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

Relationship between Fine Particulate Matter Components and the Estimated 10-Year Risk of Atherosclerotic Cardiovascular Disease

INTRODUCTION

Cardiovascular disease

Globally, cardiovascular diseases (CVDs) are the most common chronic illness and the leading cause of mortality and disease burden. Atherosclerotic cardiovascular disease (ASCVD) was found to be the primary cause of cardiovascular diseases in 2017, for around 17.8 million deaths globally.[1] A major part of ASCVD prevention is risk assessment. 10-year ASCVD risk refers to the chance of experiencing an ASCVD incident for the first time in ten years for people who did not have ASCVD at baseline.[2] Particulate matter measuring less than or equal to 2.5 μm in diameter ($PM_{2.5}$), is a complex combination mostly made up of ammonium (NH_4^+), sulphate (SO_4^{2-}), organic matter (OM), black carbon (BC), and soil particles (SOIL) in addition to nitrate (NO_3^-). There is growing evidence of harmful health effects from prior research on $PM_{2.5}$ and its components.[1] This study aimed to assess (1) the relationships between 10-year ASCVD risk and $PM_{2.5}$ and its six constituents; (2) which constituents are more lethal to 10-year ASCVD; and (3) the effects of reallocating $PM_{2.5}$ constituents.

METHODS

This was a Large-Scale Cross-Sectional Study. A total of 31,162 participant's enrolment was utilized to identify relationships between $PM_{2.5}$ and its components and ASCVD. The 3-year average concentration of $PM_{2.5}$ and its constituents—black carbon [BC], nitrate [NO_3^-], ammonium [NH_4^+], inorganic sulphate [SO_4^{2-}], organic matter [OM], and soil particles [SOIL]—were estimated through the use of hybrid machine learning. The most dangerous ingredient was determined by analysing the relationships between $PM_{2.5}$ constituents and 10-year ASCVD risk using constituent concentration, percentage, and residual models. To examine the effect of replacement between $PM_{2.5}$ elements, the isochronous substitution model (ISM) was utilized. Less than 0.05 two-sided p-values were considered statistically significant.

RESULTS

The current study showed that a 3.5%, 49.3%, 19.4%, 10.5%, 21.4%, 14%, and 28.5% greater 10-year ASCVD risk was linked to each 1 μg/m^3 increase in $PM_{2.5}$, BC, NH_4^+, NO_3^-, OM, SO_4^{2-}, and SOIL, respectively (all $p < 0.05$). Similar outcomes were noted for the residual and

proportion models. A linear relationship between BC and high 10-year ASCVD risk, as well as curvilinear relationships between $PM_{2.5}$ and other components with high 10-year ASCVD risk, were shown by the exposure-response connections of the 3-year average concentration of $PM_{2.5}$ and its six constituents with 10-year ASCVD risk (Figure 1). According to subgroup analysis, people with low levels of physical activity, education, and average monthly income showed stronger relationships. The ISM discovered that substituting other ingredients for BC will have the biggest positive effects on health.

