Vol 3, Issue 14, 2024

CARDIO ROUNDS NEWSLETTER

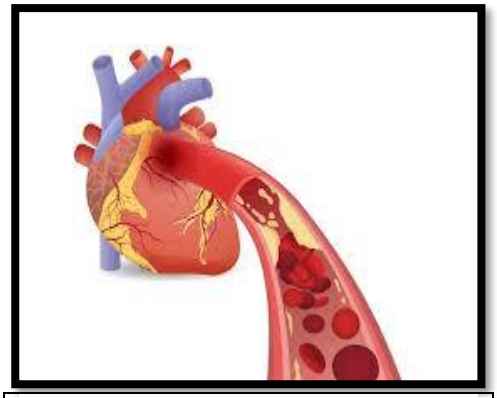
Volume 3| Issue 14| 2024

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

Predictive value of the Atherogenic Index of Plasma and Hyperuricemia for Chronic Total Occlusion



CORONARY CHRONIC TOTAL OCCLUSION

INTRODUCTION-Coronary artery disease (CAD) continues to be one of the leading causes of death and contributes significantly to the worldwide burden of illness. According to statistics, 9.44 million people worldwide passed away from CAD in 2021, posing a serious socioeconomic and public health risk.[1] Chronic total occlusion (CTO) is a severe type of CAD characterized by complete blockage of coronary arteries for at least three months. CTO is challenging to treat with percutaneous coronary intervention (PCI) due to lower success rates, higher complication risks, and uncertain clinical benefits. Identifying factors that predict CTO can be crucial in improving patient outcomes. The atherogenic index of plasma (AIP) and hyperuricemia (HUA) have been associated with CAD morbidity and mortality. However, the predictive value of AIP and HUA for CTO remains underexplored. This study investigated the individual and combined effects of AIP and HUA on predicting CTO in CAD patients.[2]

METHODS-This cross-sectional observational study enrolled 5,238 patients who underwent coronary angiography (CAG). Eligible participants were adults aged 18 to 75 with confirmed CAD. AIP was calculated using the formula: log10[triglycerides (TG)/high-density lipoprotein cholesterol (HDL-C)]. HUA was defined based on sex-specific criteria: serum uric acid ≥420 μmol/L for males and ≥360 μmol/L for females. CTO was diagnosed by cardiologists based on the presence of TIMI 0 flow in the occluded segment and a minimum three-month duration of occlusion. Data were analysed using logistic regression models to assess the relationship between AIP, HUA, and CTO, adjusted for potential confounders like age, gender, smoking status, hypertension, and other cardiovascular risk factors. Patients were stratified into four groups based on their AIP and HUA status: AIP ≤ 0.15 and non-HUA, AIP > 0.15 and non-HUA, AIP ≤ 0.15 and HUA, AIP > 0.15 and HUA. Logistic regression was used to assess the combined effect of AIP and HUA on CTO risk. Interaction between AIP and HUA was quantified using measures like the relative excess risk due to interaction (RERI), synergy index (SI), and attributable proportion (AP).

RESULTS-A total of 5,238 patients with confirmed coronary artery disease (CAD) were included in the study. Of these, 907 patients (17.3%) were diagnosed with chronic total occlusion (CTO) lesions. The cohort had a male predominance, with 75.4% being men. Patients with higher AIP levels (AIP > 0.15) were more likely to be younger, male, and smokers. They also had higher body mass index (BMI), heart rate, total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and uric acid (UA) levels, while having lower high-density lipoprotein cholesterol (HDL-C) and estimated glomerular filtration rate (eGFR) levels. Additionally, they were more likely to have a history of acute coronary syndrome (ACS), multi-

