CARDIOLOGY UPDATES NEWSLETTER

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IN THIS ISSUE

Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you "Cardiology updates" which contains latest and interesting news in the field of Cardiology

Assessment of the optimal management of patients with VonWillebrand disease (VWD) and haemophilia A for patients undergoing catheter ablation procedures

Most common types of bleeding disorders are VonWillebrand disease (VWD) and haemophilia A (HA). However, most patients with VonWillebrand disease are not aware of the disease because they have mild or shows no symptoms. VonWillebrand disease is usually inherited from parents and caused by an abnormal gene that controls the von Willebrand factor, which is a protein that plays a key role in blood clotting. Approximately 1 in 1000 (0.1%) is the prevalence of symptomatic VonWillebrand disease.[1] Three main types of VWD are type I, type II and Type III. Acquired von Willebrand occurs when secondary processes cause a functional impairment of von Willebrand factor by lowering the available quantity or by interfering with the physiological haemostasis pathway.[2]

On the other hand, haemophilia A is factor VIII deficient, X-linked recessive, congenital disorder that causes spontaneous bleeding. Haemophilia A is mostly prevalent in male and is found approximately 1 in 5000 (0.02%) and is classifies into three forms based on residual factor VIII activity: Severe (FVIIIa < 1%, <1 IU/dl), Moderate (FVIIIa 1–5%, 1–5 IU/dl), or Mild (FVIIIa < 5–40%, 6 IU/dl to <40 IU/dl)

Life expectancy in both VonWillebrand disease (VWD) and haemophilia A is almost normal with an increased incidence of cardiovascular diseases and cardiac arrhythmias. Patients with VonWillebrand disease and haemophilia are still a rare and complex population in catheter ablation (CA) procedures as they tend to have a higher risk of bleeding complications compared to other patients.[1]

Feher M aimed to analyse the optimal management of patients with VonWillebrand disease and haemophilia referred for catheter ablation procedures. Patients with VonWillebrand disease or haemophilia undergoing CA procedures were analysed at two centres and clotting factors were administered in conjunction with hemostaseological recommendations. Catheter ablation was performed as per the institutional standard.

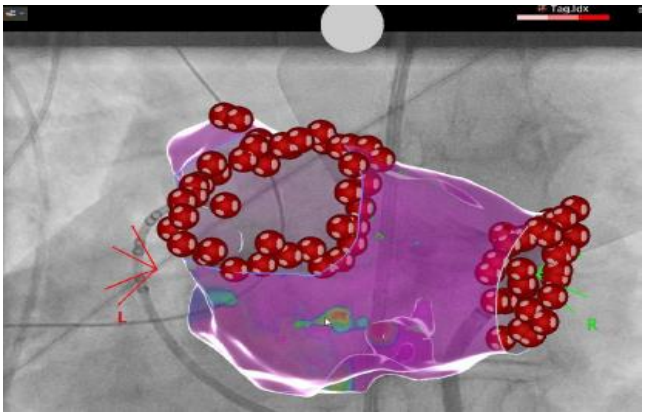

Figure 1:Electroanatomical reconstruction of a left atrium utilizing a 3D mapping system.

IV unfractionated heparin (UFH) was given with respect to the activated clotting time (ACT), during the procedure. Primary endpoints of the study were feasibility of the procedure, bleeding complications, and thromboembolic events during the procedure while Secondary endpoints were bleeding complications and thromboembolic events up to one year after catheter ablation. 7 patients (three VWD Type I, one VWD Type IIa, three HA) underwent 10 catheter ablation procedures. Patients with VonWillebrand disease received 2000–3000 IE Wilate IV and catheter ablation 30 to 45 min before ablation. Patients with haemophilia A received 2000–3000 IE factor VIII before the procedure. In RF ablation a 3D cardiac mapping system was used for 3 used for the 3D reconstruction(fig 1). All patients undergoing Pulmonary Vein Isolation received unfractionated heparin OF cumulative dose 9000–18,000 IE with a target ACT of >300 s and were started on oral anticoagulation (OAC) 12 h after ablation. For 4 weeks after the ablation of left-sided PVCs, 2 patients received aspirin. No bleeding complications or thromboembolic events were observed and reported. During a follow-up of one year, one case of gastrointestinal bleeding occurred following OAC withdrawal after LAA occlusion.

