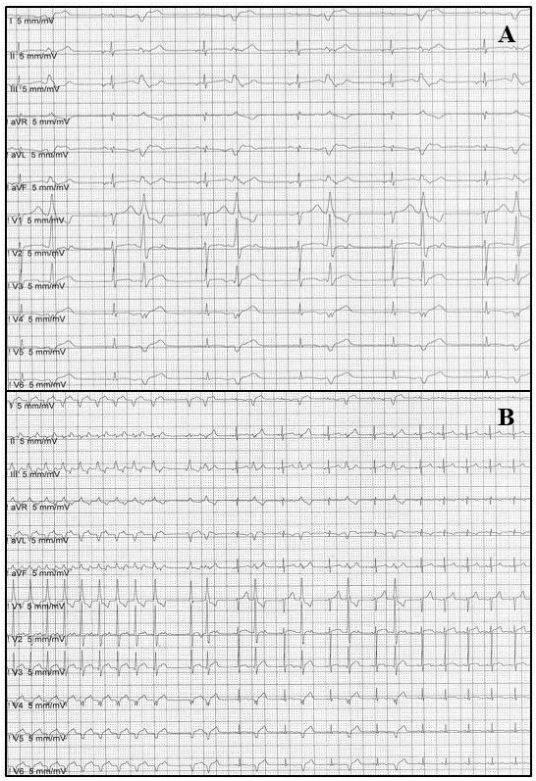
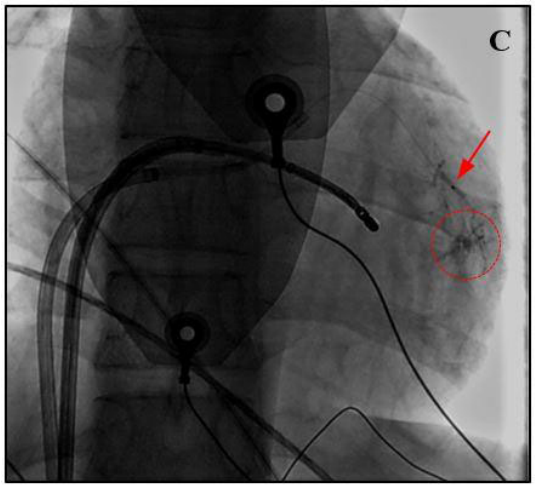


Figure: O03-1, C: myocardial contrast staining (circle) and inflated balloon to prevent backflow (arrow).



O04

An elegant way to fix pacemaker lead perforation - a heart team experience

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Introduction: Lead perforation is a known complication in cardiac device therapy. Therapeutic management includes transvenous removal of the old and re-implanting a new electrode, or even cardiac surgery via sternotomy or thoracotomy. Here, we report a case where an atrial pacemaker (PM) lead perforation was elegantly fixed minimal-invasively by a thoracoscopic procedure without the need for lead replacement.

Case Description: A 83-year-old female patient was referred to our institution with pericardial tamponade following implantation of a two-chamber pacemaker (PM). A chest CT scan revealed a perforation of the atrial electrode through the right atrial wall, while PM device interrogation demonstrated stable measurements. Despite emergency subxyphoidal pericardiocentesis, the patient deteriorated further and hence a decision was made to perform a thoracoscopy in this high-risk patient and aim for epicardial control of the bleeding site. She underwent salvage rightsided thoracoscopy with single-lung ventilation. The pericardium was opened and extended cranially for better exposure. A large pericardial effusion could be drained, leading to a rapid hemodynamic improvement. Ultimately, the tip of the electrode screw perforating through the right atrial wall as the source of the active bleeding was identified. Using a Teflon-felt pledged 4-0 Prolene mattress suture, which was further secured by a clip and fibrin adhesive (BioGlue®), the bleeding was successfully controlled epicardially. No conversion to sternotomy was necessary. Given a difficult initial implantation and stable PM measurements, no attempts were undertaken to remove and replace the perforating electrode. Intraoperative transoesophageal echocardiography confirmed no further pericardial effusion and a normal cardiac function. The hemodynamically stable patient was extubated in the operating room and transferred to the intensive care unit for one overnight stay. She was discharged from hospital 6 days after the intervention. PM interrogation as well as an echocardiography prior to discharge showed normal and stable results.

Conclusion: Epicardial bleeding control via right-sided thoracoscopy is an elegant and safe way to handle lead perforation after cardiac device implantation, especially in frail patients with limited treatment options. In addition, as in our case, re-implantation might not be necessary.

