Background: Takayasu arteritis (TA) is a chronic granulomatous large- vessel vasculitis most commonly seen in women under 50 years of age[1]. The pulmonary arteries are less often involved, with the frequency of involvement varying widely between countries (from 4% to >50%) [2-5]. Respiratory symptoms or signs and pulmonary imaging findings in TA have not been fully investigated [6–7].

Objectives: This study aimed to describe pulmonary high-resolution computed tomography (HRCT) findings in TA and to determine possible causes.

Methods: A total of 243 TA patients were enrolled from a prospective cohort after excluding 260 patients with other pulmonary disorders or incomplete data. Clinical data including symptoms, lab results, imaging information were collected. Pulmonary HRCT were interpreted by two radiologist who were blinded to patients' clinical information. Abnormal pulmonary features were recorded as nodules, stripe opacity (linear opacity), patchy opacity, ground-glass opacity, pleural thickening, pleural effusion, pulmonary infarction, mosaic attenuation, pulmonary bronchiectasis, pulmonary oedema. After evaluation, patients were divided into two groups: those with normal lung HRCT and those with abnormal lung HRCT. Clinical characteristics were compared between groups and binary logistic regression analysis was applied to identify potential risk factors for the lung lesions. Follow-up HRCT (obtained in 64 patients) was analysed to study changes in pulmonary lesions after at least 6 months' treatment.

Results: Of the 243 patients, 107 (44.0%) had normal lung HRCT while 136 (56.0%) had abnormal lung HRCT, including stripe opacity (60.3%), nodules (44.9%), patchy opacity (25.0%), pleural thickening (15.4%), pleural effusion (10.3%), ground-glass opacity (8.1%), pulmonary infarction (6.6%), mosaic attenuation (4.4%), bronchiectasis (3.7%), and pulmonary oedema (2.2%). Patients with abnormal HRCT were significantly more likely to have type II arterial involvement (25% vs. 12.2%, P = 0.04), pulmonary arterial involvement (PAI; 21.3% vs. 5.6%, P < 0.001), pulmonary hypertension (20.6% vs. 8.4%, P = 0.01), and abnormal heart function (27.9% vs. 7.6%, P < 0.001). Logistic regression analysis demonstrated that PAI (OR = 3.0, 95% CI= 1.1-8.4, P=0.03), worsened heart function (OR = 2.7, 95% CI = 1.1-6.5, P=0.03), and age (OR = 1.1, 95% CI = 0.9-1.1, P<0.01) were associated with presence of pulmonary lesions. Pulmonary infarction, pleural effusion, and patchy opacities improved partially after treatment.

Conclusion: Pulmonary lesions are not rare in patients with TA. Age, PAI, and worsened heart function are potential risk factors for presence of pulmonary lesions in TA.

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EULAR RECOMMENDATIONS ON GIANT CELL ARTERITIS IN REAL LIFE

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Background: GCA is the most frequent systemic vasculitis in patients older than 50 years involving medium-sized and large arteries. On July 2019 EULAR published its updated recommendations for the management of large vessel vasculitis, including GCA.

Objectives: To analyze how the application of the updated EULAR recommendations changed clinical practice in GCA patients in our Hospital.

Methods: All patients with a new diagnosis of GCA between January 1st, 2018 and December 31st, 2020 were enrolled in this study. Two cohorts were analyzed: patients who received GCA diagnosis in the eighteen months before EULAR recommendations publication (between January 1st, 2018 and June 30th, 2019; cohort A) and patients who received GCA diagnosis in the following eighteen months (between July 1st, 2019 and December 31st, 2020; cohort B). Data are expressed as median (IQR). No difference in treatment regiments were found between groups, whether in glucocorticoid initial dose or DMARDs adjunctive therapy.

Conclusion: After EULAR recommendations publication, two major improvements were achieved in our cohort. EULAR suggests GCA patients should be urgently referred to a specialist team. Consistently with this recommendation, time between symptoms onset and first rheumatologic evaluation was reduced by 30% (from 61 (23-131) to 43 (22-92) days). No difference in treatment regiments were found between groups, whether in glucocorticoid initial dose or DMARDs adjunctive therapy.

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