CONCLUSION: This study highlighted the safety and feasibility of catheter ablation in patients with VonWillebrand disease and haemophilia A

References

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Comparison of ventricular interaction during exercise in patients with HFpEF and chronic thromboembolic pulmonary hypertension with healthy controls

Direct force transmission between right and left ventricles, originates from the cross-talk brought about by the interventricular septum, shared myocardial fibres, and enclosing pericardium.[1] ventricular-ventricular interaction is defined as the impact that the function, geometry, and events in one ventricle have on the other.[2] Ventricular interactions are dynamic and can be seen throughout the cardiac cycle. In mediating ventricular interactions, interventricular septum plays an important role, thus, ventricular interdependence can be easily examined by evaluating the septal motion through cardiac imaging.[1] Although the physiology of ventricular interactions between the left and right ventricle are studied, but there are less data demonstrating the imaging of ventricular-ventricular interactions.[2]Ventricular interdependence has mainly been analysed in the context of right ventricular pressure or volume overload. Increased role of ventricular interdependence in patients with heart failure is observed [1]

Claeys M etal aimed to study and compare the ventricular interaction during exercise in patients with heart failure with preserved ejection fraction (HFpEF) and patients with chronic thromboembolic pulmonary hypertension (CTEPH) with healthy volunteers. Forty-six participants were enrolled, of which 10 are included in controls, 19 patients in chronic thromboembolic pulmonary hypertension group, and 17 patients in preserved ejection fraction patients underwent cardiac magnetic resonance imaging during exercise. Ventricular interaction was evaluated through analysis of the septal curvature (SC) of a mid-ventricular short-axis slice at end-diastole, end-systole, and early-diastole (fig 2)

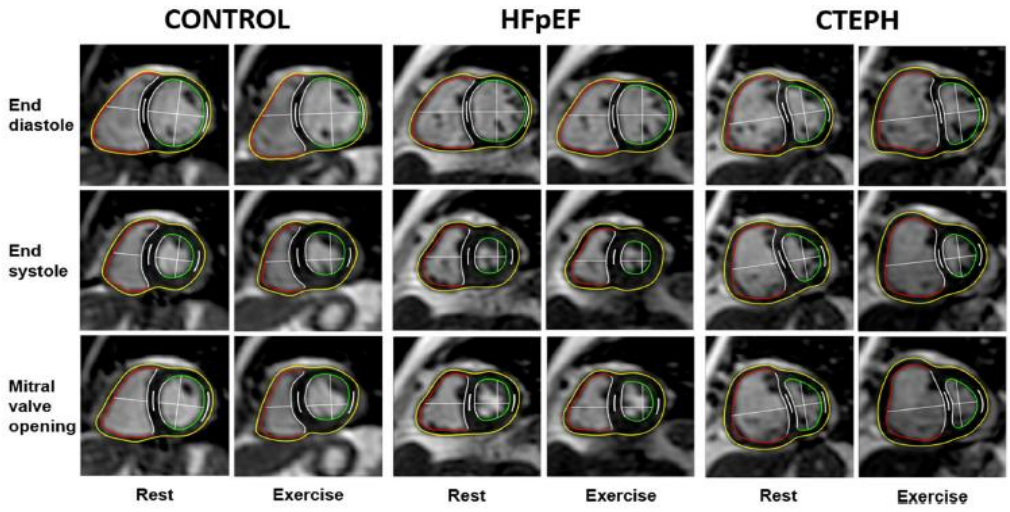

Figure 2: Illustrative mid-ventricular slice of control subject (A), HFpEF (B), and CTEPH (C) patient at ED (upper row), ES (middle row), MVO (lower row) both at rest (left column) and exercise (right column) with traced epicardial (yellow), RV endocardial (red), and LV endocardial (green) border

