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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

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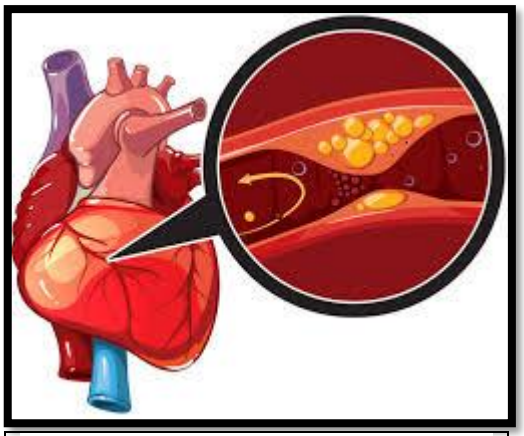
Correlation between high-sensitivity C-reactive protein and the atherogenic index of plasma for the degree of coronary artery lesion in early-stage coronary artery disease

INTRODUCTION- Coronary artery disease (CAD), commonly known as coronary heart disease (CHD) or ischemic heart disease, is a common condition that ranks among the top causes of death globally. Cardiac risk stratification is required to better preventative and therapeutic measures. Biomarkers are an important component in this activity, whether as part of a prediction model or by themselves. Several CAD risk indicators have been identified and studied in recent years, including C-reactive protein, lipoprotein-associated phospholipase A2, and fibrinogen, among others.[1] High-sensitivity C-reactive protein (hs-CRP) is a liver-produced acute phase response protein that is one of the most sensitive biomarkers of human non-specific inflammatory response. It can be used as an independent predictor of CAD development and death. The atherogenic index of plasma (AIP), a sensitive indication of dyslipidemia, was closely related to the development of cardiovascular illnesses. A prior study found a link between AIP, the start of CAD, and the degree of human atherosclerosis. Nonetheless, the relationship between AIP and the degree of coronary artery damage in premature coronary artery disease (PCAD) patients is unclear. So, the study aimed to assess the efficacy of the AIP and hs-CRP in predicting the development and severity of coronary artery lesions in patients with PCAD.[2]



Coronary Artery disease

METHODS-This retrospective study involved 238 patients, including 163 patients diagnosed with PCAD and 75 non-PCAD patients who served as the control group. The study focused on patients under 55 years of age for men and under 65 years of age for women, aiming to evaluate the correlation between plasma levels of the AIP and high-sensitivity C-reactive protein (hs-CRP) with the severity of coronary artery lesions. Patients were divided into groups based on their Gensini score (GS), a commonly used scoring system that quantifies the severity of coronary artery disease based on coronary angiography results. Higher GS values indicate more severe coronary artery lesions. The plasma levels of hs-CRP and AIP were measured for each patient. AIP was calculated using the logarithmic ratio of triglycerides to high-density lipoprotein cholesterol (HDL-C). Correlations between these markers and GS were analysed using Spearman's correlation to assess the relationship between inflammatory and lipid factors with the severity of coronary artery disease. Logistic regression analysis was performed to identify independent risk factors for PCAD. The predictive value of hs-CRP and AIP for diagnosing and assessing the severity of PCAD was determined using receiver operating characteristic (ROC) curves, with area under the curve (AUC) values used to evaluate the accuracy of these markers individually and in combination.

RESULTS -The study results found that patients with PCAD had significantly higher levels of hs-CRP and AIP compared to the control group, indicating a strong association between these markers and the presence of coronary artery disease. The median GS was higher in the PCAD group, reflecting more severe coronary artery lesions. Spearman's correlation analysis revealed a significant positive correlation between both hs-CRP and AIP levels with the Gensini score ($p < 0.05$) (Figure 1). This suggests that as levels of these markers increase, the severity of coronary artery lesions also increases. Logistic regression analysis identified multiple independent risk factors for PCAD, including smoking, elevated homocysteine levels, fasting blood glucose, hs-CRP, and AIP. Among these, hs-CRP and AIP were the most strongly associated with PCAD. ROC analysis demonstrated that both hs-CRP and AIP have good predictive value for PCAD when considered separately, with AUC values indicating significant diagnostic accuracy. However, the combination of hs-CRP and AIP further improved the predictive power, offering a more precise assessment of both the presence and severity of PCAD. This combined model showed a higher AUC, suggesting that using both markers together provides enhanced diagnostic performance compared to using either marker alone.

