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PHYSICIAN CONNECT

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Evaluation of the relationship between C-Reactive Protein to Albumin Ratios and the Risk of Mortality in Patients with Chronic Obstructive Pulmonary Disease

Introduction:

Chronic obstructive pulmonary disease (COPD) is a serious lung disease characterised by reversible and partial airflow limitation that affects over 250 million individuals globally.[1] The Global Initiative for Chronic Lung Disease (GOLD) describes COPD as "a common, treatable, and preventable disease that is characterised by persistent respiratory systems and airflow limitation that is caused by abnormalities of the airways and/or alveoli that are usually caused by significant exposure to noxious particles or gases."[2] Age, ethnicity, gender, cigarette smoking, body mass index (BMI), physical activity, air pollution, arterial carbon dioxide tension (PaCO2), and hospitalisation features are factors that are independently linked to an increase in COPD mortality. An acute-phase protein produced by the liver called C-reactive protein (CRP) is a sign of inflammation in the body (Fig 1). Previous studies demonstrated that, compared to healthy controls, COPD patients had higher levels of CRP and in COPD patients, elevated baseline CRP levels are substantially linked to increased late death. Patients' prognosis with COPD is influenced by a number of factors, including inflammation and the body's nutritional condition. Albumin (ALB) has been employed as a biochemical indicator of nutritional status. The CRP to ALB ratio (CAR), a new scoring tool, has been used to determine the prognosis of numerous disorders. Among hospitalised COPD patients, a greater CAR was linked to an increased risk of malnourishment.[1] To better understand the association between COPD patients' mortality risk and CAR, more studies are required. So, this study aimed to assess the correlation between CAR and mortality risk in individuals with COPD and to determine the predictive value of CAR for mortality risk in COPD patients.

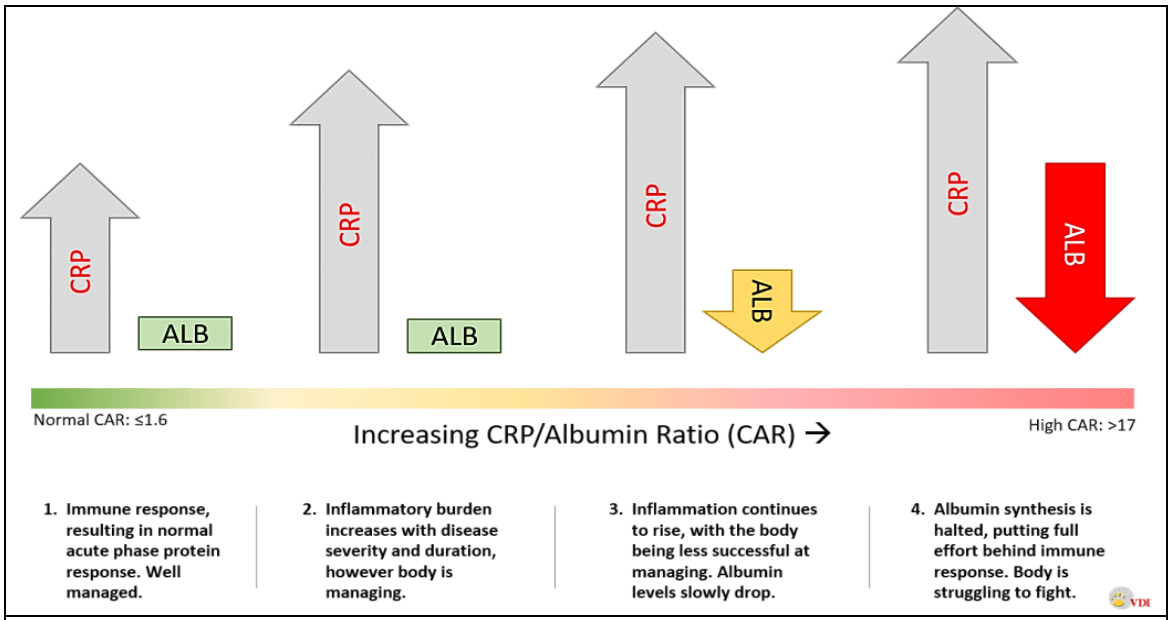

Figure 1. CRP/Albumin Ratio (CAR) – A disease severity index

Methods:

This retrospective cohort study included a total of 1210 COPD patients. The National Institutes of Health's National Health and Nutrition Examination Survey (NHANES) database was used to collect clinical data. Anthropometric, nutritional, laboratory, and questionnaire data were among the information gathered from these samples. The overall 5-year mortality rate for COPD patients was the study's outcome. The relationship between CAR and COPD patients' 5-year mortality was examined using Cox proportional hazard regressions. To determine if there was a consistent relationship between the severity of COPD, gender, body mass index (BMI), smoking status, cardiovascular disease (CVD), chronic kidney disease (CKD), and diabetes, subgroup analyses were used. CAR's predictive ability was assessed using the area under the curve (AUC) of the receiver operator characteristic (ROC) curve analysis.

Results:

Out of 1210 COPD patients enrolled, 110 of them (9.09%) passed away within five years of their diagnosis. The average age of the participants was 54.84 (0.61) years, with 61.03% of the participants being male. The average duration of follow-up was 57.76 (0.33) months. In COPD patients, a greater CAR has been associated to a higher chance of death within 5 years [hazard ratio (HR): 1.94, 95% confidence interval (CI): 1.07 to 3.50, P=0.029)]. According to subgroup analysis, COPD patients with mild COPD, those who were still smoking, those with a BMI of less than 29.9 kg/m^2, those without CVD, diabetes, and CKD were all particularly well-suited for the association between CAR and mortality. In terms of the severity of the COPD, CAR was associated with a higher risk of 5-year death in patients with mild COPD (HR: 1.46, 95% CI: 1.05–2.02, P=0.025); but in patients with moderate-severe COPD, there was no statistically significant correlation between CAR and risk of 5-year mortality. CAR had AUCs of 0.735, 0.615, and 0.608 for predicting 1-year, 3-year, and 5-year death in COPD patients, respectively (Figure 2).

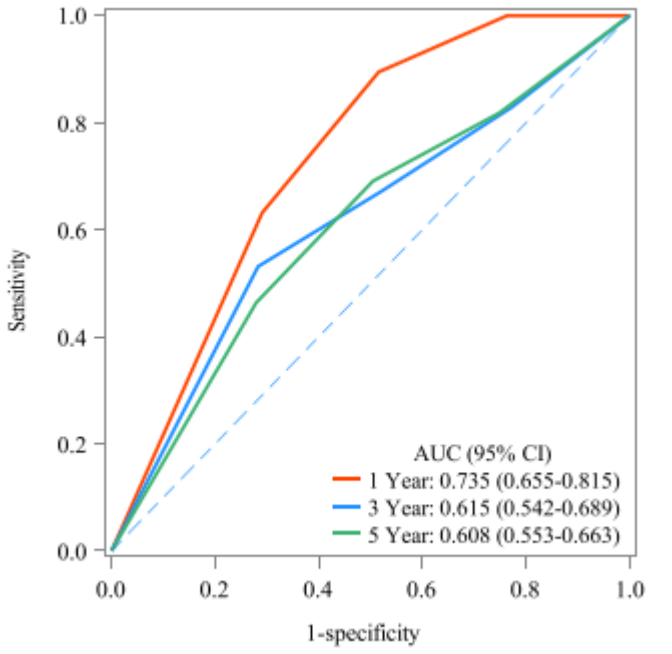

Figure 2. Predictive effectiveness of CAR in COPD mortality prediction.

