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CARDIO ROUNDS NEWSLETTER

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IN THIS ISSUE

Dear Doctor,

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

Association of heart rate control with adverse clinical outcomes in Acute Coronary Syndrome patients undergoing Percutaneous Coronary Intervention

INTRODUCTION- Acute coronary syndrome (ACS) is a primary cause of cardiovascular-related deaths in adults. The clinical manifestation of ACS is diverse, including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation MI (NSTEMI), and unstable angina.[1] Despite advancements in percutaneous coronary intervention (PCI), ACS patients remain at high risk for recurrent cardiovascular events, including myocardial infarction, heart failure, and mortality. Heart rate (HR) plays a crucial role in cardiovascular outcomes, with elevated resting HR linked to increased adverse event risks. While clinical guidelines highlight HR management, most studies assess single or average HR measurements, overlooking the importance of sustained HR control. The concept of time in target range of resting heart rate (TTR-HR) provides a more dynamic measure of HR stability and control over time. However, its prognostic significance in ACS patients undergoing PCI remains underexplored. So, this study investigates the association between TTR-HR and cardiovascular outcomes, examining whether maintaining HR within the target range can mitigate adverse events in ACS patients post-PCI.[2]

Cardiovascular Disease

METHODS- This retrospective study analyzed 1,455 ACS patients who underwent PCI and were followed for 12 months. The inclusion criteria were: (1) patients diagnosed with coronary heart disease who underwent PCI, (2) sinus rhythm confirmed by electrocardiogram (ECG), and (3) enrollment in the Center for Digital Management and Follow-up of Cardiovascular Diseases. Collected data included demographics, vital signs, family history, comorbidities, prescribed discharge medications, and relevant laboratory results from hospitalization. Resting heart rate (HR) was measured using a Bioland A223 device after ≥5 minutes of rest, with blood pressure and HR recorded twice daily. TTR-HR was categorized into three groups (0–50%, >50%–75%, >75%–100%). The primary outcome was a composite of all-cause mortality, non-fatal myocardial infarction, angina, or heart failure hospitalization. Secondary outcomes included MACE and all-cause mortality. Cox regression was used to assess the association between TTR-HR and cardiovascular outcomes.

RESULTS- Among 1,455 ACS patients (mean age 62.3 ± 10.9 years, 76.9% male), univariate Cox regression showed that hypertension, heart failure, diuretic use, and arrhythmia were significantly associated with cumulative adverse events. After adjustment, hypertension (HR: 2.4, P = 0.005), diuretic use (HR: 2.02, P = 0.019), and arrhythmia (HR: 0.54, P = 0.012) remained significant. TTR-HR >75% was an independent protective factor against adverse events in ACS, with consistent effects across subgroups. A significant interaction was noted between HR TTR and heart failure (P < 0.05). Patients in the highest TTR-HR group (>75%–100%) had a lower incidence of composite endpoints (3.3%) and MACE (3.0%) than the other groups (P < 0.05). No significant association was found between TTR-HR and mortality. Higher TTR-HR was associated with a lower risk of composite endpoints. Patients in the lower TTR-HR groups (0–50% and >50%–75%) had a significantly higher cumulative risk (HR: 2.11, 95% CI: 1.19–3.74). Kaplan–Meier analysis showed better 12-month survival in the highest TTR-HR group (>75%–100%), emphasizing the benefits of maintaining HR within the target range (P = 0.009) (Figure 1).

