



The influence of obesity and adipose tissue mediators on the course of sarcoidosis

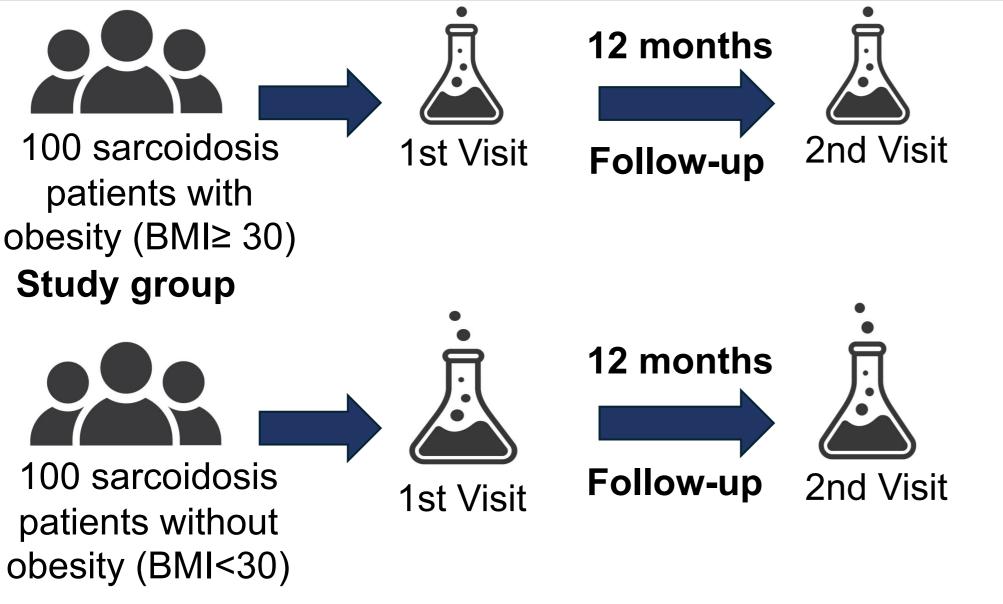
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Introduction

- Sarcoidosis is a systemic disease defined by non-caseating granulomas in various organs, with progression mainly dependent on lung manifestations and extrapulmonary features
- o Various cytokines such as TNF-alpha, interferon-gamma, and IL-17 play a crucial role in the inflammation process and persistence of sarcoidosis¹.
- Adipokines (released by adipose cells) play an important role in the pathogenesis of many inflammatory diseases, such as asthma², connective tissue diseases³, and tuberculosis⁴ by angiogenesis, lipid metabolism, and immune system regulation, which may affect the mechanism of sarcoidosis
- The relationship between obesity and sarcoidosis has yet to be thoroughly investigated. Furthermore, the interactions between the adipokines with vitamin D metabolism and calcium homeostasis—both of which may be disrupted in sarcoidosis—remain unclear.

The aim of the study is to evaluate the influence of obesity and alterations in body composition and concentrations of selected adipokines on the course of sarcoidosis and calcium metabolism

Methods



The correlation between adipokine concentrations

and other parameters with clinical severity criteria

and BMI will be evaluated

Inclusion criteria

- Diagnosis of sarcoidosis according to ATS criteria
- o BMI>=30
- Written consent
- o Age 18-80

Exclusion criteria

- Other chronic respiratory diseases
- Respiratory tract infection within the last 4 weeks
- Use of immunomodulatory drugs (systemic steroid therapy, immunosuppression such as methotrexate, azathioprine, hydrochlorquine, infliximab, adalimumab)
- Chronic diseases that, in the researchers' opinion, could affect the inflammatory parameters being studied (endocrinological diseases, diabetes)

Outcomes

- Disease progression:
 - new symptoms or exacerbation of symptoms
 - ≥ 10% decline in FVC, ≥ decline in TLCO
 - >50m distance reduction in 6-MWT
 - appearance/worsening of extrapulmonary manifestations
 - initiation of pharmacotherapy
 - treatment intensification
- Remission status

Results

- o Obtained approval from the Bioethics Committee at the Medical University of Lodz, number RNN/249/22/KE
- 15 patients recruited from Study group

Control group

- 10 patients recruited from Control group
- Each patient had the following tests performed: Chest HRCT, Pulmonary function tests (spirometry, TLCO, 6-MWT), ECG, Blood sampling (total calcium, 25(OH)vitamin D, 1,25(OH)2vitamin D, creatinine, alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, C-reactive protein CRP, complete blood count), systemic inflammation markers (IL-6, TNF-alpha), sarcoidosis activity markers (ACE, SIL2R, neopterin, YKL-60), BMI, waist circumference, body composition analysis by electrical bioimpedance, FAS scale, SHQ questionnaire.
- Each patient had the blood sample secured for adipokines measurement biobank of the Laboratory of
 Patobiology of Respiratory System at the Pneumonology Department adiponectin, leptin, resistin, chemerin, wisfatin, progranulin, FSTL-1

References

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- 3 Żółkiewicz J, Stochmal A, Rudnicka L. The role of adipokines in systemic sclerosis: a missing link? Arch Dermatol Res. 2019 May;311(4):251-263. doi: 10.1007/s00403-019-01893-1. Epub 2019 Feb 26. PMID: 30806766.
- 4 Zheng Y, Ma A, Wang Q, Han X, Cai J, Schouten EG, Kok FJ, Li Y. Relation of leptin, ghrelin and inflammatory cytokines with body mass index in pulmonary tuberculosis patients with and without type 2 diabetes mellitus. PLoS One. 2013 Nov 8;8(11):e80122 doi: 10.1371/journal.pone.0080122. PMID: 24260344.