Discussion:

CAR has been used to predict the prognosis of several illnesses. This study assessed the relationship between COPD patients' risk of death and CAR. It was found that there was a

correlation between a higher CAR and a patient's 5-year mortality in COPD patients. A large number of diseases and their prognoses have been linked to CAR.[1] According to Baldemir et al., individuals with COPD can have their nutritional risk assessed using the cut-off value of 3.26 of CAR.[3] According to the current study, 5-year death rate among COPD patients was shown to be substantially correlated with increased CAR. High CRP and low ALB levels might be the cause of CAR and death in COPD patients.[1] According to a prior study by Leuzzi G, a higher baseline CRP was substantially linked to greater mortality in COPD patients.[4]

Conclusion:

This study concluded that COPD patients' mortality were linked to elevated CAR levels. According to the findings of this study, CAR should be explored further to evaluate its use in predicting prognosis for COPD patients in practice.

The CRP to ALB ratio (CAR) has a significant correlation with mortality in Chronic obstructive pulmonary disease (COPD) patients, and it might be used as a predictive biomarker in COPD patients.

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Assessment of the hemodynamic effects of mechanical thrombectomy in acute PE with right heart overload

Introduction:

Pulmonary embolism (PE) has remained a serious global health concern and is the most prevalent cause of cardiovascular death after myocardial infarction and stroke, and the most significant preventable cause of death among hospitalised patients. Acute pressure overload in the right ventricle (RV) and hemodynamic instability are the strong indicators of poor prognosis in acute PE. As a result, risk assessment is the initial step towards designing PE treatment.[1] Systemic thrombolysis, or reperfusion treatment, appears to be beneficial for patients with high-risk PE. However, due to bleeding problems, systemic thrombolysis is not used enough. Transcatheter thrombus aspiration utilising the FlowTriever System was demonstrated to be well tolerable and feasible in intermediate and high-risk PE in the FlowTriever Pulmonary Embolectomy (FLASH) trial and a large registry. Additionally, right ventricle (RV)/left ventricle (LV) ratio and pulmonary artery pressures (PAP) were quickly lowered during mechanical thrombectomy. However, it is unclear if the reported hemodynamic benefits after thrombectomy are sustained over a longer period of time.[2] The purpose of this study was to evaluate the hemodynamic effects and safety of mechanical thrombectomy in acute PE with right ventricular overload, over a three-month period of follow-up.

Methods:

In this prospective, open-label trial, patients with acute symptomatic, computed tomography-documented PE and symptoms of right ventricular overload received mechanical thrombectomy with the FlowTriever System. Participants in the study included adults (≥18 years old) with symptoms and clinical signs of acute PE (symptom duration<30 days), as well as those with signs of right heart overload in transthoracic echocardiography or computed tomography and documented proximal filling defects in at least one main or lobar pulmonary artery. A life expectancy of less than thirty days (as determined by the investigator) and inability to receive anticoagulation were the exclusion criteria. Right cardiac catheterization was conducted before and after the thrombectomy, as well as three months later. Before thrombectomy, after discharge, and at three months, transthoracic echocardiography was carried out. This study was conducted after 20 patients had completed three months of follow-up. The primary safety outcomes were the number of patients who experienced serious adverse events, such as substantial bleeding and periprocedural device- or procedure-related adverse events, from baseline to 48 hours and survival at 30 days. The efficacy outcomes were alterations in sPAP between baseline and three months and alterations in RV/LV ratio assessed by transthoracic echocardiography from baseline to 48 h.

