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CARDIOMAG NEWSLETTER

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IN THIS ISSUE

Dear Doctor,

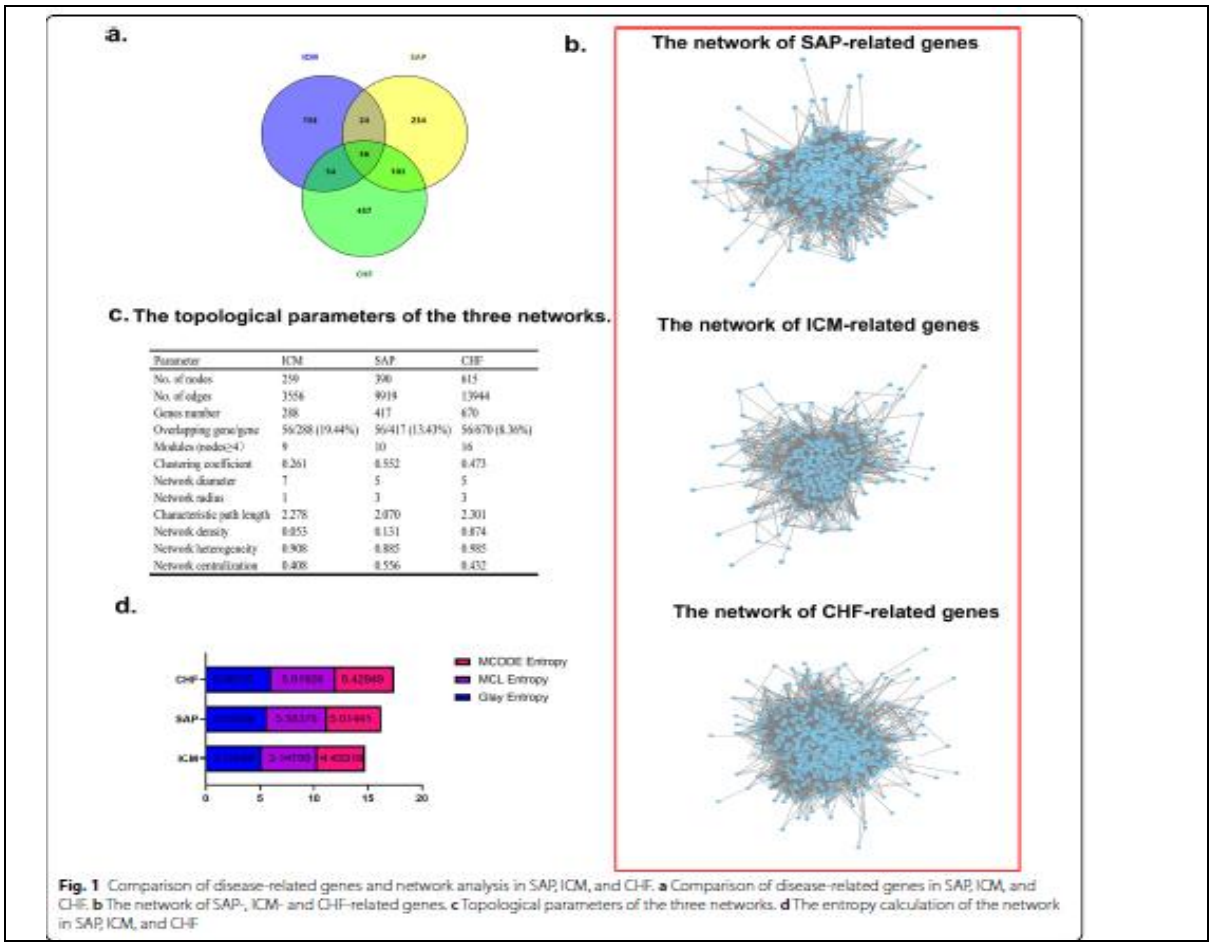
Greetings from Micro Labs Limited. We are happy to bring you "Cardio Mag" which contains latest and interesting news in the field of Cardiology

Identification of relationship among stable angina pectoris, ischemic cardiomyopathy and chronic heart failure based on restructured modules methods

Disease–disease relationships play an important role in the identification of diseases and its precision treatment. That is why, understanding the associations of diseases increases the knowledge of disease relationships, leading to further improvement in diagnosis, prognosis, and treatment. Complex diseases such as stable angina pectoris, ischemic cardiomyopathy (ICM) and chronic heart failure (CHF) are caused by the interactions of environmental and genetic factors. Stable angina pectoris is a chronic condition considered as the first warning signs of underlying coronary artery disease, with mortality rate ranging between 1.2 and 2.4%

However, molecular relationship among stable angina pectoris (SAP), ischemic cardiomyopathy (ICM) and chronic heart failure (CHF) is still unclear. Researchers in this study integrated the data and developed paired disease progression modules (PDPMs) to identify the relationship among stable angina pectoris, ischemic cardiomyopathy and chronic heart failure based on *K*-value calculation methods. Also, to verify the relationship between this disease in PDPMs, enrichment analysis, literature validation and structural variation (SV) were conducted. Among stable angina pectoris, ischemic cardiomyopathy and chronic heart failure,16 PDPMs were found with $K > 0.3777$, in which 6 pairs in stable angina pectoris – ischemic cardiomyopathy, 5 pairs for both ischemic cardiomyopathy- chronic heart failure and stable angina pectoris–chronic heart failure.