vessel disease, myocardial infarction (MI), percutaneous coronary intervention (PCI), diabetes mellitus, hyperlipidemia, hypertension, and were more frequently treated with β-blockers and hypoglycaemic therapy. CTO lesions were significantly more prevalent among patients with higher AIP and HUA. Specifically, the incidence of CTO was 28.3% in patients with AIP > 0.15, compared to only 6.8% in those with AIP ≤ 0.15 (Figure 1). Similarly, patients with HUA had a CTO incidence of 30.6%, compared to 13.0% in those without HUA. Univariate logistic regression analysis showed that both AIP and HUA were significantly associated with the presence of CTO lesions. The odds of having a CTO lesion were 5.225 times higher in patients with AIP > 0.15 (95% CI: 4.362–6.259) and 2.765 times higher in patients with HUA (95% CI: 2.350–3.254), compared to those with lower AIP and without HUA. The highest risk of CTO was observed in Group 4 (AIP > 0.15 and HUA), with an odds ratio (OR) of 11.491 (95% CI: 9.019–14.641) compared to the reference group. This indicates a significant synergistic effect between AIP and HUA on CTO risk. Subgroup analyses result consistently shown that the combined presence of high AIP and HUA was associated with a significantly higher risk of CTO across all subgroups. Interaction analysis revealed that the combined effect of AIP and HUA increased the risk of CTO by 38.5% compared to the sum of their individual effects. This synergistic effect underscores the importance of considering both AIP and HUA together when assessing CTO risk in patients with CAD.

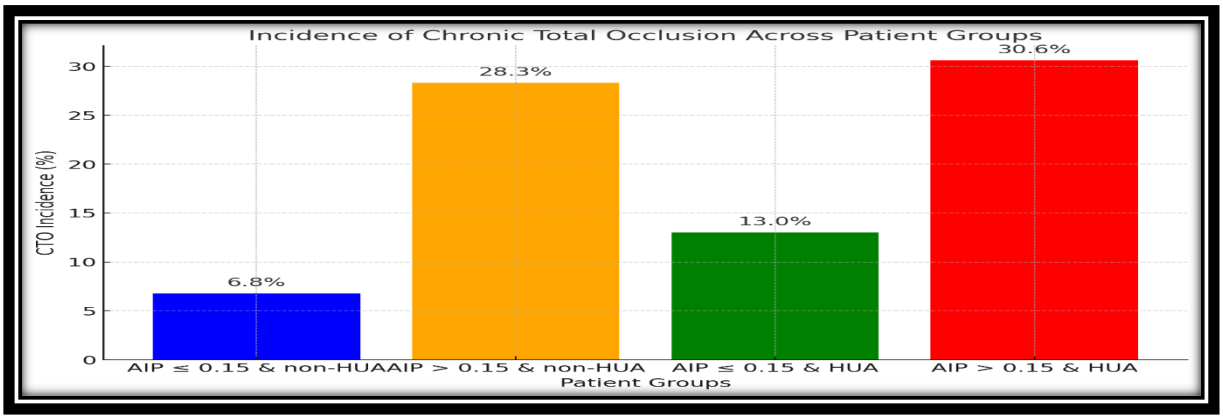


Figure 1. Bar graph illustrating the incidence of chronic total occlusion (CTO) across the four patient groups based on AIP and HUA status. The graph shows that the highest incidence of CTO is in the group with both high AIP and HUA, followed by the group with high AIP but without HUA.

DISCUSSION-The present study demonstrated that both the atherogenic index of plasma (AIP) and hyperuricemia (HUA) are significant independent predictors of chronic total occlusion (CTO) in patients with coronary artery disease (CAD).[2] Similarly, Wu T et al. reported that postmenopausal women with elevated AIP had a higher risk of developing CAD, reinforcing the link between AIP and cardiovascular disease.[3] Additionally, a meta-analysis by Braga F et al. confirmed that elevated serum uric acid levels were associated with an increased risk of CAD morbidity and mortality.[4]

CONCLUSION-This study concluded that high AIP and HUA were significantly associated with the presence of CTO in CAD patients. The combination of these two factors provides a powerful predictor for CTO risk, highlighting the need for early identification and targeted intervention in high-risk individuals. Future studies should explore the underlying

mechanisms and assess the clinical benefits of managing AIP and HUA to prevent the progression of CAD to CTO.

Patients with greater HUA and AIP levels had the highest incidence of CTO as compared to those with higher single indices. AIP and HUA together showed a strong synergistic effect on CTO lesion prediction.