Exercise amplified ventricular interaction was found in patients with chronic thromboembolic pulmonary hypertension and to a lesser extent in patients with preserved ejection fraction. In early-diastole, adverse interaction was most profound and most pronounced in chronic thromboembolic pulmonary hypertension patients due to a disproportionate increase right ventricular afterload and diastolic pericardial restraint during exercise. Inspiration enhanced diastolic interdependence was found in patients with chronic thromboembolic pulmonary hypertension and preserved ejection fraction. During rest and exercise, septal curvature was strongly correlated with right ventricular volumes and pulmonary artery pressures

This study concluded that Exercise amplifies adverse right–left ventricular interactions in patients with chronic thromboembolic pulmonary hypertension and in isolated post-capillary patients with preserved ejection fraction. Analysing ventricular interaction with exercise cardiac magnetic resonance might be valuable option from a diagnostic or therapeutic perspective

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Effect of location and size of previous myocardial infarction on LA function

After acute myocardial infarction, impairment of left ventricular systolic and diastolic function is observed. There is also a possibility of left atrial (LA) dysfunction. However, Impairment in the function of LA is related to myocardial fibrosis in left ventricular infarction and diastolic filling transferring stress from the left ventricle (LV) to left atrial. Function of left atrium is to regulate the left ventricular filling and to maintain the normal heart volume through the LA reservoir, conduit and booster pump function.Though, Percutaneous coronary intervention (PCI) has found to improve outcomes in MI patient, due to poor prognosis some patients still suffer adverse cardiovascular events, such as arrhythmias and heart failure. After PCI, during the period of recovery from myocardial. infarction, the infarct area gradually decreases, producing a degree of LA and LV recovery. Later, Necrotic tissue begins to get absorbed after 1–2 weeks of acute MI and fibrosis occurs, forming a scar within 6–8 weeks. After healing from the injury, final infarct size represents replacement fibrosis.

This retrospective study aimed to investigate the effect of location and size of previous myocardial infarction (MI) on LA function using Cardiovascular magnetic resonance feature tracking. 129 participants were enrolled, where 42 patients were already diagnosed with anterior MI and 42 as non-anterior MI and 47 as healthy controls. Late-gadolinium enhancement (LGE) imaging was used to locate the infarct and its size and were assigned and quantified. Function of LA was evaluated using CMR-FT in 2- and 4-chamber cine images, including LA reservoir, conduit and booster pump function.

Result of this study has found that Anterior MI patients had impaired LA reservoir function, conduit function and booster pump function when compared with controls. (fig 3) At the same time, Non-anterior MI patients had impaired LA strain but preserved LAEFs. No significant difference in the LA morphology and function between the anterior and non-anterior wall groups were observed.