Results:

The 2019 ESC Acute PE Guidelines state that the majority of patients were at high (17%) or intermediate-high (76%) risk. The PE severity index (PESI) mean value was 121±38. 79% of the patients experienced bilateral PE. Between the initial onset of symptoms and the procedure, the median time was 21 hours (IQR: 4–24). The procedure took 68 minutes on average (IQR: 60–90). About 29 patients (34% female) underwent mechanical thrombectomy, with 20 completing three months of right cardiac catheterization follow-up. Systolic PAP (sPAP) was significantly raised prior to thrombectomy (mean 51.3±11.6 mmHg). Following the surgery, the mean sPAP decreased by -15.0 mmHg (95% confidence interval [CI]: -18.9 to -11.0; p<0.001), and this decline continued for three months following the thrombectomy (-6.4 mmHg, 95% CI: -10.0 to -2.9; p=0.002) (Figure 1). Within two days, there was an immediate reduction of -0.37 (95%

CI: -0.47 to -0.27; p<0.001) in the RV/left ventricular (LV) ratio. Before discharge and after three months, the percentage of patients having a tricuspid annular plane systolic excursion (TAPSE)/sPAP ratio <0.31mm/mmHg dropped from 28% at baseline to 0% (p=0.007). There were no significant adverse effects associated with the procedure.

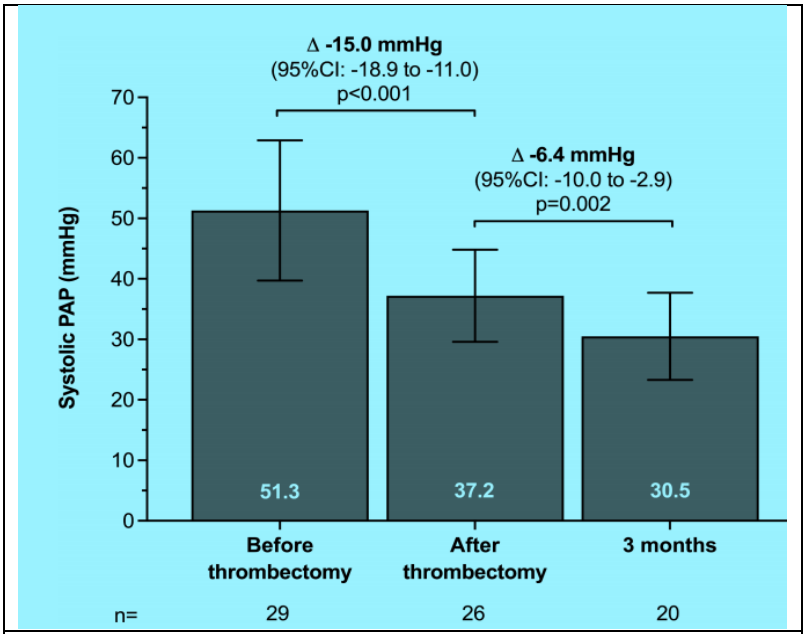

Figure 1. Systolic pulmonary artery pressure was assessed by right heart catheterization prior and after thrombectomy and at three months of follow-up.

Discussion:

In this study, the hemodynamic benefits of mechanical thrombectomy with right ventricular overload in acute PE were evaluated three months after the surgery using the FlowTriever System.[2] Reduction in sPAP and mPAP were also linked to thrombectomy, although the extent of the mPAP changes differed throughout investigations -7.6 mmHg in FLASH study by Toma C et al versus −2.0 mmHg in FLARE study by Tu T et al.[3,4] In current study, mPAP was decreased by -8.4 mmHg.[2]

Conclusion:

Mechanical thrombectomy for acute PE was well tolerable, sudden reduction in PAP and improved right cardiac function. PAP decrease was sustained after three months of follow-up.

For acute Pulmonary embolism (PE), mechanical thrombectomy was a well tolerable procedure that quickly improved right cardiac function and decreased pulmonary artery pressures (PAP).