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Relationship between Holter monitoring parameters and out-of-hospital major adverse cardiovascular events (MACEs) occurrence in STEMI patients

INTRODUCTION-Cardiovascular disorders are a primary cause of death globally, with acute ST-segment elevation myocardial infarction (STEMI) being one of the most severe forms of acute coronary syndromes.[1] Recent years have shown a significant increase in the survival rate following acute STEMI due to primary percutaneous coronary intervention (PPCI) and optimum medical care. Poor long-term cardiovascular outcomes, such as heart failure, arrhythmias, and cardiac death occur in certain people.[2] Assessing cardiovascular risk is essential for early intervention and better long-term results in patients receiving percutaneous coronary intervention (PCI) for acute STEMI. Continuous cardiac electrophysiological data was provided by 24-hour Holter monitoring, which makes it possible to identify autonomic dysfunction and arrhythmias that were missed by standard exams. To provide insights into the assessment of cardiovascular risk, this study aimed to investigate the association between Holter monitoring metrics and the incidence of major adverse cardiovascular events (MACEs) in patients with STEMI after PCI.[1]

METHODS-This prospective cohort study involved patients diagnosed with STEMI who subsequently underwent percutaneous coronary intervention (PCI). The primary goal was to evaluate the potential of 24-hour Holter monitoring indices in predicting MACEs, including cardiac death, over a three-year follow-up period. Patient with STEMI who were successfully treated with PCI within 24 hours of symptom onset were included. Holter monitoring was conducted 24-48 hours post-PCI, with the device worn continuously for 24 hours. The data recorded included Heart Rate (HR), Heart Rate Variability (HRV), Standard deviation of normal-to-normal intervals (SDNN), SDANN index, Deceleration Capacity (DC), Premature Ventricular Contractions (PVCs). Patients were followed for a period of three years, with the

primary outcome being the occurrence of MACEs, which included cardiac death, recurrent myocardial infarction, and heart failure hospitalization. The secondary outcome was specifically cardiac death. Receiver Operating Characteristic (ROC) analysis assessed the predictive value of HRV indices, DC, and PVCs for MACEs and cardiac death.

RESULTS-The study population included 172 patients, of which 57 (33.1%) experienced MACEs during the three-year follow-up period. Notably, 20 patients (11.6%) suffered from cardiac death. Lower SDNN (odds ratio [OR]: 0.980; p=0.005) and SDANN index (OR: 0.982; p=0.009) were protective against MACEs. In other words, a higher HRV was associated with a reduced risk of adverse events. A higher minimum heart rate was associated with an increased risk of MACEs (OR: 1.075; p=0.005), suggesting that poor autonomic regulation contributes to worse outcomes. Patients with frequent PVCs had a significantly higher risk of MACEs (OR: 2.685; p=0.016). For cardiac death, SDNN (OR: 0.957; p=0.001) and DC (OR: 0.702; p=0.017) were significant predictors, highlighting the importance of these metrics in long-term prognosis. SDNN had strong predictive power for both MACEs and cardiac death, with areas under the curve (AUC) indicating good discrimination. This suggested that HRV was a reliable marker for identifying high-risk patients. Kaplan-Meier curves demonstrated significantly poorer survival rates in patients with lower HRV (p<0.01) and higher PVC frequency (p<0.01), reinforcing the prognostic value of these indices. At the cut-off value of 19.315 ms, the SDANN index showed minimal efficacy in predicting MACEs, with an AUC of 0.531 (95% CI: 0.433–0.630), a sensitivity of 73.9%, and a specificity of 45.6%. According to these results, the SDNN index shows minimal predictive value, and the Slowest Heart Rate and SDNN were comparatively stronger predictors of MACE occurrence in this patient population (Figure 1A). Notably, at the cut-off of 65.875 ms, SDNN had an AUC of 0.752 (95% CI: 0.625–0.879), a high specificity of 90.8%, and a sensitivity of 55.0%, making it a significant predictor of cardiac death. With an AUC of 0.738 (95% CI: 0.621–0.854), a sensitivity of 50.0%, and an exceptionally high specificity of 86.8% at a 0.145 cut-off, DC4 again stood out (Figure 1B).

