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CARDIOMAG NEWSLETTER

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IN THIS ISSUE

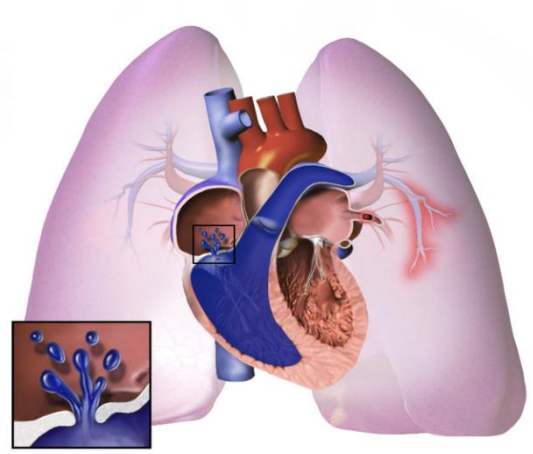
Dear Doctor,

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

Association between systemic Inflammatory Response Index and parameters in Idiopathic Pulmonary Arterial Hypertension patients

INTRODUCTION

Pulmonary arterial hypertension (PAH) is a chronic and progressive disorder characterized by angioproliferative vasculopathy in the pulmonary arterioles. It causes endothelial and smooth muscle proliferation and dysfunction, inflammation, and thrombosis. These alterations raise pulmonary vascular resistance and arterial pressure, leading to right ventricular failure and mortality if not managed.[1] A new inflammatory biomarker called the systemic inflammatory response index (SIRI) was calculated by multiplying the neutrophil count by the monocyte count and dividing the result by the lymphocyte count. Prior studies have demonstrated that SIRI was associated with an increased risk of cardiovascular death as well as the onset and prognosis of several respiratory and cardiovascular disorders, such as acute coronary syndrome, chronic obstructive pulmonary disease, and congestive heart failure. Growing evidence points to a potential relationship between the systemic inflammatory response index (SIRI) and heart failure prognosis; nevertheless, its function in idiopathic pulmonary arterial hypertension (IPAH) was not fully recognized.[2] The objective of this study was to examine the association between SIRI and parameters in IPAH patients, including functional ability, echocardiography results, hemodynamic measurements, and long-term outcomes.



Pulmonary hypertension

METHODS

This was a retrospective cohort study, that included 426 consecutive IPAH patients who underwent right heart catheterization. The following patients met the inclusion criteria: (1) those who were 18 years of age or older; (2) those who had hemodynamic characteristics of PAH as determined by RHC; and (3) those who, per the recommendations, had no other cause of PAH and were ultimately diagnosed with IPAH. On the day of admission, data on the patient was gathered, including demographics, smoking and drinking habits, World Health Organization functional class (WHO-FC), comorbidities, and medications specific to PAHs. Venous blood samples were drawn that same day to gather information for the liver function test, kidney function test, N-terminal pro brain natriuretic peptide (NT-proBNP) levels, fasting blood lipid, and blood routine inspection. Hematological parameters were measured using the SYSMEX XN-20 Automated Hematology Analyzer. After admission, an echocardiography was finished in 48 hours. When the patients' conditions were stable, the 6-minute walking test and RHC were conducted. Composite inflammation signs from standard blood testing were

used to determine SIRI. Clinical worsening served as the primary outcome indicator. To evaluate relationships between SIRI and measures of IPAH severity, Spearman correlation coefficients were employed. The best SIRI threshold and prediction power were found using a receiver operating characteristic (ROC) curve study. Utilizing Cox proportional hazard models and Kaplan-Meier analysis, the association between SIRI and clinical worsening was investigated.