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Assessment of the correlation between bone metabolic indicators and coronary artery calcification in individuals receiving maintenance haemodialysis

Introduction:

Chronic kidney disease (CKD) is a major health concern globally, which affect 10%-16% of the adult population.[1] The need for renal replacement treatment is rising rapidly as the illness worsens. For patients with end-stage renal disease (ESRD), maintenance haemodialysis (MHD) is a popular and effective renal replacement treatment. Previous studies have found that the incidence of cardiovascular disease in ESRD patients receiving MHD is 20-30 times higher than in the general population.[2] Cardiovascular diseases is one of the most prevalent complications among MHD patients and is linked to an increased risk of mortality. Furthermore, vascular calcification (VC) has been identified as an independent risk factor for cardiovascular disease. Patients with chronic kidney disease (CKD) were more likely to develop osteoporosis and VC, and VC was frequently associated with abnormalities related to bone turnover and reduced bone mineral density. Renal osteopathy and VC are both important aspects of chronic kidney disease mineral and bone disorder (CKD-MBD).[1] This study examined the correlation between serum bone metabolism markers and coronary artery calcification (CAC) in patients with MHD. Additionally, the study aimed to analyse the factors that impact CAC and identify reliable markers that connect vascular calcification and bone metabolism. These findings could contribute to the clinical diagnosis and treatment of CKD-MBD.

Methods:

A total of 123 participants were recruited in the study. Age >18 years, dialysis frequency <3 times/week, duration ≥3 months, no history of peritoneal dialysis, parathyroidectomy, or kidney transplantation, complete case file, and cooperative patient were all the inclusion criteria for this study. Patients with primary parathyroid diseases that affect the metabolism of calcium and phosphorus, rickets, patients with severe malnutrition with albumin levels below 30 g/L, pregnant or lactating women, patients with a history of severe infection or surgery within the previous six months, and use of immunosuppressants and glucocorticoids were all excluded. Multislice spiral computed tomography (MSCT) was used to measure CAC, and the CAC score (CACS) was calculated using the Agaston method. Patients were classified into two groups: calcification and noncalcification. Routine laboratory parameters such as triglycerides (TG), total cholesterol (TC), glucose (Glu), calcium (Ca), phosphorus (P), magnesium (Mg), and others were examined. Serum markers of bone metabolism were also measured, including alkaline phosphatase (ALP), calcitonin (CT), 25-hydroxy vitamin D [25-(OH)D], intact parathyroid hormone (iPTH), total type I procollagen amino-terminal peptide (tPINP), N-terminal mid-fragment of osteocalcin (N-MID OC), and β-type I collagen crosslinked carboxyl-terminal peptide (β-CTX).

Results:

Of that 123 MHD patients, 37 (30.08%) had no CAC and 86 (69.92%) had CAC, with 41 (47.67%) had mild calcification and 45 (52.33%) had moderate to severe calcification. Age, BMI, the prevalence of hypertension and diabetes mellitus, TC, Glu, P, and Ca×P were greater in the calcification group than in the non-calcification group, but Mg, iPTH, tPINP, N-MID OC, and β-CTX were lower in the calcification group ($P<0.05$). In the group with moderate to severe calcification ($CACS\geq400$), P and Ca×P levels were greater than in the mild calcification group ($0<CACS<400$), but the concentrations of ALP, iPTH, N-MID OC, tPINP, and β-CTX were reduced ($P<0.05$). According to correlation analysis, the CACS had a negative correlation ($P<0.05$) with N-MID OC and β-CTX and a positive correlation ($P<0.05$) with TC, LDL-C, P, and Ca×P. There was no significant relationship between the CACS and the other measures($P>0.05$). A logistic regression model was utilised to assess the factors that have an impact on CAC. The findings indicated that, in MHD patients, age, BMI, TC, Glu, P, and Ca×P were risk factors for CAC and its severity, whereas Mg, N-MID OC, and diabetes mellitus were protective factors against CAC. Furthermore, N-MID OC was a protective factor against the severity of CAC. The risk factor results were constant when the associated confounding variables were taken into account, while N-MID OC continued to be an independent protective factor for CAC and its severity (Table 1).