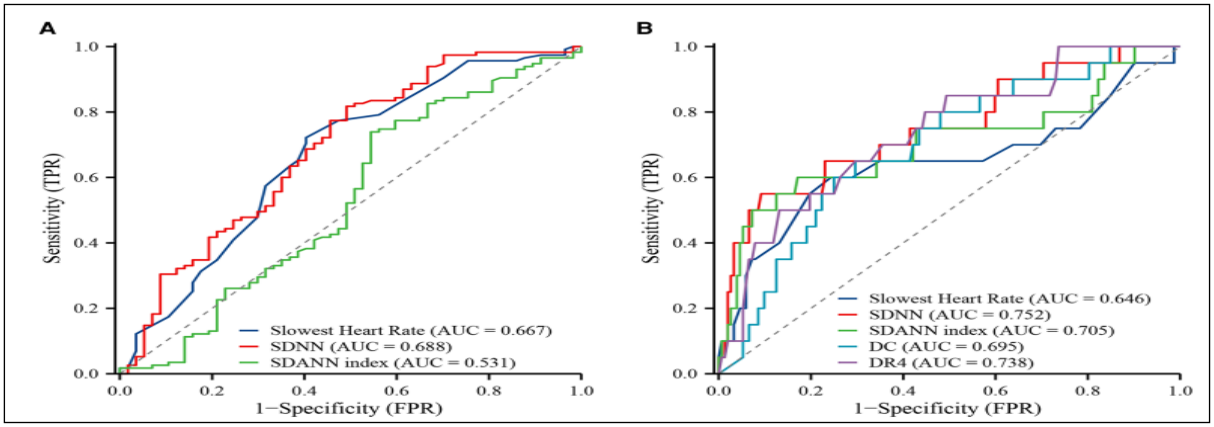

Figure 1. ROC curves for the 24-hour Holter monitoring parameters that indicate the risk of cardiovascular death and MACEs in patients with STEMI. MACEs in (A); cardiac death in (B).

DISCUSSION-According to the findings of the study, MACEs were linked to a higher lowest heart rate.[1] This result was in line with earlier study conducted by Antoni ML et al.

which hypothesized that a higher minimum heart rate could be a sign of either increased sympathetic activity or decreased parasympathetic (vagal) tone.[3]

CONCLUSION-The study concluded that HRV metrics, DC, and frequent PVCs derived from 24-hour Holter monitoring can help identify STEMI patients at high risk for MACEs after PCI. This information was crucial for clinicians aiming to improve long-term patient outcomes through early intervention and tailored post-discharge management strategies.

In STEMI patients, HRV, DC measures, and numerous PVCs discovered during 24-hour Holter monitoring were linked to an increased risk of MACEs. Clinicians can detect patients who are at risk early on with the use of these measurements, enabling prompt interventions.

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Association between Predischarge Peak Atrial Longitudinal Strain and NT-proBNP Levels as predictors of major cardiac events (MACE) in patients after Acute Heart Failure hospitalization

INTRODUCTION- Heart failure (HF) remains a significant global health issue, contributing to considerable morbidity and mortality. The post-acute heart failure (AHF) period is particularly critical, with a high risk of rehospitalization and cardiovascular mortality due to residual hemodynamic congestion.[1] Although it has a narrow specificity, N-terminal-pro-brain natriuretic peptide (NT-proBNP) is a widely recognized marker of congestion. Speckle tracking echocardiography (STE) measures peak atrial longitudinal strain (PALS), which is a marker of intracardiac pressure and heart failure prognosis.[2] This study investigates the predictive value of predischarge peak atrial longitudinal strain (PALS) and N-terminal pro-hormone brain natriuretic peptide (NT-proBNP) in forecasting major adverse cardiac events (MACE) within 90 days post-hospitalization in patients with AHF.[1]