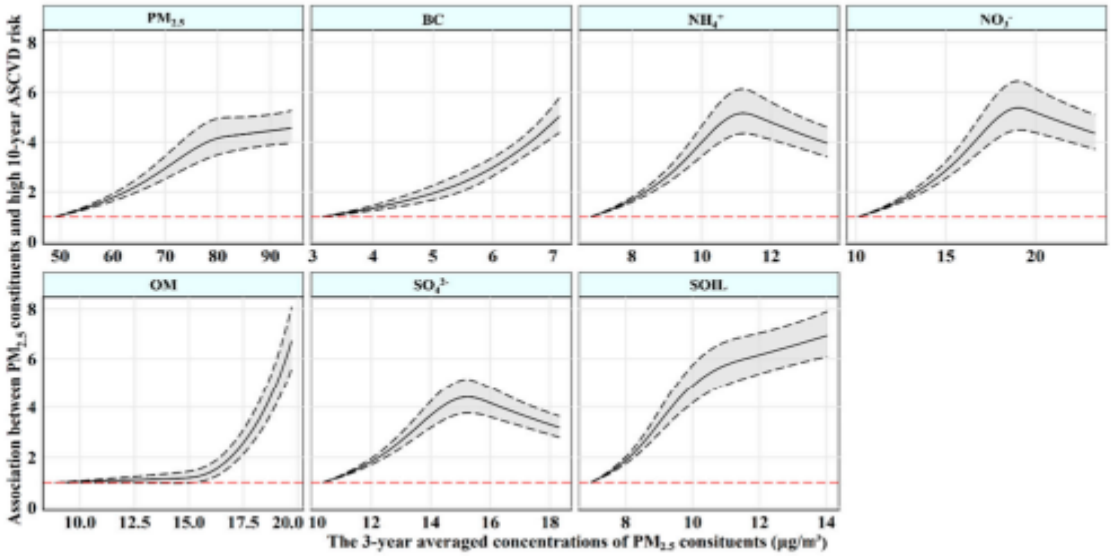

Figure 1. The 3-year averaged $PM_{2.5}$ constituent's concentration exposure-response relationships with a high 10-year ASCVD risk. Abbreviations: $PM_{2.5}$—fine particulate matter; BC—black carbon; NH_4^+—ammonium; NO_3^-—nitrate; OM—organic matter; SO_4^{2-}—sulfate; SOIL—soil particles; ASCVD—atherosclerotic cardiovascular disease.

DISCUSSION

The current study showed that Long-term exposure to $PM_{2.5}$ and its components was linked to a higher 10-year risk of ASCVD.[1] Similar to this study, Li J et al. demonstrated that a high 10-year ASCVD risk was positively correlated with long-term exposure to $PM_{2.5}$ mass, black carbon, ammonium, nitrate, organic matter, sulphate, and soil particles.[2]

CONCLUSION

The current study concludes that the risk of ASCVD linked to extended exposure to $PM_{2.5}$ and its six components (BC, NO_3^-, NH_4^+, OM, SO_4^{2-}, and SOIL), with BC playing a key part.

Populations with low average monthly income, poor educational attainment, and low physical activity levels showed stronger relationships between Fine particulate matter and Atherosclerotic cardiovascular disease, suggesting the need of reducing $PM_{2.5}$ pollutions in these vulnerable groups.

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Association between Mean Platelet Volume and mortality among Peritoneal Dialysis patients

INTRODUCTION

Platelets are important in haemostasis and thrombosis, and their function is influenced by a variety of factors, including platelet size. Mean platelet volume (MPV) is a platelet size metric that may be easily measured in a complete blood count test.[1] In comparison to hemodialysis (HD), peritoneal dialysis (PD) is a significant therapy option for individuals with chronic kidney disease (CKD) that may offer benefits in terms of cost, logistics, and clinical outcomes. Approximately 50% of these individuals' deaths and 33% of their hospital stays are attributable to cardiovascular disease (CVD).[2] In the general population, elevated MPV has been linked to an increased risk of significant adverse cardiovascular events and mortality in patients with acute coronary syndrome, as well as ischemic stroke and hypertension. Increased MPV in diabetic individuals has been associated with poor glycaemic control and a higher risk of diabetic complications. Cardiovascular morbidity and death are more likely to occur in hemodialysis patients, and MPV has been identified as a promising biomarker for predicting difficulties in these individuals.[1] This study aimed to assess the relationship between MPV and cardiovascular and overall mortality in Peritoneal Dialysis (PD) patients.

METHODS

A total of 1322 patients were recruited in this study. Age, gender, and previous comorbidities including diabetes and premorbid CVD were recorded under the patients' demographic data. The end-stage renal disease (ESRD) etiology, body mass index (BMI), Charlson Comorbidity Index (CCI), hypertension, estimated glomerular filtration rate (eGFR), white blood cell count, platelet count, neutrophils, lymphocytes, haemoglobin, creatine, urea nitrogen, albumin, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), phosphate, intact parathyroid hormone (iPTH), and antiplatelet drugs were among the principal clinical and biological parameters evaluated. Before PD therapy started, baseline data were gathered. Mortality from all causes and cardiovascular disease were the main outcomes. According to the cut-off value, which was established using maximally selected rank statistics, patients were separated into two groups. The Cox regression models were used to evaluate the mortality hazard ratio.

