improved dramatically by angiotensin-converting enzyme inhibitor therapy, but SRC still has a poor prognosis. Factors predictive of SRC include early diffuse skin involvement, rapid skin thickening, anti-RNA polymerase (RNAP) III antibodies, arthralgia/synovitis, and high glucocorticoid dosage. Although classical data have implicated pericardial effusion as another predictive factor of SRC, its role in SRC has not been well established.

**Objectives:** To clarify the clinical impact of pericardial effusion as a predictor of SRC.

**Methods:** Ninety-five patients diagnosed with SSc at our hospital between January 2003 and December 2017 were enrolled in the study. They were divided into a pericardial effusion group (n=21) and a non-pericardial effusion group (n=74), and their clinical features retrospectively compared. Cox-regression analysis was performed to identify factors predictive of SRC.

**Results:** The patients comprised 14 men and 81 women with an average age of 57.4 years (range, 14 to 82) and the mean observation period was 65 months (range, 1 to 125). Pericardial effusion was detected in 21 of 95 (22.1%) cases. In the pericardial effusion group, SRC, modified Rodnan’s total skin thickness score (mRSS), C-reactive protein, maximum glucocorticoid dose, proteinuria, finger apex ulcers, and interstitial pneumonia were significantly more prevalent compared to the non-pericardial effusion group. Cox regression analysis indicated that pericardial effusion (hazard ratio: HR 11.1 [95% CI 2.0–69.6], p=0.005) was independent risk factor for SRC, while mRSS (HR 1.0 [95% CI 0.9–1.1], p=0.12), finger apex ulcers (HR 0.57 [95% CI 0.073–4.2], p=0.57), max glucocorticoid dose (HR 1.0 [95% CI 0.9–1.0], p=0.89), and interstitial pneumonia (HR 0.9 [95% CI 0.3–3.7], p=0.98) were not. In the Kaplan-Meier method, SRC was significantly increased in the pericardial effusion group compared to non-pericardial effusion group (p=0.0001 by log rank test).

**Conclusions:** Pericardial effusion is another independent factor predictive of SRC in addition to anti-RNAP III antibodies.

**REFERENCES:**


**Disclosure of Interest:** None declared

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**Vasculitides**

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**THE PREVALENCE OF SPONDYLOARTHROPATHY IN PATIENTS WITH TAKAYASU ARTERITIS**

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**Background:** Takayasu arteritis (TA) is characterised by inflammation of large arteries causing stenosis, occlusion, dilatation and/or aneurysm of affected vessels. TA is most commonly seen in younger women between 20–30 ages. Etiopathogenesis of TA is largely unknown although evidence suggest complex interplay between environmental and genetic factors such as HLA (human leukocyte antigen) groups. The coexistence of TA with spondyloarthropathies (SPA) has been reported in limited case series, raising hypotheses about shared pathogenic mechanisms.

**Objectives:** To determine prevalence of spondyloarthropathy in patients with TA.

**Methods:** Detailed clinical and demographic features of TA patients were recorded and all were screened for the presence of SPA following recommendations of (ASAS). Patients were questioned for inflammatory back pain, enthesitis, uveitis, inflammatory bowel disease, peripheral arthritis, and investigated according with HLA-B27; plain X-rays of lumbar, sacroiliac and sacroiliac magnetic resonance imaging. Radiographic spondyloarthrititis was reported in case of bilateral grade ≥2 or unilateral grade ≥3 sacroiliitis.

**Results:** There were 65 patients (61 female, 4 male) in the cohort. Mean age was 43±13 years and age at the diagnosis of TA was 35±13 years. Inflammatory bowel disease, psoriasis and psoriatic arthritis were observed in four, three and one patients. Chronic axial pain was reported by 26 (40%) patients but inflammatory back pain was evident in 13 (20%) patients. Chronic arthritis was observed in 4 patients. HLA-B27 was positive in three patients. Six patients were diagnosed as radiographic SPA and 3 were diagnosed as non-radiographic SPA. In sum nine patients were diagnosed as SPA (14%).

**Conclusions:** Our study demonstrated that SPA is common in patients with Takayasu arteritis suggesting shared pathogenic mechanisms.

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