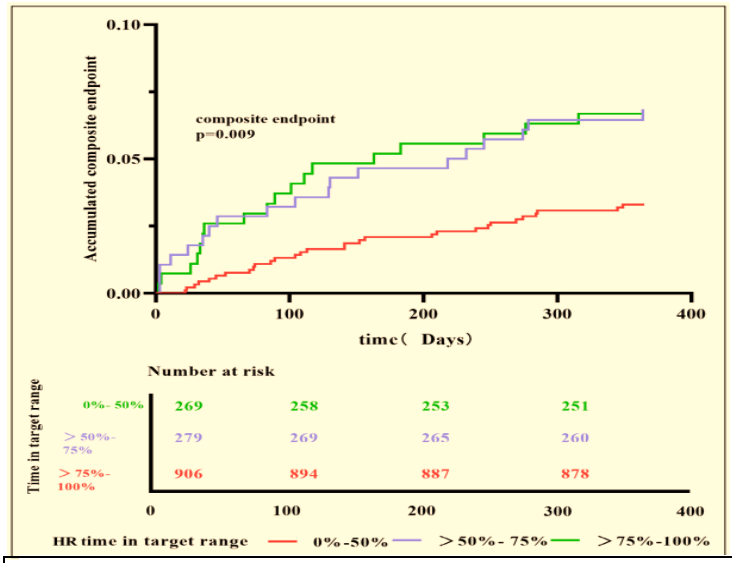

Figure 1. The 12-month follow-up Kaplan-Meier survival curve was stratified based on heart rate within the target range.

DISCUSSION- The current study showed that a higher TTR-HR (>75%) was independently linked to a lower risk of composite endpoints in ACS patients post-PCI. Maintaining HR within the target range reduced MACE incidence, including myocardial infarction, angina, and heart failure hospitalization.[2] The current findings align with Lai Y et al.[3], which demonstrated the prognostic value of TTR-HR in atrial fibrillation patients, and Jensen et al.[4], which highlighted the predictive role of HR at discharge after PCI, emphasizing the need for sustained HR control.

CONCLUSION- TTR-HR has strong prognostic value, with TTR-HR values greater than 75%-100% independently related to lower composite endpoint risk in ACS patients after PCI. These data highlight the need for efficient heart rate regulation in ACS patients following PCI.

TTR-HR is a significant predictor of outcome, and in ACS patients after PCI, TTR-HR > 75%–100% is independently linked to a lower risk of composite endpoints.

REFERENCES

1. Kang A et al. Real-world assessment of antiplatelet treatment therapies for acute coronary syndrome (ACS) patients in the United States in a nationwide Electronic Health Record in 2018-2020. European Heart Journal. 2023 Nov;44(Supplement_2):ehad655-1480.
2. Zheng J et al. Association of Time in Target Range of Resting Heart Rate With Adverse Clinical Outcomes in Patients With Acute Coronary Syndromes After Percutaneous Coronary Intervention. Glob Heart. 2025 Jan 17;20(1):3.

3. Lai Y et al. New Insights into Rate Control: Time in Target Range of Resting Heart Rate and Major Adverse Outcomes in Atrial Fibrillation. Glob Heart. 2024; 19(1):3.
4. Jensen MT, et al. Heart rate at discharge and long-term prognosis following percutaneous coronary intervention in stable and acute coronary syndromes—results from the BASKET PROVE trial. Int J Cardiol. 2013; 168(4):3802–6.

Effect of Non-Alcoholic Fatty Liver Disease on the Development of Abdominal Aortic Aneurysms

INTRODUCTION- Non-alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease globally and is characterized by hepatic steatosis when no other causes of secondary hepatic fat accumulation (e.g., excessive alcohol consumption) can be found.[1] NAFLD is a prevalent metabolic disorder closely linked to systemic conditions such as diabetes, obesity, and cardiovascular diseases. Recent studies highlight the association between NAFLD and arterial stiffness, inflammation, and oxidative stress, all of which may influence the progression of abdominal aortic aneurysms (AAAs). Despite growing evidence, the role of NAFLD as a predictor of AAA progression remains underexplored. So, this study aimed to assess the association between NAFLD, identified via non-enhanced computed tomography (CT), and AAA progression using data from multiple medical centers.[2]

METHODS- This multicenter retrospective study included 151 patients diagnosed with AAA who underwent abdominal CTA and non-enhanced CT between January 2015 and January 2023. Patients who had a non-enhanced abdominal CT examination and the availability of a follow-up CTA scan at least six months following the initial CTA examination were included. Based on abdominal CT results, patients with AAA were categorized as non-progression and progression (growth rate >10 mL/year), as well as NAFLD and non-NAFLD. CT and CTA scans were performed using standardized protocols across participating centres. Univariate and multivariate Cox regression analyses were conducted to identify factors associated with AAA progression. Receiver operating characteristic (ROC) curves were used to evaluate diagnostic performance, and Kaplan-Meier survival curves were plotted to compare progression rates.