Most closely related with smallest average *K*-value ($K = 0.3899$) was Stable angina pectoris–ischemic cardiomyopathy while the maximum is stable angina pectoris–chronic heart failure ($K = 0.4006$). It was found that inflammatory response was found in all stages of stable angina pectoris-ischemic cardiomyopathy-chronic heart failure but stable angina pectoris-ischemic cardiomyopathy was uniquely involved in fibrosis, and genes were related in affecting the upstream of PI3K–Akt signalling pathway. Of 11 genes, 4 genes (FLT1, KDR, ANGPT2 and PGF) were found in SAP–ICM–CHF related to angiogenesis in HIF-1 signalling pathway (fig 1). Also it was identified that 62.96% SVs were protein deletion in SAP–ICM–CHF, and 53.85% SVs were protein replication in SAP–ICM, while ICM–CHF genes were affected by protein deletion



Parameter	ICM	SAP	CHF
No. of nodes	259	390	815
No. of edges	3556	8919	13944
Genes number	288	417	670
Overlapping gene/gene	56/288 (19.44%)	56/417 (13.43%)	56/670 (8.36%)
Modules (nodes≥4)	9	10	16
Clustering coefficient	0.261	0.552	0.473
Network diameter	7	5	5
Network radius	1	3	3
Characteristic path length	2.278	2.070	2.301
Network density	0.053	0.131	0.074
Network heterogeneity	0.908	0.885	0.885
Network centralization	0.408	0.556	0.432

Fig. 1 Comparison of disease-related genes and network analysis in SAP, ICM, and CHF. **a** Comparison of disease-related genes in SAP, ICM, and CHF. **b** The network of SAP-, ICM- and CHF-related genes. **c** Topological parameters of the three networks. **d** The entropy calculation of the network in SAP, ICM, and CHF

CONCLUSION: This study concluded that paired disease progression modules analysis along with genomic structural variation provides new insights for determining target associations contributing to disease progression. Finding of this study also revealed that inflammation and angiogenesis may be important links for the progression of stable angina pectoris (SAP), ischemic cardiomyopathy (ICM) and chronic heart failure

Reference: Chen L, etal. Modular networks and genomic variation during progression from stable angina pectoris through ischemic cardiomyopathy to chronic heart failure. Mol Med. 2022 Nov 26;28(1):140

Evaluation of ventricular interaction during exercise in patients with heart failure with preserved ejection fraction (HFpEF) and patients with chronic thromboembolic pulmonary hypertension

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)

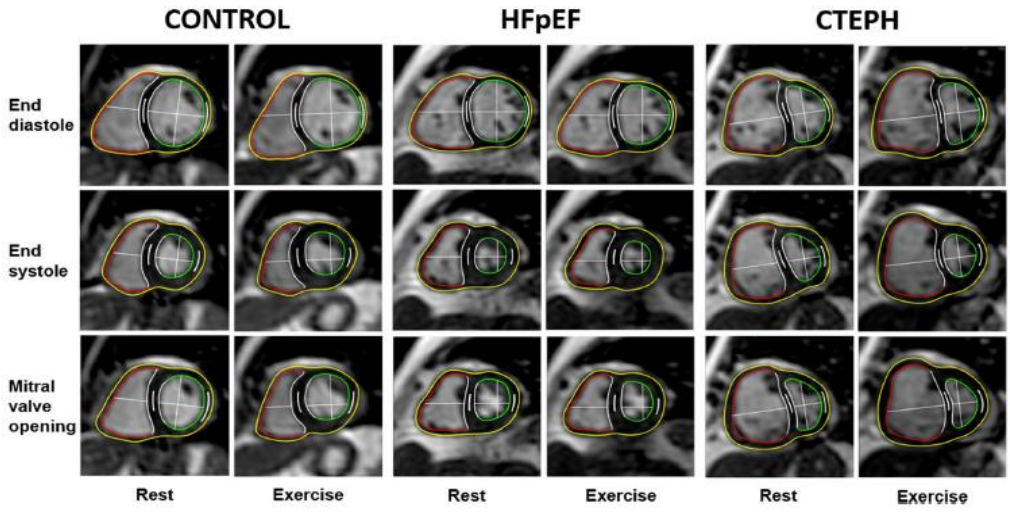


Figure 2: Illustrative mid-ventricular slice of control subject (A), HFpEF (B), and CTEPH (C) patient at ED (upper row), ES (middle row), MVO (lower row) both at rest (left column) and exercise (right column) with traced epicardial (yellow), RV endocardial (red), and LV endocardial (green) border