O05

Noninfective endocarditis ulceropolyposa of the aortic valve in a 28y old male

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A 28y old male experienced fever and acute embolic closure of the left popliteal artery after a game of football, necessitating emergency intervention. Displaying splinter hemorrhages, but no signs of infection, empiric antibiotic treatment was initiated for 4 weeks, as well as phenprocoumon in therapeutic dose. Diagnostic workup showed a visible vegetation on the aortic valve in echocardiography.

Six weeks later, due to sudden progressive growth and moderate aortic insufficiency the indication for urgent aortic valve replacement was given by the interdisciplinary

Figure: O05-2. Histological analysis showing fibrinous material on the native leaflet, with inflammatory cells

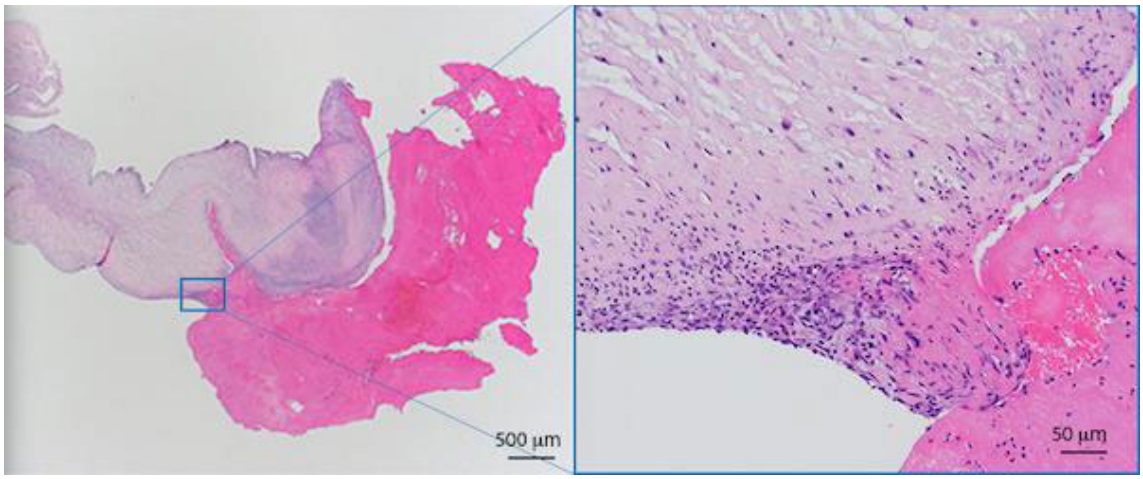
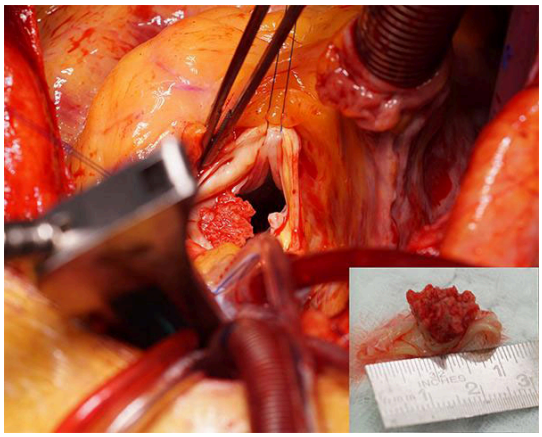


Figure: O05-1. Intraoperative view of the aortic valve with superposed thrombotic material. Inset: Close up view.



Heart Team. Intraoperatively, the valve was tricuspid with a large vegetation fusing two leaflets, creating a functionally bicuspid valve. A mechanical aortic valve was implanted (Medtronic Open Pivot™AP 360®, 28mm). The histopathological workup showed no identification of pathogens or organisms. Light microscopy demonstrated a destructive, ulceropolyposis of the native valve combined with a florid inflammation. The patient recuperated well and was discharged after a short hospitalization. Hematological investigation revealed a hereditary heterozygous prothrombin-mutation (G20210A-Mutation).

Seven months later the patient presented himself to the emergency ward with typical symptoms of instable angina pectoris after cycling. High-sensitive Troponin T was elevated to a maximum value of 1521 ng/L (0-14 ng/L) and CK-MB to 76 µg/L (0-5 µg/L). The coronary angiogram showed multiple coronary embolisms with no signs of sclerosis. Echocardiography demonstrated the perfectly functioning mechanical valve without signs of adhering material. No intervention was performed and the patient was monitored on the intensive care unit. Reanalysis of the hematological bloodwork and coagulation factors showed a Factor-VII deficiency leading to false high INR values. Anticoagulation monitoring was changed to daily Factor II-analyis with target values of 20-25%. The patient was discharged after a short hospitalization.

Conclusion: In Patients with rare hereditary coagulation disorders, anti-coagulation therapy monitoring can be changed to Factor-II analysis in order to prevent possible complications.