RESULTS

The study results showed that individuals who experienced clinical deterioration had a significantly lower WHOFC rating and a lower 6 MWD. Patients with worsening clinical conditions showed considerably lower values of albumin, SvO2, and CI, and significantly higher levels of NT-proBNP, mPAP, and PVR than patients whose conditions did not deteriorate clinically. Compared to patients without clinical worsening, those with clinical worsening exhibited significantly higher SIRI, SII, and NLR levels. The SIRI had an AUC of 0.761 in predicting clinical worsening, according to the ROC curve study. It was found that the ideal threshold was 0.714, resulting in a sensitivity of 73.5% and a specificity of 70.1%. PVR and mPAP were greater in patients with SIRI >0.714 than in those with SIRI ≤0.714, indicating significantly worse pulmonary hemodynamics. SIRI was positively correlated with indicators including pericardial effusion, mean pulmonary arterial pressure, pericardial diastolic diameter, N-terminal pro-brain natriuretic peptide, and pulmonary vascular resistance. On the other hand, SIRI and left ventricular end-diastolic diameter showed inverse associations across a 6-minute walking distance. According to Kaplan-Meier curves, patients with SIRI >0.741 had a considerably higher risk of clinical deterioration than those with SIRI ≤0.741 (P <0.001) (Figure 1). According to an adjusted Cox analysis, SIRI was still able to predict clinical worsening independently (hazard ratio: 1.366, 95% confidence interval: 1.073–1.738, P=0.011). The European Society of Cardiology/European Respiratory Society risk assessment score was not the only predictive value that SIRI offered, according to ROC analysis.

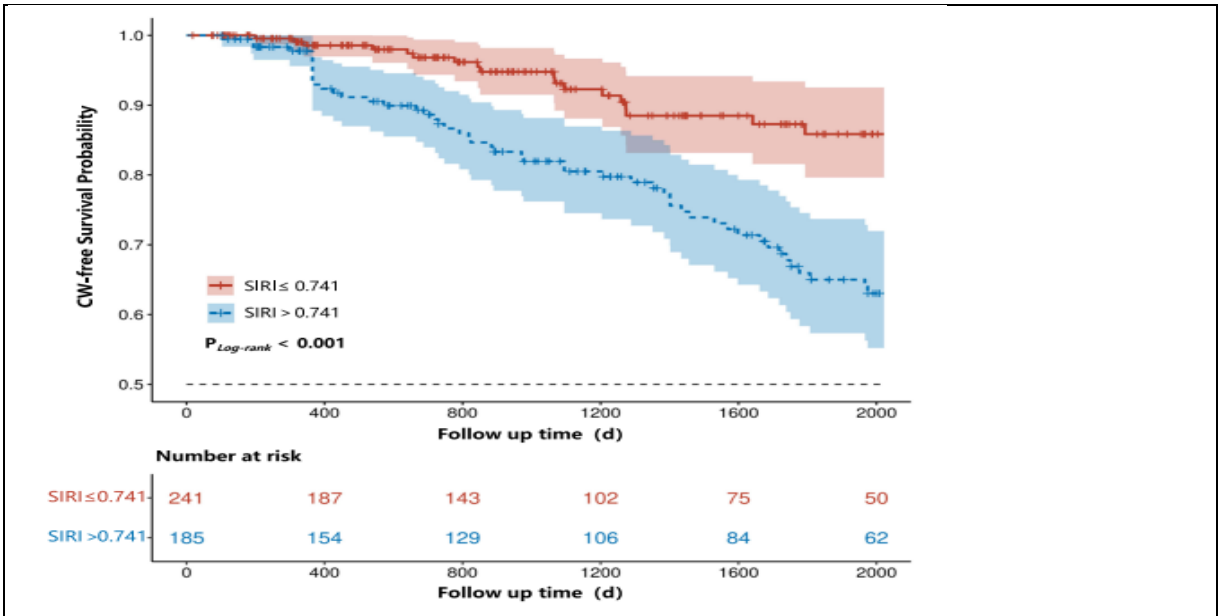


Figure 1. Kaplan-Meier graphs categorising IPAH patients according to their baseline SIRI levels

DISCUSSION

The current study demonstrated that the SIRI can predict clinical decline in IPAH patients on its own and provides extra advantages when combined with the concise ESC/ERS risk score.[2] Similar to this, Yogeswaran A, et al. showed that the neutrophil-to-lymphocyte ratio (NLR), a haematological indicator associated with inflammation, also serves as an independent predictor of clinical decline and mortality in PAH.[3]

CONCLUSION

The current study concluded that the authors observed a relationship between the functional status, echocardiography, hemodynamic parameters, and SIRI in IPAH patients. The results shown that the SIRI can independently predict clinical decline in IPAH patients and provides extra advantages when combined with the short ESC/ERS risk score. As a result, the SIRI functions as a useful and reliable prognostic indicator.