Variables	Univariate analysis					Multivariate analysis				
	B	Wald	P	OR	95%CI of OR	B	Wald	P	OR	95%CI of OR
P	1.058	5.059	0.025	2.881	1.146–7.242	1.518	6.923	0.009	4.563	1.473–14.139
Ca×P	1.579	4.022	0.045	1.035	1.001–1.071	0.051	6.099	0.014	1.053	1.011–1.096
ALP	-0.005	1.802	0.179	0.995	0.988–1.002	-0.002	0.333	0.564	0.998	0.990–1.006
iPTH	-0.001	2.887	0.089	0.999	0.998-1.000	0.000	0.249	0.618	1.000	0.999–1.001
tPINP	-0.002	4.527	0.033	0.998	0.997-1.000	-0.001	0.765	0.82	0.999	0.998–1.001
N-MID OC	-0.012	9.478	0.002	0.988	0.981–0.996	-0.012	6.577	0.010	0.988	0.979–0.997
β-CTX	0.000	5.617	0.018	1.000	0.999-1.000	0.000	1.169	0.280	1.000	0.999-1.000

Abbreviation: P phosphate, Ca×P the product of calcium and phosphorus, ALP alkaline phosphatase, iPTH intact parathyroid hormone, tPINP total type I procollagen amino-terminal peptide, N-MID OC N-terminal mid-fragment of osteocalcin, β-CTX β-Type I collagen crosslinked carboxyl-terminal peptide

Table 1. Univariate and multivariate analysis of influencing factors for the severity of CAC in MHD patients

Discussion:

CKD-MBD is one of the most prevalent problems in CKD patients, particularly in MHD patients, and VC is an important component of CKD-MBD, which is directly associated to the incidence and mortality of cardiovascular illnesses.[1] MSCT was employed in the current study to evaluate CAC. According to the findings, the rate of CAC in MHD patients was 69.92%.[1] This result was in consistent with the China Dialysis Calcification Study (CDCS), which reported the 72.1%.[3]

Conclusion:

In conclusion, this study found a significant incidence of CAC in MHD patients. Bone metabolism indicators and aberrant bone metabolism are closely linked to CAC. The onset and progression of CAC may be promoted by comparatively poor bone turnover. Serum P and CaP levels were independent risk factors for CAC in MHD patients, whereas serum Mg levels may be an independent protective factor. In individuals with MHD, N-MID OC may be a reliable bone metabolic marker that connects vascular calcification and bone metabolism.

Serum phosphorus (P) and the product of calcium and phosphorus (CaP) levels were independent risk factors for coronary artery calcification (CAC) in maintenance haemodialysis (MHD) patients, but serum Mg levels may be an independent protective factor.

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Case report on sarcoidosis-related head and neck tuberculosis

Introduction:

Sarcoidosis is a chronic inflammatory disease with unknown aetiology that is characterised by the formation of non-caseating granulomata in one or more organs. Extrathoracic symptoms of the disease can involve otorhinolaryngological issues, with a preference for upper and lower airway involvement.[1] Tuberculosis (TB) is caused by the bacteria Mycobacterium tuberculosis or bovis. The pathologic characteristic of TB is caseous necrosis. The natural history of TB has two stages: active and latent. When M. tuberculosis or bovis, persists in the body without producing obvious symptoms, it is known as latent TB. Both sarcoidosis and tuberculosis are long-term granulomatous diseases that share similar histological and clinical characteristics. In the absence of microbiological confirmation, it may be challenging to differentiate between tuberculosis and sarcoidosis granulomatosis. The correlation between TB and sarcoidosis may be complicated and unknown.[2] This case report aimed to further examine the ongoing discussion and provide a critical assessment of the connection between sarcoidosis and TB.