METHODS-This single-center, prospective observational cohort study included patients aged 18 years or older who were hospitalized for AHF, either as a new or recurrent episode, regardless of the heart failure phenotype. Patients with heart failure with preserved ejection fraction (HFpEF), heart failure with mildly reduced ejection fraction (HFmrEF), and heart failure with reduced ejection fraction (HFrEF) were all eligible for inclusion. The study

included echocardiographic assessments and plasma NT-proBNP measurements within 24 hours before discharge. PALS was measured using speckle-tracking echocardiography to assess left atrial reservoir function, and NT-proBNP levels were quantified using an enzyme-linked immunosorbent assay. Patients were followed for 90 days post-discharge to monitor the occurrence of MACE, defined as rehospitalization or cardiovascular mortality. Statistical analyses included receiver operating characteristic (ROC) curves, Kaplan-Meier survival analysis, and Cox regression modeling to identify independent predictors of MACE.

RESULTS-This study included 67 patients, with a mean age of 56.88 ± 14.57 years. The cohort was predominantly male (65.7%). Of the patients, 59.7% were admitted due to recurrent heart failure, while 40.3% were new cases. Most patients (61.2%) had heart failure with reduced ejection fraction (HFrEF), while 23.9% had HFpEF, and 14.9% had HFmrEF. Hypertension was the most common comorbidity (62.7%), followed by dyslipidemia (44.8%), smoking (43.3%), diabetes mellitus (34.3%), and coronary artery disease (23.9%). During the 90-day follow-up period, 22 patients (32.8%) experienced MACE. Of these, 21 patients (31.3%) were rehospitalized due to worsening heart failure, and one patient (1.5%) died from sudden cardiac death at home. The analysis revealed significant differences between patients who experienced MACE and those who did not, particularly in terms of left atrial volume index (LAVi), left ventricular volume index (LVVi), and predischarge NT-proBNP levels, which were higher in the MACE group. Additionally, the MACE group had lower PALS and LVEF values. ROC curve analysis identified PALS <12% and NT-proBNP >750 pg/mL as optimal thresholds for predicting MACE (Figure 1). PALS had an area under the curve (AUC) of 0.816, with a sensitivity of 77.3% and a specificity of 75.6%. NT-proBNP had an AUC of 0.856, with a sensitivity of 81.8% and a specificity of 77.8%. There was no significant difference between the AUCs of PALS and NT-proBNP (P = 0.553). When both PALS <12% and NT-proBNP >750 pg/mL were combined, the specificity increased to 93.3%, though sensitivity decreased to 63.4%. Kaplan-Meier survival curves demonstrated that patients with PALS <12% had significantly lower survival free of MACE compared to those with PALS ≥12% (43.3% vs. 86.5%; P < 0.001). Similarly, patients with NT-proBNP >750 pg/mL had lower survival rates compared to those with NT-proBNP ≤750 pg/mL (35.7% vs. 89.7%; P < 0.001). Only 17.6% of patients with both low PALS and high NT-proBNP levels were free from MACE during the follow-up period. Cox proportional hazards models identified NT-proBNP >750 pg/mL (adjusted HR: 12.696, 95% CI: 3.446–46.771, P < 0.001) and PALS <12% (adjusted HR: 4.617, 95% CI: 1.394–15.287, P = 0.012) as significant independent predictors of MACE. Additional independent predictors included a history of coronary artery disease (CAD) and diabetes mellitus (DM).