RESULTS

The current study results showed that the average age of the 1322 PD patients who were enrolled was 49.3 ± 14.5 years, 57.6% of them were men, and 18.8% of them had diabetes. Chronic glomerulonephritis, which affected 66.1% of this group, was the primary cause to ESRD, followed by diabetic nephropathy (14.7%) and hypertension (12.0%). 360 individuals passed away during a median follow-up of 50 months (IQR: 30-80); 167 of these deaths were related to cardiovascular illnesses. According to correlation analyses, MPV had a negative correlation with the NLR ($r=-0.072$, $p=0.009$), cholesterol ($r=-0.098$, $p<0.001$), platelet count ($r=-0.266$, $p<0.001$), and LDL-C ($r=-0.072$, $p<0.001$). All-cause and cardiovascular death rates were lower in the higher-MPV group than in the lower-MPV group, according to a survival analysis ($p<0.001$ and $p<0.001$, respectively) (Figure 1). A larger MPV was substantially linked to a hazard ratio of 0.77 for all-cause death and 0.75 for cardiovascular mortality after full adjustment (95% CI: 0.60-0.98, $p=0.036$). Age and MPV had a significant interaction, according to subgroup analysis ($p<0.001$). Only in patients under 60 years old had decreased MPV, linked to a higher death risk.

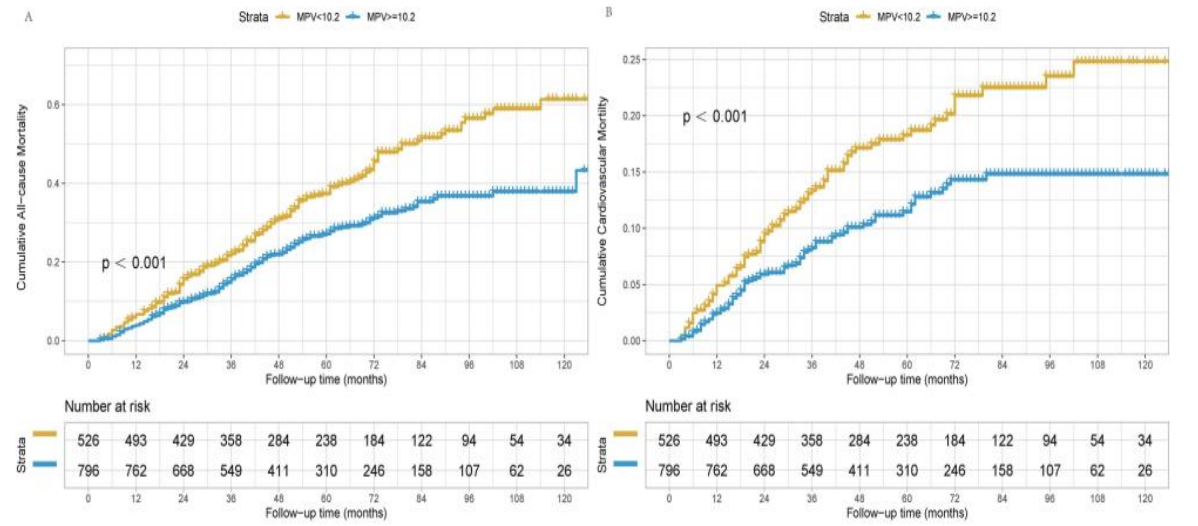

Figure 1. Patient survival curves Stratified by MPV (A) Cumulative all-cause mortality (B) Cumulative cardiovascular mortality

DISCUSSION

The current study showed that PD patients with greater MPV levels had a considerably reduced risk of cardiovascular and all-cause death than PD patients with lower MPV levels. Similar to this, Chen J et al. showed that in PD patients less than 60 years old, decreased MPV/PC level was an independent risk factor for all-cause and CV death.[3] Zhu Y et al. showed that increased MPV or platelet count linked to both cardiovascular death and all-cause death. [4]

CONCLUSION

This study concludes that PD patients who have high MPV have a lower risk of cardiovascular and all-cause mortality. This study finding add to the body of knowledge regarding the connection between MPV and cardiovascular and all-cause mortality, and they may be crucial for managing and treating PD patients.

Peritoneal dialysis patients who have high Mean Platelet Volume have a lower risk of cardiovascular and all-cause mortality.