SIRI can independently predict clinical decline in IPAH patients and provides extra advantages when combined with the short ESC/ERS risk score.

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Relationship between impaired cardiovascular fitness and the triglyceride-glucose index in non-diabetic young population

INTRODUCTION- Cardiorespiratory fitness (CRF) is commonly regarded as the best indicator of overall body health and function since it was closely linked to the integrated functioning of many physiological systems, such as the cardiovascular, musculoskeletal, and respiratory systems. A low estimated cumulative risk factor is one of the best indicators of non-communicable diseases, such as cancer and cardiovascular disease (CVD), and has been linked to an increased risk of traditional cardiovascular risk factors.[1] It has been demonstrated that the triglyceride-glucose (TyG) index was linked to the incidence of CVD. Individuals with the highest TyG index had a nearly twofold increased risk of coronary heart disease in comparison to those with the lowest TyG index. In middle-aged and older people, the triglyceride-glucose index has been associated with the development, progression, and prognosis of cardiovascular

disease. However, in the young, non-diabetic population, the link between the TyG index and poor cardiovascular function (CVF) was yet unknown.[2] The aim of the study was to investigate the link between the TyG index and impaired CVF in a non-diabetic young population.

METHODS-This was a cross sectional study, included a total of 3364 participants who completed CVF examination and had fasting blood samples taken. Only survey participants aged 12 to 49 were eligible for the CVF assessment. Participants in CVF tests were frequently asked to conduct aerobic exercise on a treadmill. Impaired CVF was defined as low to moderate CVF levels assessed by estimated maximum oxygen consumption (Vo2max) using sex and age-specific parameters. The TyG index was computed as Ln [TG (mg/dL) × FPG (mg/dL)/2]. This study's statistical analysis was performed using R version 4.3.1 (R Foundation). The study population was grouped into quartiles using population-weighted descriptive statistics and the TyG index. The study used restricted cubic splines (RCS) with weight to investigate the correlation between the TyG score and impaired CVF.

RESULTS-The study results showed that the TyG index (median±standard deviation) was 8.36±0.52, and the study population's age (median with interquartile range) was 28 (19–37) years. Impaired CVF was positively correlated with non-Hispanic Black individuals' BMI, waist circumference, SBP, DBP, metabolic syndrome, HbA1c, FPG, TG, and TyG index. Impairment in CVF was negatively correlated with Hb and level 6 physical activity. Whether elevated by one unit or one standard deviation, the TyG index showed a strong positive correlation with reduced CVF in three weighted multivariable logistic models. The TyG index was further divided into quartiles, with the Q1 group serving as a standard for assessing the relationship between impaired CVF and other quantile groups. In model 3, Q3 (OR: 1.61; 95% Cl 1.21–2.13) and Q4 (OR: 1.55; 95% Cl 1.06–2.26) showed a significant rise in odds ratios (OR) when compared to Q1, and the p-value for the trend was 0.009 (Table 1). Multivariable logistic regression analysis indicated a significant relationship between the TyG index and impaired CVF (per 1-unit increase in the TyG index: OR, 1.46; 95% Cl 1.13–1.90). Restricted cubic splines (RCS) demonstrated a dose-response association between the TyG index and impaired CVF. In individuals under the age of 20, a significant interaction (p=0.027) was seen between the TyG score for impaired CVF and sex.