RESULTS- The study population had a mean age of 69.1 ± 10.5 years, with 88.1% being male. NAFLD prevalence was significantly higher in the AAA group compared to controls (43.7% vs. 31.1%, $p = 0.024$). Hypertension (62.9% vs. 51.0%, $p = 0.036$) and diabetes (28.5% vs. 15.9%, $p = 0.009$) were also more prevalent in the AAA group, while BMI and smoking history showed no significant differences. Over a median follow-up of 10.7 months (range: 6.0–76.0 months), 57 patients (37.7%) experienced AAA progression (growth rate > 10 mL/year). Progression was more frequent in patients with NAFLD (77.2% vs. 23.4%, $p < 0.001$) and was associated with larger aortic diameter [45.6 mm vs. 37.5 mm, $p < 0.001$], greater AAA volume [65.2 mL vs. 41.2 mL, $p < 0.001$], higher intraluminal thrombus (ILT) prevalence (94.7% vs. 83.0%, $p = 0.035$) and larger ILT maximal diameter [12.3 mm vs. 6.7 mm, $p < 0.001$]. Patients with NAFLD had significantly lower progression-free survival rates (log-rank $p < 0.001$) (Figure 1). Univariate Cox regression identified NAFLD (HR: 5.99; $p < 0.001$), AAA maximal diameter (HR: 1.07 per mm; $p < 0.001$), AAA volume (HR: 1.01 per mL; $p < 0.001$), and ILT maximal diameter (HR: 1.06 per mm; $p < 0.001$) as predictors. Multivariate analysis confirmed NAFLD (HR: 4.28; $p < 0.001$) and AAA diameter (HR: 1.06 per mm; $p = 0.010$) as independent predictors. For predicting AAA progression, the AUCs were 0.769 for

NAFLD, 0.793 for AAA maximal diameter, and 0.857 for the combined model. Sensitivity and specificity were 77.2% and76.6% (NAFLD), 80.7% and 71.3% (AAA diameter), and 73.7% and 85.1% (combined) respectively. The combined model had a significantly higher AUC than NAFLD ($p<0.001$) and AAA diameter ($p=0.034$), while NAFLD and AAA diameter showed no significant difference ($p=0.608$). Cohen's kappa values for intra-observer agreement were 0.917 (ILT) and 0.933 (NAFLD), while inter-observer values were 0.930 and 0.906, respectively. The intraclass correlation coefficients for maximal aneurysm diameter, total aneurysm volume, ILT maximal diameter, and growth rate ranged from 0.963 to 0.982 for intra-observer agreement and 0.954 to 0.977 for inter-observer agreement.

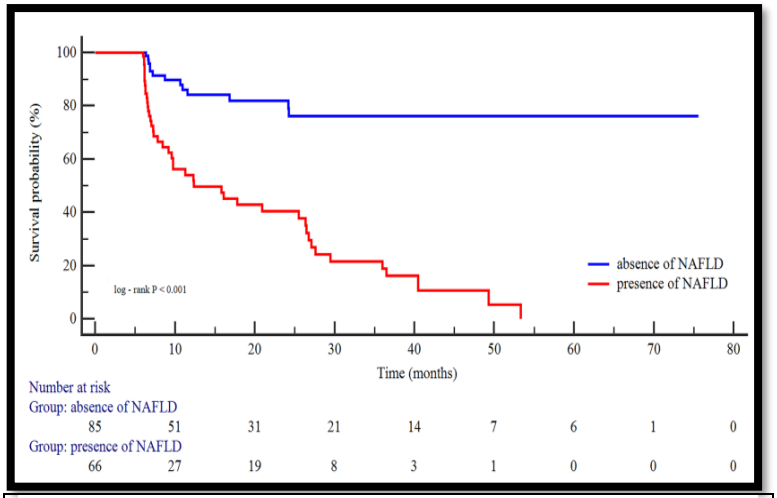

Figure 1. Kaplan-Meier event-free survival curves for AAA progression events in patients.

DISCUSSION- The current study demonstrated that NAFLD is an independent predictor of AAA progression. Additionally, the combined model incorporating AAA maximal diameter and NAFLD provided superior predictive accuracy for AAA progression, as evidenced by an AUC of 0.857.[2] These findings align with previous studies, which also reported a significant association between NAFLD and cardiovascular conditions. For instance, Mahamid et al. observed that patients with AAA had a higher prevalence of NAFLD compared to healthy controls.[3]

CONCLUSION- There was a relationship between the occurrence and development of AAA and NAFLD. Also, this study found that the maximum diameter of AAA and NAFLD were separate predictors of AAA development. Therefore, NAFLD based on CT could be a non-invasive, dependable, and simple marker to predict the course of AAA.