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Correlation between the atherogenic index of plasma and adverse long-term prognosis among chronic coronary syndrome patients

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death from cardiovascular diseases, accounting for 244.11 million individuals worldwide. It also poses a large public health burden. According to the American College of Cardiovascular Diseases' 2016 data, it is predicted that by the year 2030, about 18% of adults would have chronic coronary syndromes（CCS), raising serious concerns for people's general wellbeing. In order to prevent or manage the condition, it is essential to develop significant biomarkers linked to CCS for early detection, risk assessment, and specific interventions. A recently discovered biomarker known as the Atherogenic Index of Plasma (AIP) that is connected to lipid metabolism has shown to have strong predictive potential in people with cardiovascular disease.[1] The ratio between the amount of triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) is used to construct the atherogenic index of plasma (AIP), which is a measure of the features and severity of abnormal lipid metabolism. As an alternative for small, dense low-density lipoprotein (sdLDL) in determining plasma atherogenicity, it has also been frequently employed.[2] Its effect on chronic coronary syndrome (CCS) have not yet been fully studied. The objective of the

current study was to investigate any potential links between patient AIP levels and long-term clinical outcomes in diagnosed CCS patients.

METHODS

This was a single-center retrospective observational study and included 404 participants with a diagnosis of CCS who had coronary angiography. High-density lipoprotein cholesterol and triglycerides were used to calculate the AIP index. AIP is calculated mathematically from log (TG/HDL-C) and is based on the base 10 logarithms of the ratio of the TG level to HDL-C level in molar concentration (mmol/L). The AIP values of each patient were then used to create major adverse cardiovascular events (MACE) and non-MACE groups as well as quartile groups (Q1: < -0.064; Q2: -0.064–0.130; Q3: 0.130–0.328; Q4: > 0.328). The main clinical outcomes in this study were major adverse cardiovascular events (MACE), which refers to cardiovascular death, ischemia-driven revascularization, nonfatal myocardial infarction (MI), heart failure, and nonfatal stroke. MACE were monitored during each patient's follow-up period. To investigate the connection between AIP and MACE, Cox regression analysis and Kaplan-Meier curve analysis were used. Additionally, the ideal AIP cut-off value for clinical MACE prediction was determined using ROC analysis.

RESULTS

The current study showed that 144 patients (35.6%) out of 404 had diabetic mellitus (DM). A total of 88 patients (21.8%) experienced MACE after a median follow-up of 35 months, including 7 (1.7%) cardiovascular deaths, 29 (7.2%) Ischemia-driven revascularizations, 5 (1.2%) nonfatal myocardial infarction (MI), 27 (6.7%) heart failure, and 20 (5.0%) nonfatal strokes. Patients in the Q4 group who had higher AIP levels also tended to be more hyperlipidemic, diabetic, and had higher BMIs. The Q4 group's blood parameters, such as TG, total cholesterol (TC), remnant-C, non-HDL, haemoglobin A1c (Hb1AC), haemoglobin (Hb), alanine aminotransferase (ALT), and fasting blood glucose (FBG), were considerably greater than those of the Q1, Q2, and Q3 groups, but HDL-C decreased as AIP values increased. Notably, patients in the Q4 group had a substantially greater incidence of MACE than patients in the Q1, Q2, and Q3 groups who had lower AIP values (31.7% vs. 16.8%, 15.7%, and 23.0%, respectively; P=0.023). According to the Kaplan Meier curves, patients in the Q4 group had a higher probability of developing MACE than those in the other groups (log-rank P=0.014). According to the results of the multivariate Cox regression analysis, people in the Q4 group had a 7.892-fold higher risk of MACE than people in the Q1 group (adjusted HR, 7.892; 95% CI 1.818-34.269; P=0.006). The findings demonstrated a greater correlation between the risk of MACE and AIP in younger, male, hypertensive, high-sensitivity C-reactive protein (hs-CRP<10), and non-diabetic CCS individuals. Additionally, AIP has strong correlations with age ($r = -0.193$; $p = 0.021$), FBG ($r = 0.206$; $p = 0.015$), TC ($r = 0.288$; $p<0.001$), low-density lipoprotein cholesterol (LDL-C) ($r = 0.220$; $p = 0.009$), HDL-C ($r = -0.640$; $p<0.001$), and TG in CCS patients with DM, ($r=0.948$; $p<0.001$). The ROC curve study also identified an ideal AIP cut-off value of 0.24 for clinical MACE prediction in CCS patients (Figure 1).