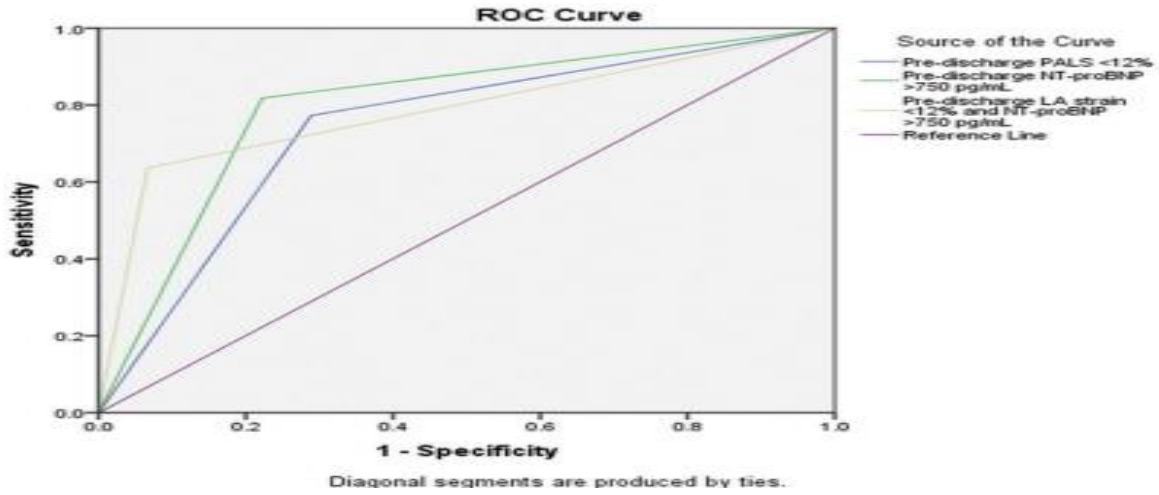

Figure 1. NT-proBNP (nucleotide prohormone brain natriuretic peptide) >750 pg/mL, PALS <12%, and a combination of both NT-proBNP and PALS > 750 pg/mL were compared in terms of receiver operating characteristic curves among patients. NT-proBNP = N-terminal prohormone brain natriuretic peptide, PALS = Peak atrial longitudinal strain, and ROC = Receiver Operating Characteristic

DISCUSSION-The current study underscored the utility of predischarge PALS and NT-proBNP levels as independent predictors of short-term adverse outcomes in patients with AHF.[1] Similar to this, Park et al. analysed the predictive power of PALS in a large cohort of patients with acute heart failure (AHF) using data from the AHF registry in Korea (2009–2016). The study included 3,818 patients and found that those with PALS values between ≥8.8% and <16.5% had a 1.357 times higher hazard ratio (HR) for rehospitalization or all-cause mortality compared to those with higher PALS values. Patients with PALS values <8.8% had the highest risk of adverse outcomes, with an adjusted HR of 3.818.[3]

CONCLUSION-This study concluded that predischarge PALS and NT-proBNP serve as valuable, independent predictors of MACE in AHF patients, with potential implications for risk stratification and management strategies. These measures could be integrated into routine clinical practice to enhance short-term outcome prediction and guide therapeutic decision-making.

Before discharging, like NT-proBNP levels, PALS is an independent predictor of short-term MACE following AHF hospitalization.

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Case report on COVID-19-related severe necrotizing encephalopathy and acute myocarditis

INTRODUCTION-In December 2019, a case of pneumonia brought on by an infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was reported in Wuhan City, Hubei Province, China. The World Health Organization (WHO) formally designated the illness brought on by a SARS-CoV-2 infection as coronavirus disease 2019 (COVID-19) on February 11, 2020.[1] COVID-19, caused by the SARS-CoV-2 virus, primarily affects the respiratory system but has been increasingly recognized for its potential to cause severe extrapulmonary complications, including cardiovascular and neurological conditions. Among these complications, myocarditis and acute necrotizing encephalopathy (ANE) are rare but life-threatening manifestations. The underlying mechanisms of these conditions are not fully understood, but they may involve direct viral invasion, immune-mediated damage, or both. Here, a novel case of simultaneous COVID-19-related myocarditis and ANE in a young adult female, highlighting the need for prompt diagnosis and aggressive treatment to improve patient outcomes.[2]