NAFLD upon non-enhanced CT is an independent predictor of abdominal aortic aneurysms progression.

REFERENCES

1. Pouwels S et al. Non-alcoholic fatty liver disease (NAFLD): a review of pathophysiology, clinical management and effects of weight loss. BMC Endocr Disord. 2022 Mar 14;22(1):63.
2. Wang X et al. Association between non-alcoholic fatty liver disease and progression of abdominal aortic aneurysm: a multicenter study. BMC Med Imaging. 2025 Jan 20;25(1):24.
3. Mahamid M et al. The interplay between abdominal aortic aneurysm, metabolic syndrome and fatty liver disease: a retrospective case-control study. Diabetes Metab Syndr Obes. 2019;12:1743–9.

Correlation between the Triglyceride-Glucose Index and Contrast-Induced Nephropathy in Patients Receiving Percutaneous Coronary Intervention for Chronic Total Occlusion

INTRODUCTION- The occurrence of coronary artery disease is rising with increasing life expectancy and the elderly population, leading to the necessity of percutaneous coronary intervention (PCI) and coronary angiography (CAG) for chronic total occlusion (CTO).[1] A significant complication of PCI is contrast-induced nephropathy (CIN). Recently, researchers have been examining whether certain metabolic indicators may improve CIN risk categorization. The triglyceride-glucose (TyG) index has been considered a measure of insulin resistance and metabolic state. [2] Although its predictive value for CIN in selected populations has been demonstrated, limited evidence regarding its role in CTO patients undergoing PCI. So, this study assessed the association between the TyG index and the development of CIN and mortality in this population.

METHODS - This multicenter, retrospective, cross-sectional study included 218 patients who underwent procedural coronary angiography (CAG) and PCI for CTO lesions in at least one coronary artery. Patients were divided into two groups based on their TyG index: Group 1 (TyG index ≥8.65) and Group 2 (TyG index <8.65). This threshold was established based on previous studies assessing the relationship between TyG index and CIN. The inclusion criteria were adults aged 18–90 years with ejection fraction (EF) ≥40% and at least one CTO lesion requiring PCI. The primary outcomes were the development of CIN and all-cause mortality over a 96-month follow-up period. Logistic regression identified CIN predictors, including variables with $p \leq 0.1$ in univariate analysis. Kaplan-Meier analysis assessed the association of the TyG index with CIN and mortality. ROC curves determined the TyG index cutoff for CIN prediction. A p-value < 0.05 was considered statistically significant.

RESULTS – In this study, the mean age of Group 1 was significantly higher than Group 2 (65.8 ± 10.94 vs. 61.68 ± 11.4 years, p = 0.009). Diabetes mellitus (44.8% vs. 13.1%, p <0.001) and dyslipidemia (38.8% vs. 25%, p = 0.036) were also more prevalent in Group 1. Other notable differences included higher rates of cerebrovascular disease (4.5% vs. 0%, p = 0.049) and in-stent restenosis (16.4% vs. 7.1%, p = 0.046) in Group 1. Biochemical analyses revealed that glucose (p < 0.001), triglycerides (p < 0.001), and uric acid levels (p = 0.044) were significantly elevated in Group 1. No significant differences were observed between the groups for ejection fraction (EF), hypertension, smoking status, chronic kidney disease, or peripheral artery disease. Univariate logistic regression analysis identified several predictors of CIN, including age, chronic kidney disease (CKD), peripheral artery disease, left ventricular ejection fraction (LVEF), LDL cholesterol, and the TyG index (p < 0.05 for all). The ROC analysis identified a TyG index cutoff of 8.86 for predicting CIN, with a sensitivity of 59% and specificity of 54% (AUC = 0.60, 95% CI: 0.51–0.68, p = 0.044) (Figure 1). Kaplan-Meier survival analysis demonstrated a significant association between CIN and mortality (Log-rank = 19.409, p < 0.001), while no significant relationship was observed between the TyG index and mortality (Log-rank = 0.808, p = 0.369).