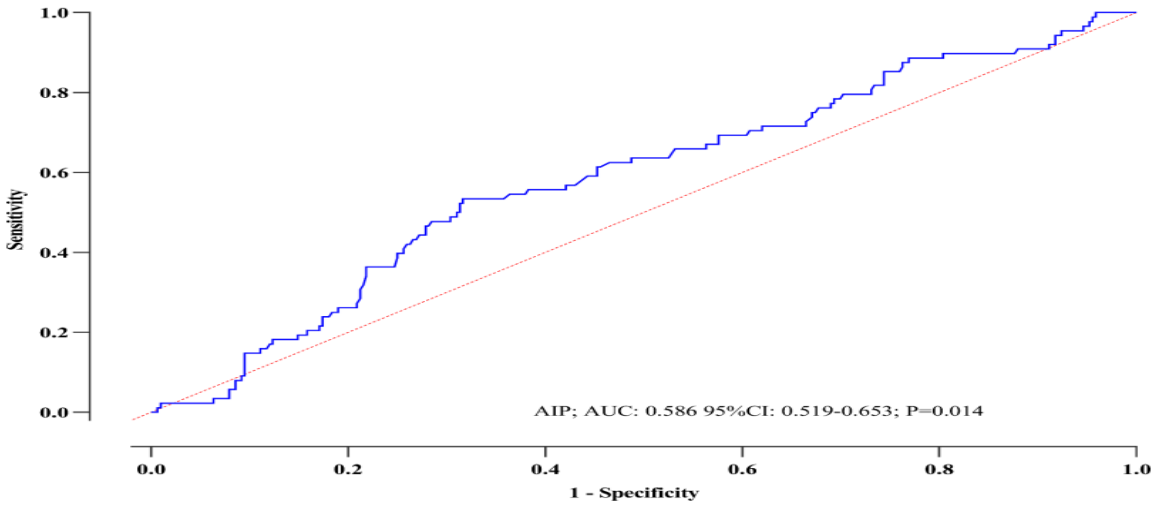

Figure 1. Analysis of the receiver operating characteristics for the AIP's capability of predicting MACE in the study population. **AUC** area under the curve, **AIP** atherogenic index of plasma, **CI** confidence interval

DISCUSSION

The current study showed that in individuals with CCS, elevated AIP level was observed to be independently related with a higher risk of MACE.[1] Similar to this, Hang F et.al showed that among elderly non-diabetic hypertensive patients, an independent and favorable correlation between high AIP and risk of MACEs.[2] The prognosis of diabetic individuals with high AIP levels included higher MACEs and was unaffected by LDL-C levels.[3] For CCS patients, AIP has become a substantial independent risk indicator for predicting the likelihood of MACE.[1] Among older persons with non-diabetic hypertension, the value of AIP was a reliable indicator of cardiovascular prognosis.[2]

CONCLUSION

This study concludes that increased AIP is closely associated with higher risk in CCS patients. The ideal cut-off value for AIP, an independent predictor of MACE in CCS, is 0.24. This study data also demonstrated the critical therapeutic importance of AIP in the context of CCS early risk prediction and preventive interventions.

Increased atherogenic index of plasma (AIP) is closely associated with higher risk in chronic coronary syndromes (CCS) patients.

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Case report on Erdheim-Chester Disease as a Massive Pericardial Effusion identified by Bone Marrow Biopsy

INTRODUCTION

Erdheim-Chester disease (ECD) is a non-Langerhans cell histiocytosis characterized by organs and tissues invaded by foam cells, resulting in lipoid granulomatosis and fibrosis. Since it was originally recorded in 1930, the prevalence of ECD has been very low.[1] It affects several organs, including the brain, skin, kidneys, heart, lungs, and bones. The clinical signs of ECD include arthralgia, keloid development, and systemic symptoms.[2] Similar to other common conditions, cardiovascular involvement is frequently asymptomatic and hence not always clinically obvious.[3] This case report describes an ECD case that was diagnosed by bone biopsy and manifested as a pericardial effusion.