TyG index	HR (95%CI)		
	Model 1	Model 2	Model 3
Per 1 unit increase	1.60 (1.31–1.94)**	1.39 (1.11–1.74)*	1.46 (1.13–1.90) *
Per 1 SD increase	4.46 (2.38–8.35)**	2.84 (1.38–5.83)*	3.38 (1.47–7.76) *
Q1	Reference	Reference	Reference
Q2	1.26(0.99–1.60)	1.22(0.94–1.57)	1.24 (0.96–1.60)
Q3	1.80 (1.35–2.38)**	1.53 (1.17–2.00)*	1.61 (1.21–2.13)*
Q4	1.81 (1.34–2.44)**	1.45 (1.02–2.06)*	1.55 (1.06–2.26)*
p for trend	**< 0.001**	**0.011**	**0.009**

Table 1. Relationship in various logistic models between the TyG index and impaired cardiovascular fitness

DISCUSSION-The current study identified a link between the TyG index and poor CVF in young non-diabetic individuals. This study also discovered a significant sex interaction between the TyG index and poor CVF in individuals under the age of 20.[2] Lu YW et al. and Wu S et al. have found that the TyG index was linked with subclinical atherosclerosis, and arterial stiffness.[3,4]

CONCLUSION-The current study concluded that those with higher TyG index values among young people without diabetes were more likely to have impaired CVF. Furthermore, sex may have an impact on CVF, with men more likely to experience reduced CVF in settings with similar TyG index values.

Higher TyG index values in the young, non-diabetic population increase the risk of experiencing impaired CVF.

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Investigation of the association between symptomatic intracranial hemorrhage occurrence and glial fibrillary acidic protein levels in the bloodstream

INTRODUCTION- Intracranial hemorrhage (ICH) is a common consequence of endovascular treatment (EVT), with rates as high as 46.1% in clinical trials. Asian individuals may have an even higher risk of ICH following endovascular therapy for acute ischemic stroke (AIS) due to the higher prevalence of cerebral atherosclerosis. Hemorrhagic consequences have the potential to reduce or eliminate the benefit-risk ratio of endovascular treatment, particularly in cases of symptomatic ICH (sICH). Consequently, it was critical to determine the predictors of sICH following endovascular therapy in real-world settings in order to continually enhance the effectiveness of this novel therapeutic approach.[1] It has recently been shown that glial fibrillary acidic protein (GFAP) serves as a valid biomarker to differentiate intracerebral hemorrhage from acute internal shock. In addition, individuals with major artery blockage who had endovascular therapy had an adverse prognosis at 90 days if their GFAP levels were increased. Moreover, patients' risk of parenchymal bleeding was 2.51 times higher in those with greater GFAP levels 24 hours after the stroke. However, no research has been done on the connection between GFAP and sICH in AIS patients receiving EVT.[2] The purpose of this study was to determine whether GFAP and sICH were related in AIS patients receiving EVT.

METHODS- This was a retrospective cohort study, included 142 patients diagnosed with AIS and treated with endovascular therapy were initially screened. Patients who presented with major artery blockage in the anterior circulation and received EVT using contemporary thrombectomy procedures were eligible. Patients having concurrent disorders such as aneurysms, vasculitis, arterial dissection, arteriovenous malformation, Moyamoya syndrome, or hematological system abnormalities were not eligible for the study. The control group was made up of Physical Examination Center members who never had a stroke. GFAP levels in the bloodstream were measured using an enzyme linked immunosorbent assay before endovascular therapy (T1) and 24 hours later (T2). sICH was identified using the Heidelberg Bleeding Classification. To evaluate independent risk factors for sICH, multivariable logistic regression models were adjusted for age, gender, and variables with a p-value <0.1 in the univariate analysis. Furthermore, the C-statistic, net reclassification index (NRI), and integrated discrimination improvement (IDI) were calculated to assess the discriminatory and predictive power of GFAP when used in conventional models.

RESULTS- The study showed that patients that were included had an average age of 70.9 ± 10.1 years, with 64.1% of them being male. The baseline median ASPECTS score was 9 (IQR 8–9) while the baseline median NIHSS score was 15 (interquartile range [IQR], 12–18). Of the patients, 118 (83.1%) had effective recanalization, whereas 77 (54.2%) had poor collateral status. There was a notable increase in serum GFAP levels at T2 compared to T1 (3.813 [1.474, 5.876] versus 0.249 [0.150–0.576] ng/mL, p=0.001). Serum GFAP levels in AIS patients were considerably higher at T1 than in the controls (0.249 [0.150–0.576] versus 0.065 [0.041–0.110] ng/mL, p = 0.001). Following EVT, 18 (14.5%) of the 142 AIS patients experienced sICH. Both the multivariable adjusted model (OR 1.518, 95% CI 1.153–2.000, p = 0.003) and the unadjusted model (OR 1.513, 95% CI 1.269–1.805, p = 0.001) demonstrated significant correlations between serum GFAP levels at T2 and sICH. The violin plot of serum Log GFAP levels in controls and AIS patients was shown in Figure 1. In the control group, the average Log GFAP levels were 4.2 ± 0.6 pg/mL. Patients with AIS showed a substantial rise at T1 (5.7 ± 1.0 pg/mL) or T2 (8.0 ± 0.9 pg/mL) when compared to controls. Additionally, Log GFAP levels at T2 were greater than T1 (p = 0.001). Additionally, GFAP at T2 significantly improved risk categorization for sICH when added to the conventional model (integrated discrimination improvement [IDI] 0.183, 95% CI 0.070–0.295, p = 0.001).