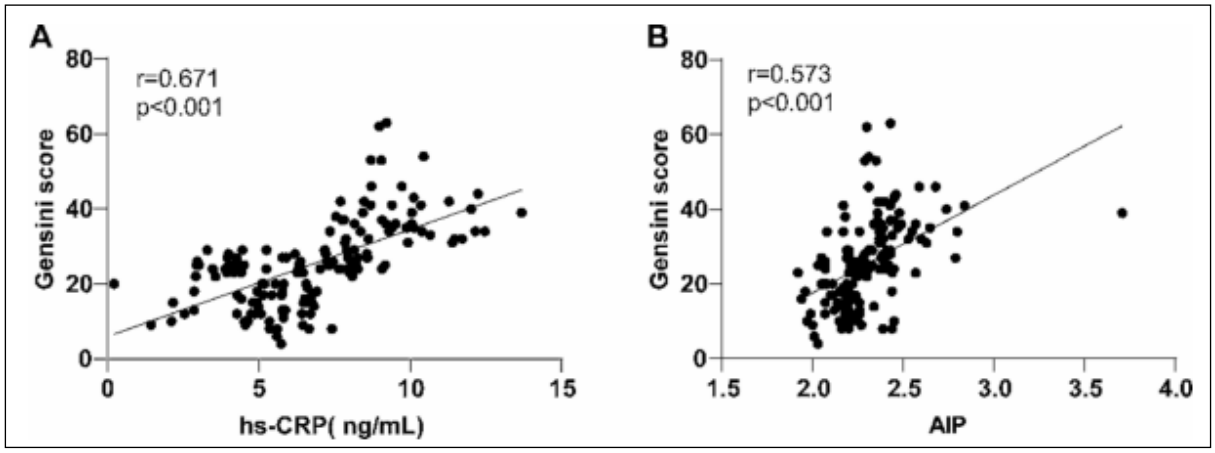


Figure 1. Plasma hs-CRP and AIP were correlated with the Gensini score in PCAD patients. (A-B) Using Spearman correlation analysis, the relationships between plasma hs-CRP and AIP with Gensini score were examined. Panels A–B's data were all examined using the Spearman coefficient.

DISCUSSION- This study showed that the degree of coronary artery lesion and the likelihood of developing PCAD were directly connected, and the combination of AIP and hs-CRP has a significant predictive value for both PCAD occurrence and disease severity.[2] Similar to this, Askin L et al. showed that combining AIP with traditional lipid measurements, it has been found to be a more accurate predictor of cardiovascular illnesses than either HDL-C or TG. This finding has significant implications for the prevention and therapeutic guidance of cardiovascular disorders.[3]

CONCLUSION- The current study concluded that AIP and hs-CRP were significant independent predictors for PCAD. Their combination offers enhanced predictive accuracy for assessing both the occurrence and severity of coronary artery lesions in PCAD patients, making them valuable tools for early diagnosis and management.

AIP and hs-CRP are two independent risk factors for PCAD development; when combined, they have a strong predictive value for PCAD occurrence and disease severity, which are both positively connected with the extent of coronary artery lesion.

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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).

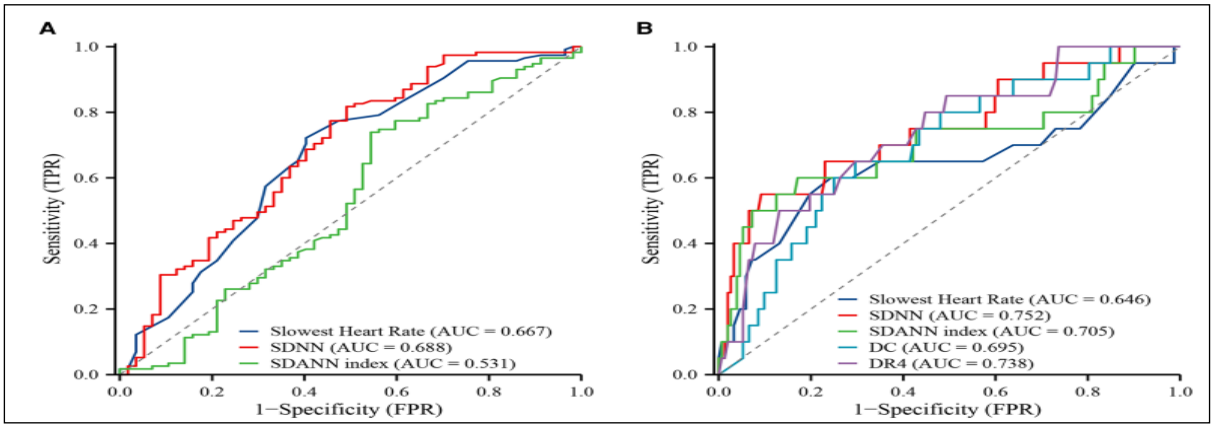

Figure 1. ROC curves for the 24-hour Holter monitoring parameters that indicate the risk of cardiovascular death and MACEs in patients with STEMI. MACEs in (A); cardiac death in (B).