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

References

1. Claeys M,etal. Dynamic aspects of ventricular interaction during exercise in HFpEF and in pre-capillary pulmonary hypertension. ESC Heart Failure. 2022 Nov 24
2. Friedberg MK. Imaging Right-Left Ventricular Interactions. JACC Cardiovasc Imaging. 2018 May;11(5):755-771

Investigation of the effect of location and size of previous myocardial infarction (MI) on Left Atrial Function using Cardiovascular Magnetic Resonance Feature Tracking Study

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.

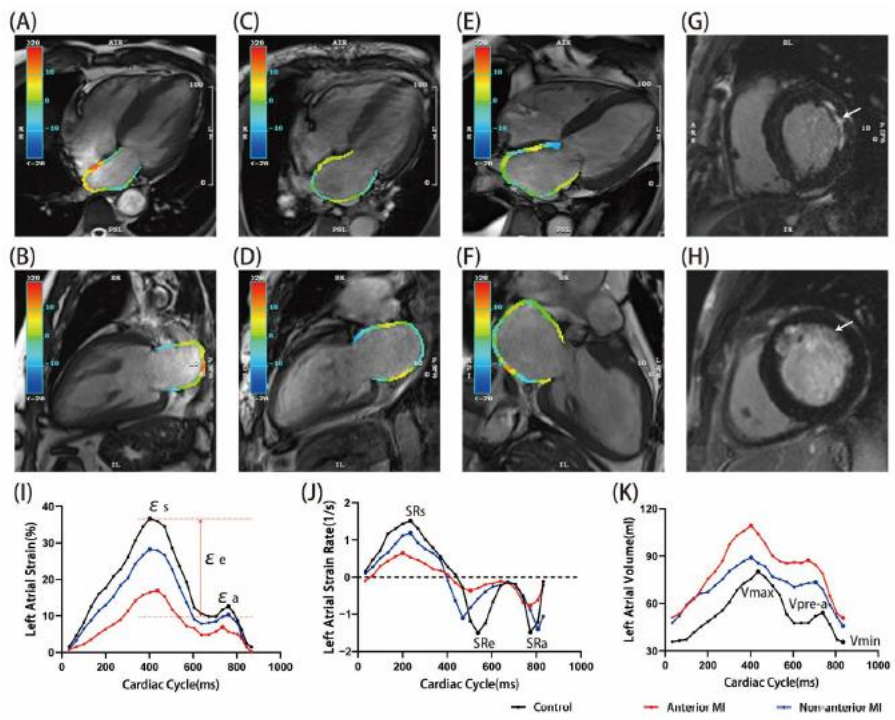


Fig:3 Typical examples of control ((**A**,**B**) and black line), non-anterior MI ((**C**,**D**,**G**) and blue line) and anterior MI ((**E**,**F**,**H**) and red line). (**A**,**C**,**E**) 4-chamber view of LA systolic phase, (**B,D**,**F**) 2-chamber view of LA systolic phase (the LA color varies strain 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.

Clinical presentation of Fulminant pseudomonas pneumonia after cardiac surgery: A Case Report

Hospital-acquired pneumonia develops at least 48 hours after admission to the hospital. Ventilator-associated pneumonia (VAP), accounts for about 80% of Hospital-acquired pneumonias develops within 48 hours of intubation. After cardiac surgery, common primary infectious complication observed is postoperative pneumonia which is associated with increased medical expenses, significant morbidity, mortality. *P.aeruginosa* is considered as the main causal pathogens of this type of pneumonia. Mechanically ventilated patients with nosocomial P. Aeruginosa infection have days-long colonization that slowly develops to pulmonary infiltrates. Because of which, pseudomonas Ventilator-associated pneumonia infections after cardiac surgery typically take days to develop and require long hospital stay. This case report describes about the case of fulminant ventilator-associated pneumonia post CABG surgery, where P. aeruginosa was isolated after the lung autopsy of the patient.