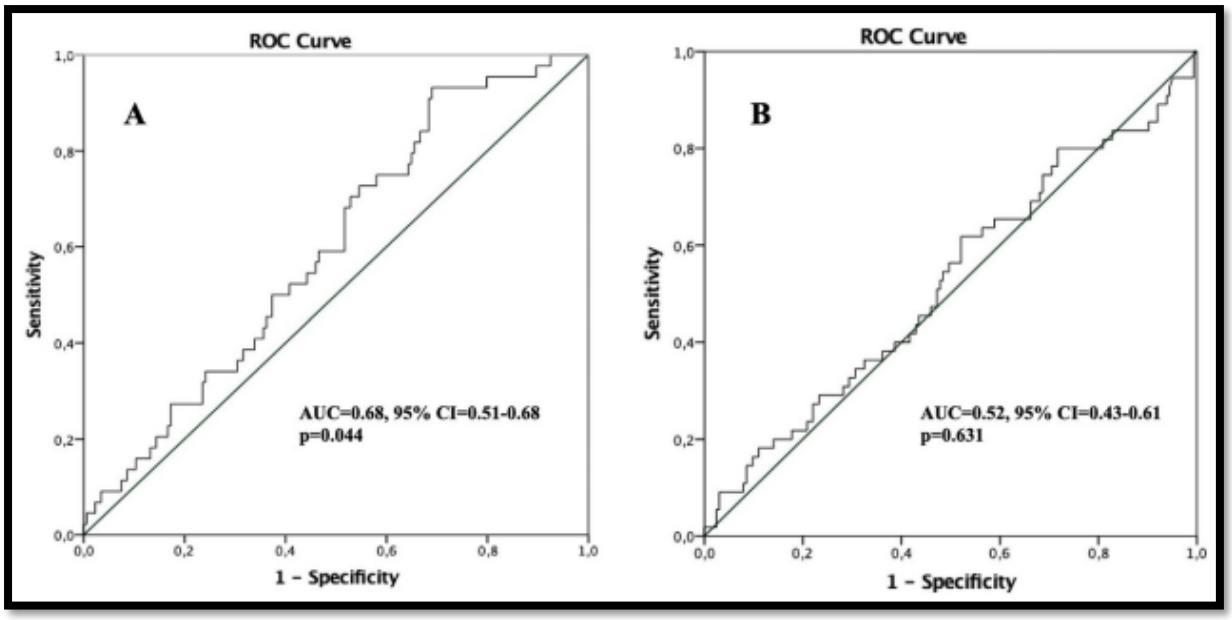

Figure 1. ROC curve analysis of TyG index to predict the development of CIN (A) and death (B)

DISCUSSION- This study demonstrated that a higher TyG index is an independent predictor of CIN in CTO patients undergoing PCI, aligning with prior studies that reported similar findings in different patient groups.[1] Qin Y et al. observed a significant association between the TyG index and CIN in patients with type 2 diabetes and coronary artery disease[3], while Gursoy E et al. noted this relationship in non-ST elevation myocardial infarction patients undergoing PCI.[4]

CONCLUSION- The frequency of CIN in patients with CTO having PCI was correlated with the TyG index. The TyG index is a useful tool for risk stratification in clinical settings due to its affordability and simplicity of use. Patients with CTO having PCI may be protected against the development of CIN by including the TyG index in their clinical evaluation.

The development of CIN may be prevented by including the TyG index in the standard clinical evaluation of CTO patients receiving PCI.

REFERENCES

1. Söner S et al. Association between the triglyceride-glucose index and contrast-induced nephropathy in chronic total occlusion patients undergoing percutaneous coronary intervention. BMC Cardiovasc Disord. 2025 Jan 20;25(1):31.
2. Chang WT et al. Diagnostic efficacy of the triglyceride-glucose index in the prediction of contrast-induced nephropathy following percutaneous coronary intervention. Front Endocrinol (Lausanne). 2023 Nov 22;14:1282675.
3. Qin Y et al. A High Triglyceride Glucose Index Is Associated With Contrast-Induced Acute Kidney Injury in Chinese Patients With Type 2 Diabetes Mellitus. Front Endocrinol (Lausanne). 2021;22(11):522883.
4. Gursoy E, Baydar O. The triglyceride-glucose index and contrast-induced nephropathy in non-ST elevation myocardial infarction patients undergoing percutaneous coronary intervention. Medicine (Baltimore). 2023;102(2):e32629.