Case presentation:

A 42-year-old female with no medical history presented with the major complaint of bilateral neck masses three months ago. Upon physical examination, the left posterior nodes and bilateral jugular nodes were found to be 1 cm in size, firm, non-tender, and movable. The biopsy revealed mucosal hypertrophy at the rhinopharyngeal posterior wall, although the biopsy results were normal. After 72 hours, the tuberculin skin test revealed no induration. Radiography of the chest was normal. The sputum sample tested negative for mycobacteria. Histopathological analysis of an excision sample revealed necrotizing granulomatous inflammation indicative of TB. The patient received antitubercular medications with good adherence (isoniazid 100 mg, rifampicin 900 mg, ethambutol 1,6g, and pyrazinamide 2 g: for two months followed by eight months of isoniazid and rifampicin bitherapy). Rapid improvement was seen in the patient, with notable reduction of the swollen cervical lymph nodes. She had cervical lymph nodes once again during the tenth month of antituberculosis medication; this time, they were associated with nasal obstruction. Nasal endoscopy revealed mucosal hypertrophy as well as multiple nodules on the nasal septum and inferior turbinate, with a biopsy revealing nasal TB. The combination of ofloxacin 600 mg for

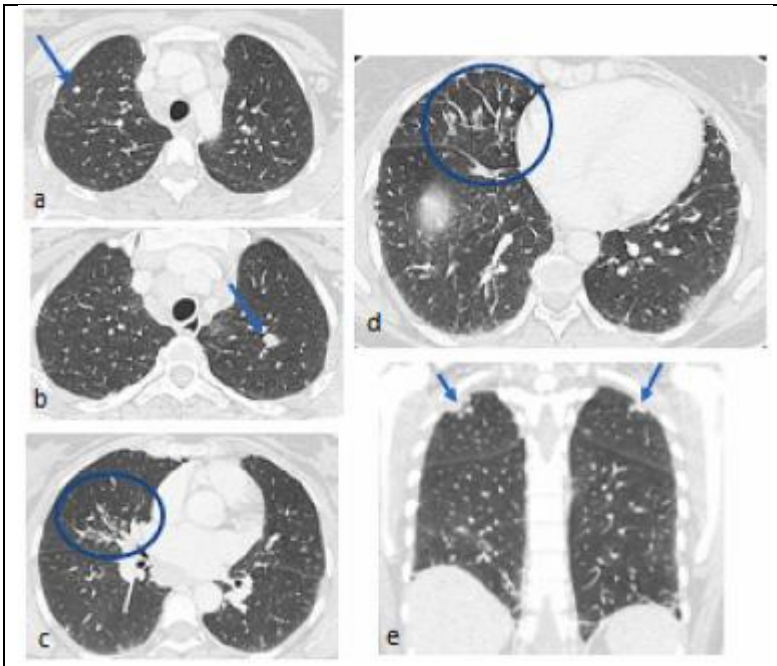

Figure 1. Solid nodules of the left and right upper lobes (Arrows) are seen in the axial CT sections (a, b) and coronal section (e). Peribronchiolar heterogeneous, poorly defined consolidation is seen in the middle lobe of the axial CT section (c, d).

two months and ethambutol 1200 mg for six months was added to the original regimen of isoniazid 100 mg and rifampicin 900 mg. Given that the patient had been experiencing cough and

arthralgia for two months, the negative results of the tuberculin skin test, the later development of nasal TB, and the emergence of new lymphadenopathies after receiving antitubercular medication, it was assumed that the patient may have either HIV infection or sarcoidosis. A CT scan of the thorax and abdomen were performed. It revealed many nodular components with peri-broncho-vascular and subpleural distribution in both lungs (figure 1). A CT scan also revealed enlarged mediastinal lymph nodes (figure 2). The HIV serology of the patient was negative. Following the patient's referral to the interventional pneumology department, bronchoalveolar lavage was performed. The results revealed a lymphocytic alveolitis with a CD4/CD8 ratio of 5, and the lavage fluid did not include any acid fast bacilli. The results of the bronchoalveolar liquid culture were negative. All of these findings indicate the diagnosis of pulmonary sarcoidosis. The pneumologists had a thorough discussion about the introduction of steroid medication, which was not initially indicated. Antitubercular medications were stopped after an 18-month course of treatment due to the cervical adenopathy's gradual recovery and the nasal mucosa biopsy's negative results. Given the worsening of the cough with recent dyspnea, the presence of pulmonary fibrosis on the CT scan, and the findings of the lung function test, corticosteroid medication in the form of oral prednisolone, 0.5 mg/kg daily, was initiated. After two months, the patient's clinical condition had significantly improved. After roughly a year, the prednisolone was progressively reduced, and after three years, when all of the lesions had been cleared up, it was stopped completely. At the time of the most recent follow-up evaluation, four years after she stopped using corticosteroids, her sarcoidosis was still quiescent.