CASE PRESENTATION-A 27-year-old woman with a two-month history of depression and recent positive SARS-CoV-2 antigen test presented to the emergency department with a two-day history of fever and a nine-hour episode of altered consciousness. Her initial symptoms included intermittent fever with a maximum body temperature of 39.5°C, accompanied by a sore throat but without other respiratory symptoms like chest tightness or pain. Despite taking antipyretics, her fever persisted with minimal improvement. Approximately nine hours before presenting to the emergency department, she began to experience reduced consciousness. Upon arrival at the hospital, her physical examination revealed a Glasgow Coma Scale score of 13 (E4/V4/M5), indicating a reduced level of consciousness. Her vital signs were notable for a body temperature of 39°C, tachycardia at 130 beats per minute, hypotension with a blood pressure of 102/60 mmHg, a respiratory rate of 26 breaths per minute, and oxygen saturation of 94% on room air. Cardiac examination revealed signs of hemodynamic instability, including cool extremities, jugular venous distention, a ventricular gallop rhythm, and rales at the lung bases, without murmurs. Neurological examination was significant for nuchal rigidity and bilateral positive Babinski signs, though her pupils were reactive to light. Initial laboratory investigations showed normal white blood cell, red blood cell, and platelet counts but notable absolute lymphopenia. Inflammatory markers were elevated, including C-reactive protein (CRP) at 16.50 mg/L and interleukin 6 (IL-6) at 668.36 pg/ml. A nasopharyngeal swab confirmed the presence of SARS-CoV-2 through reverse transcription polymerase chain reaction (RT-PCR), with low cycle threshold (Ct) values indicating a high viral load. Imaging studies revealed significant findings: whole-body computed tomography (CT) showed hypodensity in the right basal ganglia region and

signs of bilateral pneumonia. An electroencephalogram (EEG) demonstrated mild to moderate abnormal brainwave patterns, consistent with encephalopathy. Transthoracic echocardiography (TTE) revealed severe left ventricular dysfunction with an ejection fraction of 17%, and an electrocardiogram (ECG) showed sinus tachycardia, poor R-wave progression, and mild ST-T changes, without indications of acute coronary syndrome (Figure 1). Within four hours of admission, her condition deteriorated rapidly. She became stuporous and developed shortness of breath, progressing to cardiogenic shock. Orotracheal intubation was performed immediately, and she was placed on mechanical ventilation with settings that included a fraction of inspired oxygen (FiO2) of 60% and positive end-expiratory pressure (PEEP) of 10 cm H2O. Despite these measures, her hemodynamic status remained precarious, necessitating the initiation of veno-arterial extracorporeal membrane oxygenation (ECMO) to provide circulatory support. Upon admission to the intensive care unit (ICU), the patient exhibited severe hemodynamic instability, with sinus tachycardia at 150 beats per minute and hypotension (90/46 mmHg) despite norepinephrine infusion. Blood tests revealed elevated lactate levels, indicative of poor tissue perfusion, and significantly elevated cTnI levels (4.98 ng/ml), suggesting ongoing myocardial injury. Central venous oxygen saturation (ScvO2) was severely impaired, and a high central venous-to-arterial carbon dioxide tension difference (PCO2 gap) indicated inadequate tissue oxygenation. Given the critical nature of her condition, bedside ultrasound-guided femoro-femoral ECMO was initiated, which led to stabilization of her hemodynamics. Subsequent next-generation sequencing (NGS) of bronchoalveolar lavage fluid confirmed the presence of the SARS-CoV-2 virus, specifically identifying the Omicron EG.5.1 variant. The patient was started on broad-spectrum antibiotics and antiviral therapy with molnupiravir, alongside immunosuppressive therapy with intravenous methylprednisolone and intravenous immunoglobulin (IVIG). Over the following days, the patient showed some improvement in cardiac function, with a gradual reduction in cardiac injury markers. On day 13, she was successfully weaned from ECMO. However, her neurological status remained poor, and she continued to be comatose. A lumbar puncture performed on day 15 revealed normal cerebrospinal fluid (CSF) findings, including no pleocytosis, normal protein and glucose levels, and negative results for SARS-CoV-2 and other pathogens. Despite this, MRI on day 25 showed bilateral, symmetrically distributed lesions in the pons, bilateral hippocampal regions, and bilateral basal ganglia, consistent with acute necrotizing encephalopathy (ANE). Despite the stabilization of her cardiac function, her neurological condition did not improve significantly. A follow-up CT scan on day 31 showed partial resolution of the brain and chest lesions. On day 32, the patient was discharged to a rehabilitation facility for further management.