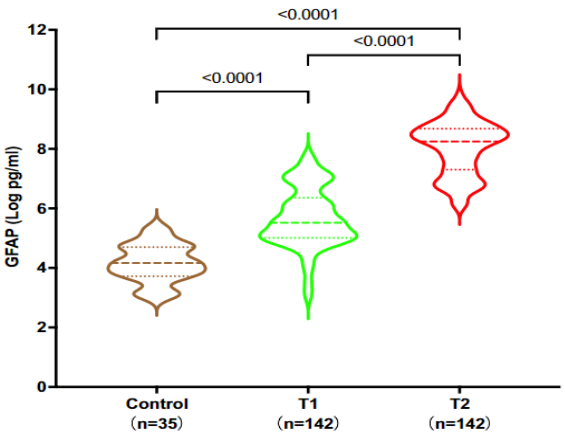

Figure 1 Serum Log GFAP levels in AIS patients and controls plotted using a violin plot.

DISCUSSION- The current study indicated that an increased risk of sICH was connected with greater GFAP levels.[2] Similar to this, Chen CH et al. shown that a higher risk of parenchymal bleeding was associated with elevated GFAP.[3]

CONCLUSION-The current study concluded that in AIS patients, serum GFAP levels significantly increased 24 hours after EVT. An increased risk of sICH was connected with greater GFAP levels.

GFAP may function as a reliable marker for sICH in AIS patients receiving EVT treatment.

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Case report of late manifestation of pericardial rupture and traumatic tricuspid valve chordal rupture combined with cardiac herniation

INTRODUCTION

Rarely, blunt cardiac trauma leading to tricuspid regurgitation is most frequently connected to auto accidents. These injuries can also be brought on by falling from a height, which can lead to arrhythmias and hemodynamic compromise. The severity of injury determines when the symptoms of traumatic tricuspid regurgitation manifest. They may manifest early or later.[1] Surgery to repair the tricuspid valve was done very later than the injury's beginning since traumatic tricuspid regurgitation was typically well tolerated throughout the acute period. In order to diagnose valvular apparatus injuries and provide prompt, effective therapy, transthoracic echocardiography, or TTE, was a crucial tool.[2] The aim of the report was to present the case of a patient who got injuries in a traffic accident and displayed pericardial rupture, partial cardiac herniation, and tricuspid valve symptoms after ten years.

CASE PRESENTATION

A 58-year-old patient was brought to the hospital with dyspnea, tiredness from physical activity, and chest pain. Two weeks prior to admission, the symptoms first appeared. Other than a car accident ten years ago that resulted in chest trauma and a double rib fracture, his past medical history was uneventful. Upon physical examination, the patient had a pansystolic murmur at the lower left sternal border, evidence of right ventricular failure, and cyanosis. Her blood pressure was 110/80 mmHg and her heart rate was 110 beats per minute. A right bundle branch block was seen on the electrocardiogram (ECG). TTE demonstrated chordal rupture-related flailing of the tricuspid valve's anterior leaflet, which left the anterior half of the leaflet entirely unsupported. Moreover, the posterior and septal leaflets were somewhat tethered. After all other potential reasons were ruled out, massive tricuspid regurgitation which was detected by a colour doppler (Figure 1) and then diagnosed as post-traumatic. Through echocardiography, the Ebstein anomaly was ruled out (the septal displacement index of the tricuspid to mitral leaflet insertion was 0.76 cm/m2, or less than 0.8 cm/m2). Normal results from laboratory blood tests, such as those for inflammatory markers and blood cultures (carcinoid, infectious endocarditis), along with the lack of any gastrointestinal symptoms, invasive cardiac intervention, or radiation therapy, ruled out other possible causes. A thorough evaluation using transoesophageal echocardiography (TEE) and TTE revealed additional findings, including an enlarged right ventricle (diastolic diameter of 5,1 cm from the PLAX view, compared to LV diameter of 4,8 cm), a moderate reduction in right ventricular function, and TV annular dilatation (4,4 cm) with decreased motion amplitude. Ventricular overload and