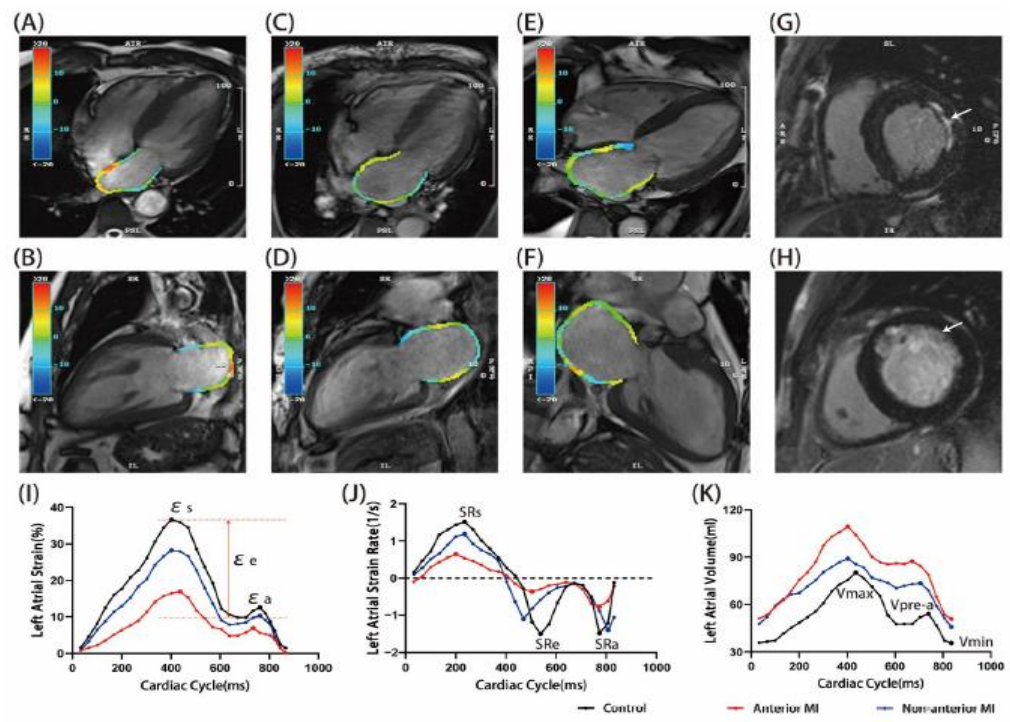

Fig:3 Typical examples of control ((**A**,**B**) and black line), non-anterior MI ((**C**,**D**,**G**) and blue line) and anterior MI ((**E**,**F**,**H**) and red line). (**A**,**C**,**E**) 4-chamber view of LA systolic phase, (**B,D**,**F**) 2-chamber view of LA systolic phase (the LA color varies strain parameters). (**G**) = lateral MI, (**H**) =anterior MI. (**I**–**K**) Curves showing LA strain, strain rate and volume in 25 phases, respectively.

According to the Stratification analysis, LA volumes and LAEFs were unchanged in patients with MI size _< 15% compared with controls. Increased LAVI pre-a, LAVI min and reduced LA total emptying fraction, and LAAEF were found in patients with MI size > 15% compared with the MI size _< 15% group

Conclusion: Location of myocardial infarction doesn't affect the morphology and function of the left atrium. Patients having MI size > 15% shows more pronounced post-infarction LA remodelling and dysfunction compared to patients with MI size _< 15%

Reference: Zhang H, Tian Z, Huo H, Li H, Liu H, Hou Y, Dai X, Liu T, Jin S. Effect of Infarct Location and Size on Left Atrial Function: A Cardiovascular Magnetic Resonance Feature Tracking Study. Journal of Clinical Medicine. 2022 Nov 24;11(23):6938.

Overt hypothyroidism complicated by complete heart block and severe hyperlipidaemia. A case report

Pathologic disorders that impair the structure or function of the conduction system leads to complete AV block which is mostly seen in conditions such as ischemic heart disease or cardiomyopathies, degenerative disorders characterized by escalating conduction system fibrosis, and medications that interfere with AV conduction. Thyroid conditions such as Hyperkalemia can also cause AV blockage. On the other hand, Hypothyroidism can also cause a variety of diseases, including cardiovascular disorders and also infrequently results in a complete atrioventricular block. Few electrographic abnormalities linked to hypothyroidism include sinus bradycardia, flattened P waves, flat or inverted T waves, low voltage, and delayed intraventricular conduction

A 19-year-old female was presented with a history of fatigue and facial swelling, constipation and dizziness. Patient medical history revealed a long-standing history of menstrual irregularities. No known chronic disease as well as a family history of cardiac disease was found. On admission, heart rate was 45 beats per minute and was alert with a swollen face, and swollen thyroid could not be found on the neck. Systemic reviews showed two bi-lateral non-pitting lower limb oedema. ECG report showed complete atrio-ventricular block, atrioventricular dissociation, and a maximum heart rate of 42 beats/min (see Fig. 1)

Figure 4: Electrocardiogram showing complete heart block with heart rate of 40 bpm

Laboratory tests showed severe elevated Thyroid Stimulating Hormone levels (213 mIU/ml), and LDL levels (198 mg/dl) for which 50mg of levothyroxine was given.1 month later, follow-up was done which revealed no further complaint with menstrual cycle normalization. ECG showed improved heart rate without any atrioventricular blockage and marked reduction in TSH and lipid profiles. Later, levothyroxine was reduced to 25mg with further follow-up.

This case report demonstrated about the case of severe dyslipidaemia and a complete heart blockage caused due to thyroid dysfunction. This case also highlighted the importance of identifying reversible causes of AV block prior to unnecessary pacemaker placement and statin medication

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