CASE PRESENTATION

A 66-year-old female had complaints of 2 months of dyspnea and leg edema, as well as a 1-week exacerbation of paroxysmal nocturnal dyspnea. Her medical history included hypertension, for which she had been taking medication on a daily basis. One week ago, the patient was diagnosed with heart failure. Both a chest radiograph and a transthoracic echocardiogram revealed substantial pericardial effusion, and an abdomen ultrasound revealed hydronephrosis on both sides. Her vital signs at the time of admission were as follows: blood pressure 193/98 mmHg, pulse rate 88 beats/min, respiration rate 20 breaths/min, and body temperature 36.3°C. No arrhythmia or hypo-phonetic noises. Her chest exam indicated just minor wet crackles at both bases. Type II respiratory failure was revealed by blood gas analysis (O_2 partial pressure of 69.2 mmHg and CO_2 partial pressure of 69.1 mmHg upon oxygen inhalation). The autoantibody screen was negative for rheumatoid factor, antinuclear antibody,

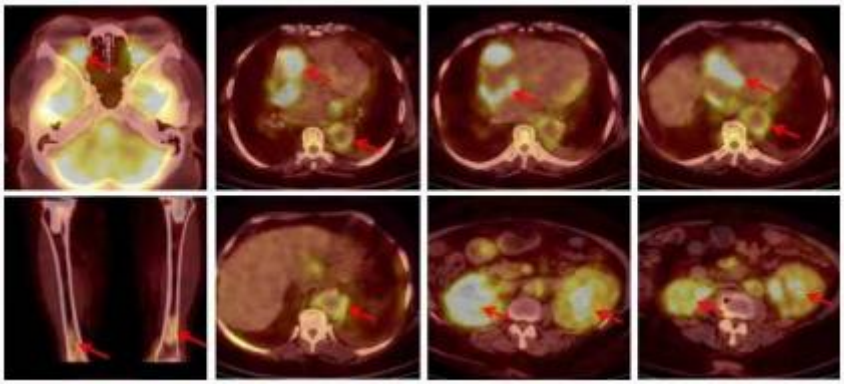

Figure 1-A kidney biopsy was done, and the pathology report revealed an inflammatory change.

anti-dsDNA, anti-Sm, anti-SSA (Ro), anti-RNP, and anti-neutrophil cytoplasmic antibodies, in addition to markers of rheumatic immune system illnesses. They had considered TB, but in the end they ruled it out. CT angiography of the urinary tract (CTU) revealed bilateral renal alterations that seemed to be inflammatory, although it did not rule out a tumour. Following a kidney biopsy, an inflammatory change was seen in the pathology data (Figure 1). Further testing using whole-body Positron Emission Tomography (PET-CT) showed that fluorodeoxyglucose was most absorbed in a number of organs, including all levels of blood vessels, optic organs, kidney, bones, and nerves. Increased uptake in multifocal bone lesions, particularly in both distal femurs, was seen in the whole-body bone scan. Identification of involvement of aortic and cardiac lesions was made. A systemic illness was suggested by the findings. Erdheim-Chester illness was highly suspected following a multidisciplinary course of therapy. Histiocytosis is the defining pathologic hallmark of ECD. They conducted an additional bone marrow biopsy because of the limitations of the cardiac biopsy. A diffuse infiltration of foamy non-Langerhans histiocytes encircled by fibrosis was seen in the bone marrow sample. ECD-compatible immunohistochemical staining with CD68 produced positive findings and negative results for CD1a and S100. Even though a kidney biopsy revealed negative pathologic results, the ECD was ultimately verified by a bone marrow biopsy.

DISCUSSION

The current study showed that a moderate-to-large volume pericardial effusion was seen on the echocardiography in a 66 years old female.[1] Similar to this, Yoon M et al. revealed that a significant amount of pericardial effusion and echogenic mass on the right atrial (RA) side and atrioventricular (AV) groove was observed during echocardiography.[2]

CONCLUSION

This study concludes that Cardiovascular involvement is common in ECD, a rare histiocytic tumour that carries a dismal prognosis. In this instance, they describe an ECD case that primarily manifested as a pericardial effusion and was identified by a combination of pathologic, radiologic, and clinical features. Diagnosing and treating ECD remains significant challenges.

Cardiovascular involvement is common in Erdheim-Chester disease. Diagnosing and treating Erdheim-Chester disease remains significant challenges.

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