A Challenging Case of Right Heart Masses in an Acute Myeloid Leukemia Patient

INTRODUCTION- Cardiac masses are uncommon, although they remain a significant component of cardio-oncology practice. These include benign tumors, malignant tumors (both primary and secondary), and tumor-like diseases (such as thrombus, Lambl's excrescences, and pericardial cysts).[1] The differentiation of intracardiac masses presents a significant diagnostic challenge, especially in patients with hematologic malignancies. Cardiac masses can have various etiologies, including thrombotic, infectious, neoplastic, and inflammatory causes, each requiring distinct treatment approaches. Acute Myeloid Leukemia (AML) patients may present with intracardiac masses that complicate treatment decisions, particularly when urgent interventions, such as allogeneic hematopoietic stem cell transplantation (allo-HSCT), are necessary. This case report describes a 33-year-old male diagnosed with AML who developed right heart masses of unknown etiology, highlighting the complexity of diagnosis and management in such cases.[2]

CASE PRESENTATION- A 33-year-old male was diagnosed with AML (M2a, based on the FAB classification) in November 2021 after presenting with fatigue and petechiae. Initial laboratory investigations revealed a white blood cell count of 29.1 × 10^9/L, hemoglobin level of 57 g/L, and platelet count of 17 × 10^9/L. Bone marrow examination confirmed the AML diagnosis and subsequent molecular and cytogenetic analysis categorized the patient as intermediate risk based on European Leukemia Net (ELN) criteria. The patient underwent induction chemotherapy with an IA regimen (Idarubicin and Cytarabine), which failed to achieve complete remission. Subsequent cycles of DAC+CAG (Decitabine, Aclarubicin, Cytarabine, and G-CSF) and CLAG (Cladribine, Cytarabine, and G-CSF) were administered, gradually reducing blast count levels. A fourth round of CLAG chemotherapy achieved complete remission, with minimal residual disease (MRD) dropping to <0.001%. Given the refractory nature of his leukemia, allo-HSCT was planned as the next step in treatment. During the pre-transplant evaluation in April 2022, an echocardiogram unexpectedly revealed multiple mobile, homogenous masses in the right atrium and right ventricle. Further imaging with cardiac MRI and PET-CT confirmed the presence of nodular lesions with calcified capsules, yet no definitive signal changes were observed between first-pass perfusion and delayed enhancement sequences, complicating the differential diagnosis (Figure 1). Given the urgency of proceeding with the transplant, a multidisciplinary team—including cardiologists, hematologists, and radiologists—deliberated on the potential risks of these masses. Open-heart biopsy was deemed too risky due to the patient’s fragile hematologic condition, and an interventional biopsy was considered technically challenging with a low success rate. Despite the uncertainty regarding the nature of these cardiac masses, the decision was made to proceed with allo-HSCT while initiating anticoagulation therapy with low-molecular-weight heparin in case the masses were thrombotic in origin. The transplant was successfully performed with peri-transplant management, including antifungal prophylaxis and continued anticoagulation therapy. Post-transplant follow-up echocardiograms showed no reduction in mass size, though calcification gradually increased. Over two years post-transplant, the patient remained in leukemia remission, but the cardiac masses persisted with progressive calcification, still of undetermined etiology.