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).

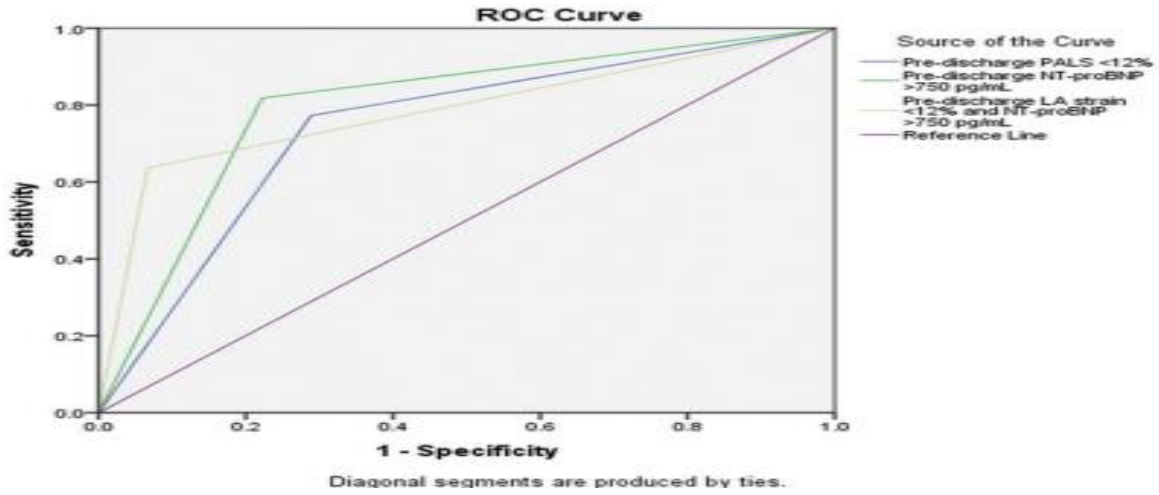

Figure 1. NT-proBNP (nucleotide prohormone brain natriuretic peptide) >750 pg/mL, PALS <12%, and a combination of both NT-proBNP and PALS > 750 pg/mL were compared in terms of receiver operating characteristic curves among patients. NT-proBNP = N-terminal prohormone brain natriuretic peptide, PALS = 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 constrictive pericarditis in a patient with fiberglass lung disease

INTRODUCTION-Constrictive pericarditis (CP) is a type of diastolic heart failure that occurs when an inelastic pericardium prevents cardiac filling. This condition should be explored in the differential diagnosis of unexplained heart failure, especially if the left ventricular ejection fraction is retained. Prior heart surgery and radiation therapy are risk factors for developing CP, however most cases are still considered idiopathic.[1] Fiberglass is less likely to enter the lungs by inhalation because it has a wider aerodynamic diameter. Moreover, even if it is mechanically split into smaller bits and breathed into the lungs, it will still be eliminated. While long-term exposure to fiberglass has been well-documented, the condition was once believed to be treatable and not likely to result in serious illness. Histopathological results and exposure history are key factors in the diagnosis of fiberglass lung disease. Nevertheless, because mixed chemical exposure is common and fiberglass disease is less well-known than asbestosis, it might be challenging to pinpoint the precise occupational exposure history.[2] This case report described a 66-year-old patient who developed constrictive pericarditis and lung fibrosis because of fiberglass lung exposure.[2]

CASE REPORT-The 66-year-old man ex-smoker, who worked as a medical equipment operator and self-reported 40 years of significant asbestos exposure, arrived at the hospital with a huge transudative pericardial effusion. Two pleural–pericardial windows were used to treat the condition at another institution. His final histological diagnosis was asbestosis, made during the second pleural–pericardial window following a lung biopsy. But over the course of the next 18 months, his dyspnea and leg edema grew worse. Peristalsis constriction was verified by cardiac catheterization. The patient was admitted because of the congestive liver's growing degradation of liver function and poor response to diuretics. When the patient was first admitted, a computed tomography (CT) scan of the chest showed parenchymal fibrosis bands, thicker intra- and interlobular lines, bilateral pleural effusion, pericardial effusion, and pericardial thickening. Nevertheless, no overt pleural calcification or plaque was seen, which are classic indicators of asbestosis (Figure 1A). A transudative pleural effusion was discovered during thoracentesis, however there was no mesothelioma evidence. Tests for pulmonary function showed a restricted trend. As a result, the patient had an open lung biopsy in addition to a drastic pericardiectomy. Along with his lungs being extremely frail, the surgeons saw that the pericardium was as rigid as corrugated paper and was securely linked to the epicardium. There were no asbestos or ferruginous bodies seen in the lung pathology, but there was minor interstitial fibrosis of the lung and pleura. Different translucent particles were found in the lung parenchyma using polarized microscopy. Upon pathology investigation, the epicardium and pericardium were found to be significantly swollen, together with a few small translucent particles. Pathological specimens from the other hospital were re-examined, and the results