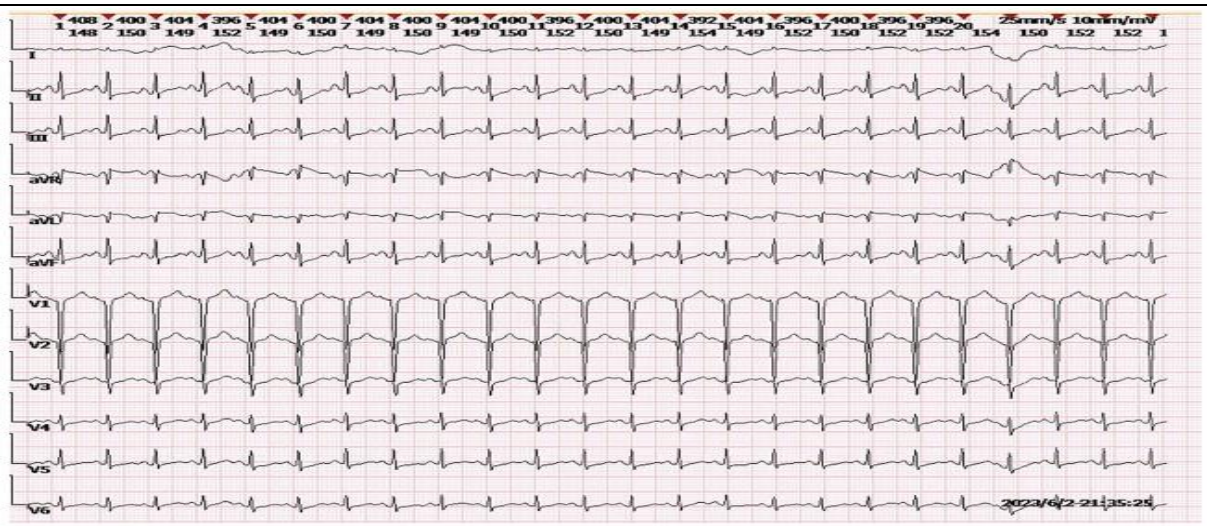

Figure 1. Twelve-lead ECG revealed modest ST-T alterations, poor R-wave progression in V1-3, and sinus tachycardia. ECG measured at 10 mm/mV and 25 mm/s.

DISCUSSION-This case report highlighted the rare occurrence of simultaneous myocarditis and acute necrotizing encephalopathy (ANE) in a patient with COVID-19. The rapid progression and severity of these conditions in this patient underscore the complexity of managing COVID-19 when it affects multiple organ systems. The patient's successful stabilization with the use of veno-arterial extracorporeal membrane oxygenation (ECMO) and aggressive immunosuppressive therapy provides valuable insights into potential therapeutic strategies for such severe cases.[2] In a case report by Poyiadji N et al., a female patient with COVID-19 developed acute haemorrhagic necrotizing encephalopathy, characterized by symmetric thalamic lesions. This patient also presented with severe neurological symptoms, including altered mental status and seizures, like present case patient. Like current case, the MRI findings in Poyiadji's report were crucial in diagnosing ANE, showing characteristic brain lesions. However, unlike current case patient, the case reported by Poyiadji N et al. did not involve myocarditis, highlighting the variability in the clinical presentation of COVID-19-related complications.[3]

CONCLUSION-The current case report concluded that the simultaneous occurrence of myocarditis and ANE in COVID-19 patients was rare but associated with high mortality. Early and accurate diagnosis, coupled with timely intervention, is crucial for improving survival outcomes. This case emphasized the importance of multidisciplinary management in severe COVID-19 cases and the need for continued research into the pathophysiology and treatment of COVID-19-related extrapulmonary complications.

COVID-19 may have negative effects on the cardiovascular and central nervous systems at the same time and with rapidity. When required, prompt invasive bridging therapy and an accurate diagnosis can save lives. It will be crucial to investigate potential processes underlying COVID-19 symptoms in the cardiovascular and central neurological systems (CNS).

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