A 55-year-old man presented with the history of dyspnoea on exertion from a few months was admitted for elective CABG (coronary artery bypass grafting) surgery. Patient was a driver with opium addiction. Patient medical history showed diabetes mellitus, hypertension, diabetic neuropathy, and major depressive disorder. Patient had a family history of ischemic heart disease in both parents. Angiography revealed significant stenosis (90-95%) at LAD (left anterior descending artery), LCX (left circumflex artery), and RCA (right coronary artery), resulting in the diagnosis with three-vessel disease with an LVEF (Left ventricle ejection fraction) of 30% and PAP (Pulmonary arterial pressure) of 28 mmHg. CT scan of first lung revealed minor opacities at the two ends of the horizontal fissure of the right lung, along with generalized vascular marking. No evident of consolidation was found. WBC count of 5800, with 57.9% neutrophils and 34.9 percent lymphocytes was seen.

CABG surgery was performed by an expert cardiac surgery team. A pacing wire and a chest tube were also inserted. On the 3rd day after surgery, around 8 a.m, blood pressure decreased unexpectedly,a continuous low EF, an IABP (Intra Aortic Balloon Pump) was inserted before a control chest X-ray confirmed its correct position (Figure 4)

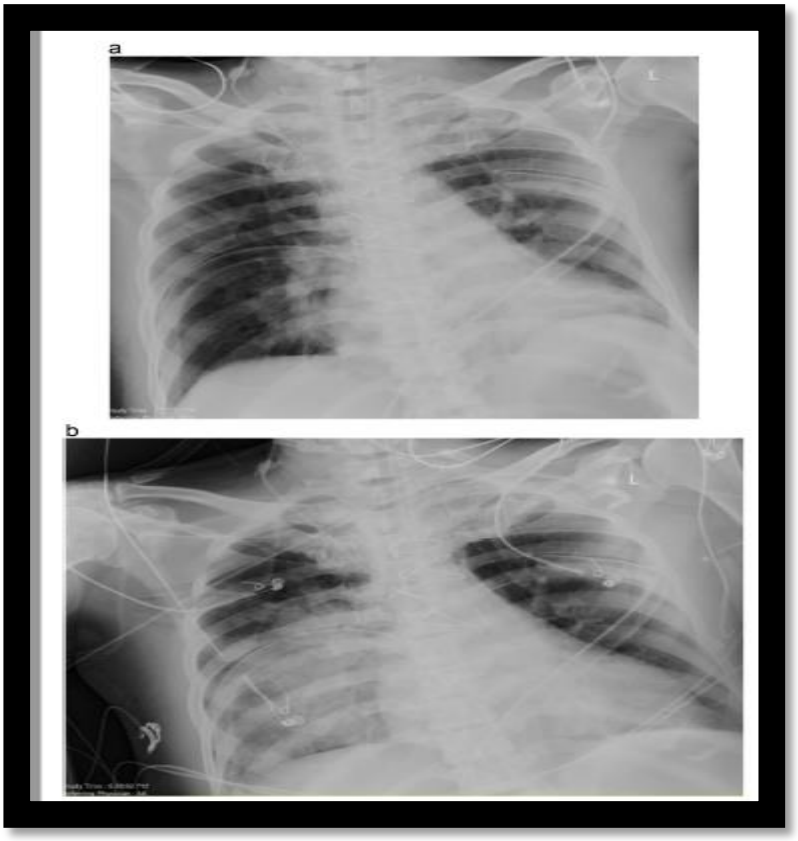


Figure 4: control chest X-ray confirmed its correct position

Echocardiogram demonstrated a dilated RV (Right ventricle) which brought up Pulmonary Embolism as a differential diagnosis. BP dropped again around 7oclock in the afternoon, along with a low-grade fever, tachycardia, tachypnea, loss of consciousness, and a decline in o2 saturation, was observed, blood ABG revealed acidosis. Inj Meropenem 500 mg and Vancomycin 500 mg were administered as an empirical antimicrobial therapy. second chest X-ray of the day, taken just hours after the first, showed a large haziness in the right lung's middle and inferior lobes.

Patient had a homogenous opacity at the base of right lung and a severe consolidation in middle lobe with an air bronchogram pattern on lung CT scan. Patients last lab results showed WBC of 3140 with 86.9% neutrophils and 11.8% lymphocytes. Unfortunately, patient was declared dead after a 40-minute Cardiopulmonary resuscitation(CPR).actual cause of death was not discovered until an autopsy of the lung had been done.Result of Autopsy revealed, a huge amount of foamy pus from the right main bronchi. Pseudomonas Aeruginosa fulminant pneumonia was confirmed to be the cause of death after an autopsy of the lung tissues

CONCLUSION: Considering pseudomonas aeruginosa as a cause of pneumonia after cardiac surgery, an organized, modified guideline is require to analyse and to determine the best treatment strategies for this complication

Reference: Sakhaei D, Parvas E, Ilkhani S, Tafti SH, Jameie M, Mohebbi H. Fulminant psudeumonas pneumonia following coronary artery bypass grafting-Case report. International Journal of Surgery Case Reports. 2022 Nov 26:107785.