showed that there was only one ferruginous body and no asbestos bodies. Transparent particles of different sizes were found using polarized microscopy. Every test for fungus, TB, and autoimmune illnesses came back negative. Following debate, it was suggested that the translucent particles might be fiberglass. As a result, a thorough review of the patient's work exposure history was conducted. He claimed that although he had been exposed to fiberglass extensively throughout the previous 40 years, he had only been exposed to asbestos very little during the first three years. Ultimately, it was determined that the patient had fiberglass lung illness rather than asbestosis. His symptoms, pleural effusion, and pulmonary function finally subsided ten months following the radical pericardiectomy (Figure 1B).

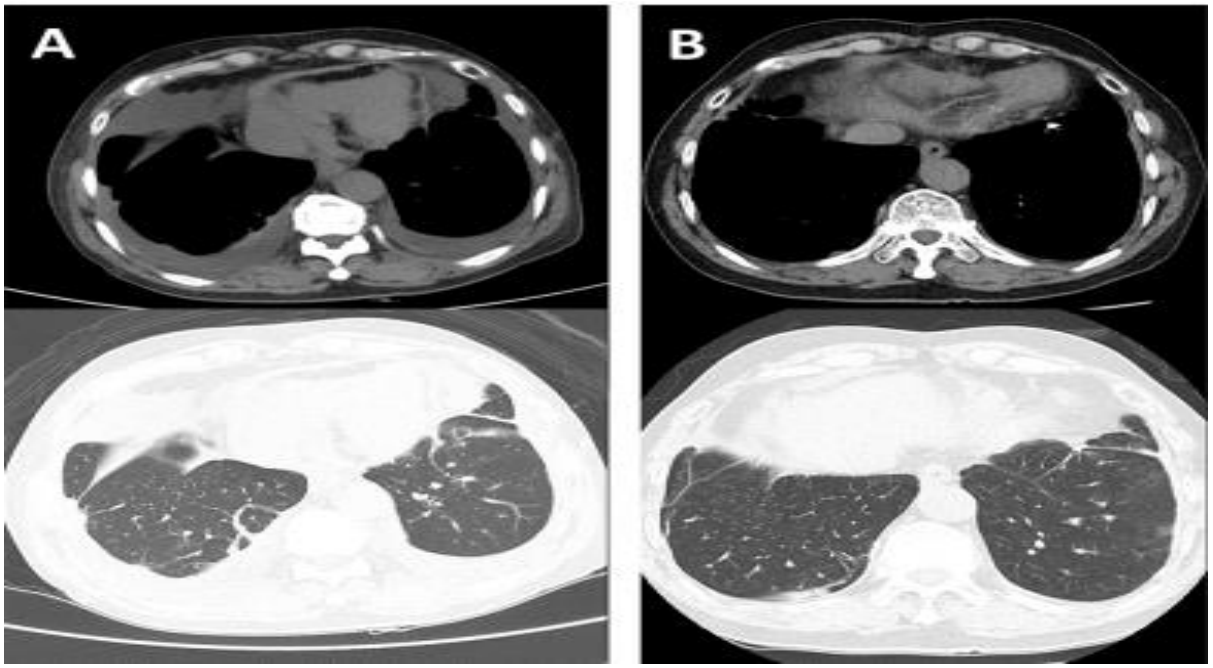

Figure 1. HRCT imaging. The results of the chest computed tomography revealed a modest amount of pericardial effusion, bilateral pleural effusion with some longitudinal consolidation, and a calcified granuloma within the pleura in the left lower lobe (A). After surgery, there was an improvement in pleural effusion, pericardial effusion, and linear consolidation (B).

DISCUSSION-This case highlighted a unique instance of constrictive pericarditis caused by long-term fiberglass exposure, a condition that was rarely reported in the literature.[2] Similar to previous case reports, Rapisarda et al., this case demonstrated that while fiberglass was generally considered less harmful than asbestos, chronic exposure can lead to significant respiratory and cardiac complications.[3]

CONCLUSION-The current study concluded that fiberglass has the potential to inflame the pericardium, leading to pericardial effusion and, in turn, severe constrictive pericarditis that necessitates a major pericardiectomy. For a precise diagnosis, doctors must carefully review the exposure history and perform histological exams.

Fiberglass has the potential to inflame the pericardium, which can lead to pericardial effusion and, in turn, severe constrictive pericarditis that needs a major pericardiectomy. For a precise diagnosis, doctors must carefully review the exposure history and perform histological exams.

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