the left ventricle's size and form were both within normal bounds. Pericardial effusion did not appear to be considerable. Following ten days of severe heart failure treatment, the patient was sent to the cardiac surgery unit so that the tricuspid valve could be rebuilt. Before heart surgery, coronary angiography was used to rule out the unusual discovery of multiple dynamic stenotic lesions at the same level of all left-sided coronary vessels, primarily on the circumflex coronary artery's first, second, and third obtuse marginal branches. An ancient pericardial rupture with a thick margin was seen upon medial sternotomy. The pericardial defect had a dimension of 7 by 6 cm, and the heart was herniated through it. According to the earlier coronary angiographic results, the coronary arteries were constricted along the line by the margin of the burst pericardium. The pericardial rupture was sutured, and the heart was put back in its proper location. During the operation, it was determined that the rupture of the main common chord at the level of the papillary muscle had caused the anterior leaflet of the tricuspid valve to totally lose its support. The CorMatrix patch and a tube for the reconstruction of the tricuspid valve were created based on the measurements determined by transesophageal echocardiography. Following the removal of the leaflets, the CorMatrix patch was sutured three times to the tricuspid annulus and the bases of the papillary muscles. The reason for performing the specific tricuspid surgery with CorMatrix patch was that there was not enough leaflet tissue to cover the valve area and enable neochordal implantation. A tube that connects to the annulus and the base of the papillary muscles was confirmed on control transthoracic echocardiography. Right ventricular systolic pressure remained at 38 mmHg, with some persistent tricuspid regurgitation. Following his recovery, the patient was sent home without showing any indications of right heart failure.

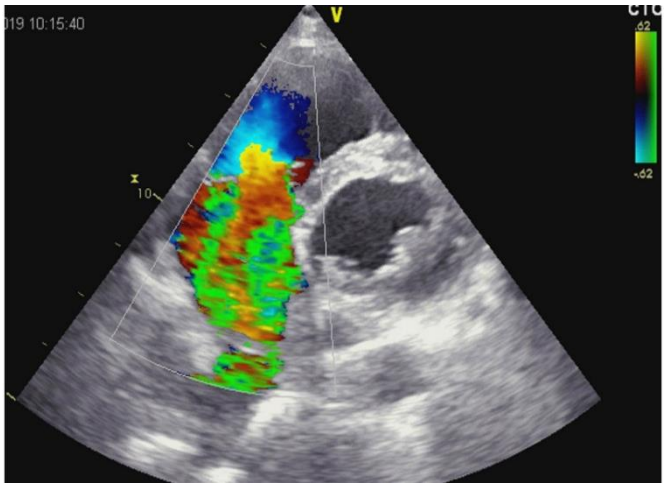

Figure 1. Transthoracic color doppler reveals massive tricuspid regurgitation

DISCUSSION

The current case report showed that transthoracic color doppler reveals massive tricuspid regurgitation.[2] Similar to this, Mehrotra D et al. showed that transesophageal echocardiograms reveal a ruptured tricuspid valve with significant tricuspid regurgitation.[1]

CONCLUSION

The current study concluded that this case highlights a mechanism that causes a delayed rupture of the tricuspid valve apparatus and highlighted the significance of prompt diagnosis. For every patient who has a chest trauma, repeat echocardiogram may be extremely important.

Cardiac computed tomography should be carried out due to the restricted usefulness of echocardiography in cases of posttraumatic pericardial rupture and cardiac herniation.

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