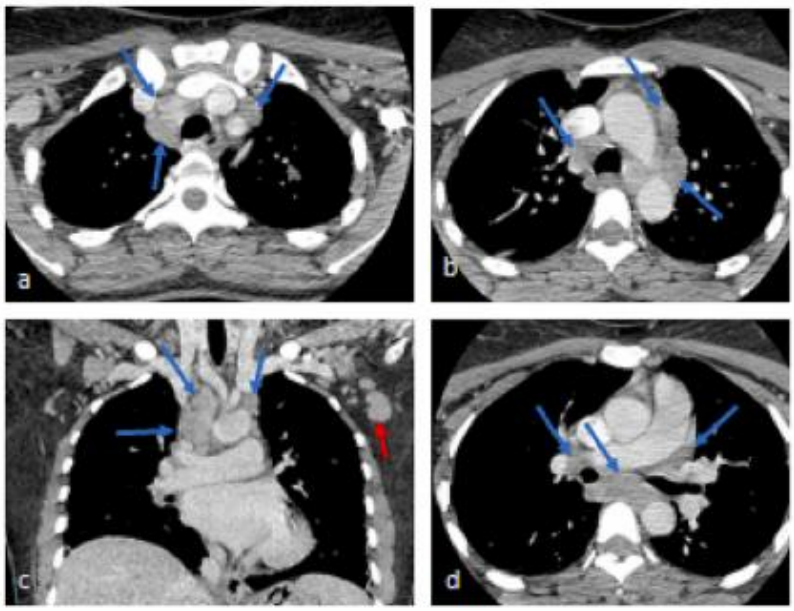

Figure 2. Axial CT sections (a, b, d), as well as the coronal section (c), indicate bilateral hilar lymph nodes and numerous heterogeneous enhancement in the mediastinal region (blue arrows). The left axillary lymph nodes are shown by a red arrow in the coronal panel.

Discussion:

There are sporadic case reports in the literature describing the co-occurrence or sequential incidence of sarcoidosis and TB in the same patient, or vice versa. This case report highlights the need to consider a potential correlation between sarcoidosis and recurrent or resistant TB.

[2] Recently, a bidirectional cohort research was reported in which researchers created two cohorts. 56 TB patients from the first cohort and 8 controls had sarcoidosis at the conclusion of the research. In the second cohort, 74 sarcoidosis patients developed TB compared to 187 controls. These findings support the theory that TB is a risk factor for sarcoidosis. Compared to non-TB participants, patients with TB had an 8.09-fold increased chance of acquiring sarcoidosis, whereas those with sarcoidosis had a 1.85-fold increased risk of developing TB. Furthermore, Patients with extrapulmonary TB had the highest chance of developing sarcoidosis. Patients with sarcoidosis only had an increased chance of getting tuberculosis within the first year of treatment, but sarcoidosis post-tuberculosis was likely to occur gradually, long after the tuberculosis has been cured.[3]

Conclusion:

This case report demonstrated that it was important to consider a potential link to sarcoidosis when recurrent or resistant TB is identified. The diagnosis of sarcoidosis and TB can be difficult since they have similar pathophysiology and clinical manifestations, and they can even coexist in the same patient, particularly in areas where tuberculosis is widespread.

Since sarcoidosis and tuberculosis have a similar aetiology and clinical symptoms, and since they can coexist in the same patient, especially in locations where tuberculosis is prevalent, diagnosing them can be challenging.

References:

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