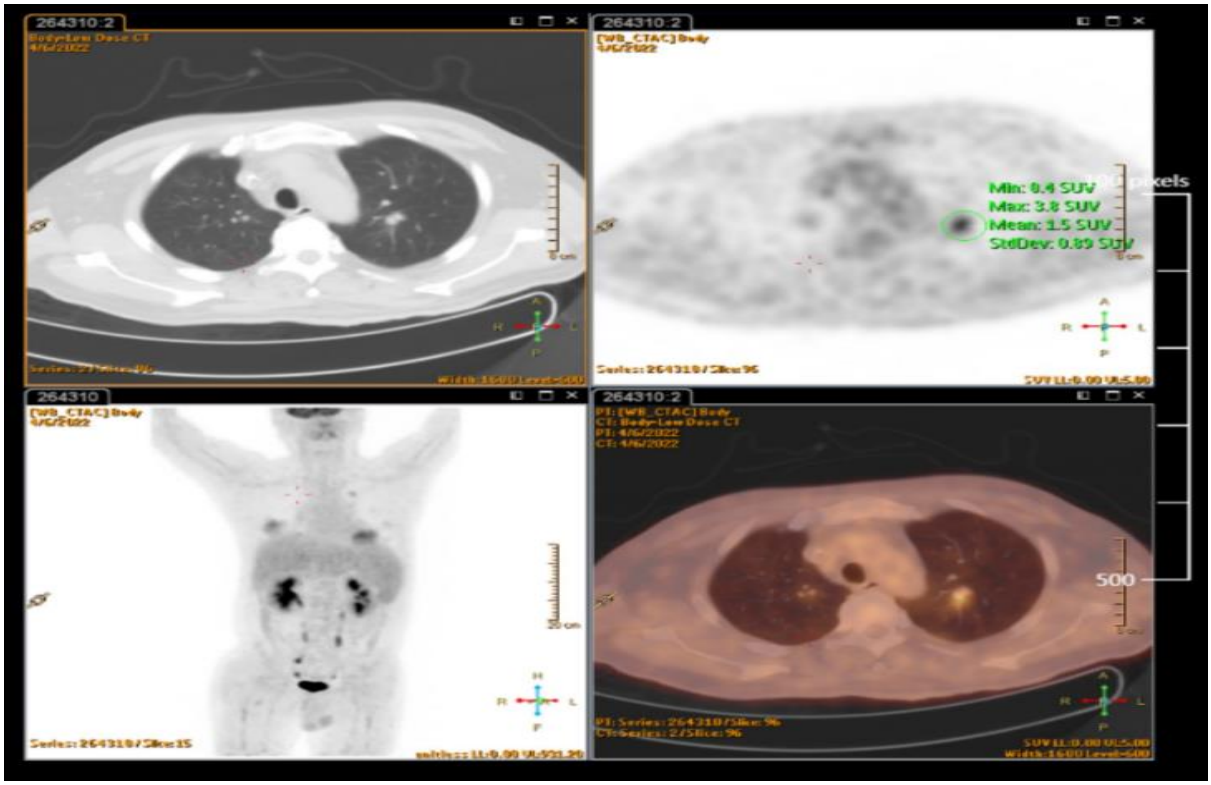

Figure 1. PET-CT revealed no abnormalities in the mediastinum. Patchy shadows can be seen in the lower lobes of both the right and left lungs. The lung abnormalities are consistent with the chest CT scan.

DISCUSSION- This case highlights the diagnostic challenge of right heart masses in AML patients. The differential included thrombosis, infective endocarditis, sarcoidosis, neoplasia, and calcified amorphous tumor (CAT). Thrombosis was unlikely due to a lack of response to anticoagulation, and imaging did not support infective endocarditis or sarcoidosis. Neoplasia was ruled out given the patient's remission and lesion stability. Gradual calcification suggested CAT, a rare non-neoplastic lesion.[2] Lee et al. reported a similar case confirmed histologically, underscoring the difficulty of diagnosing CAT non-invasively, as the current case report declined biopsy.[3]

CONCLUSION- This case highlights the complexities of diagnosing intracardiac masses in AML patients, particularly when time-sensitive treatments like allo-HSCT are required. Despite extensive imaging and multidisciplinary evaluation, the etiology of the right heart masses remained inconclusive. The increasing calcification pattern over time led to a suspected diagnosis of CAT, though confirmation was not possible due to the patient's refusal of biopsy.

Despite comprehensive imaging and multidisciplinary consultations, including echocardiography, MRI, and PET/CT, the precise nature of these calcified, mobile nodular masses in the right atrium and right ventricular lateral wall is unknown.

REFERENCES

1. Tyebally S et al. Cardiac Tumors: JACC CardioOncology State-of-the-Art Review. JACC CardioOncol. 2020 Jun 16;2(2):293-311.
2. Wang X et al. A complex case of right heart masses in a leukemia patient: a case report. J Cardiothorac Surg. 2025 Jan 10;20(1):52.
3. Lee YS et al. Cardiac Calcified Amorphous Tumor in the Left Atrium: a Case Report. J Chest Surg. 2022;55(1):95–7.