

premature CHD. Classical risk factors are observed more frequently in CHD patients as expected but interestingly, no additional risk factors other than age were detected between premature and other CHD patients. This suggests that the primary factor triggers premature CHD is the underlying inflammatory rheumatic disease. The presence of CHD was determined by patient history which the limitation of our study. We did not assess to subclinical CHD in this study.

References:

[1] Comorbidity in rheumatoid arthritis, Carl Turesson, Swiss Medical Weekly, 2016.

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THU0132 IMMUNOGENICITY AND SAFETY OF 23-VALENT PNEUMOCOCCAL VACCINE FOR PATIENTS WITH RHEUMATOID ARTHRITIS: RESULTS FROM 2-YEAR FOLLOW UP

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Background: Comorbid infections have significant impact on morbidity and mortality, especially in autoimmune diseases. Prevention of infection is an integral part of supervision of these patients.

Objectives: To investigate immunogenicity and safety of 23-valent polysaccharide pneumococcal vaccine in patients with rheumatoid arthritis (RA) treated with diseases modifying anti rheumatic drugs (DMARDs) and biologic diseases modifying anti rheumatic drugs (bDMARDs) during the 2-year follow-up.

Methods: Out of 110 subjects (81 females (73,6%), 29 males (26,4%) aged 23–76 years) included into the study, 79 were RA patients and 31 were controls with a history of ≥ 2 episodes of lower respiratory tract infections (bronchitis, pneumonia). 52 patients with RA were on methotrexate (MTX), 14 were on leflunomide (LEF), 13 were on tumor necrosis factor alpha inhibitors (iTNF- α)+MTX. One dose (0,5 ml) of 23-valent polysaccharide pneumococcal vaccine was administered subcutaneously without discontinuation of MTX/LEF or 28–30 days prior to initiation of iTNF- α . Totally four study visits were preplanned: initial vaccination visit and 3 control visits in 1, 3 and 12 months after vaccination for 110 patients. 39 RA patients were followed up for 2 years (24 months). Routine evaluation during each visit included physical exams and laboratory tests. Levels of antibodies to pneumococcal capsular polysaccharide were measured using VacciZyme™ PCPIgG 2 kit (The Binding Site Group Ltd, Birmingham, UK). Post-immunization response coefficient was calculated for each participant as the ratio of AB levels during visits II, III, IV and V to baseline AB level at Visit I.

Results: Not a single case of clinically or radiographically confirmed pneumonia was documented during the follow up period. Pronounced positive immune reaction after administration of the vaccine under investigation was documented in RA patients during different therapies, i.e., significant post-immunization response coefficient increase. There were 61% responders among RA patients and 70% responders among the controls during one-year follow-up. Dynamics of post-immunization response coefficient in RA patients during 2-year follow up are presented in the Table. RA patients and the control group are marked more than 2-fold significant increase in the content of antibodies in 3 months after the vaccination. Despite the decline in their concentrations to 12 months, it remained at the proper level and was increased to 24 month follow-up. Good tolerability of the vaccine was documented in 65% of cases, satisfactory (injection site pain, swelling and hyperemia of the skin up to 2 cm in diameter and subfebrile fever) in 35% of cases. As these reactions had no causal relationship with current RA therapy, and fully resolved within 24 hours without additional treatment, no RA therapy modification was required. Pronounced DAS28 positive dynamics in RA patients (4,27 and 2,68 at Visit I and Visit V, respectively, $p < 0,001$) indicates the absence of any negative impact of vaccination on disease activity.

Table. Post-immunization response coefficient dynamics in RA patients during 2 year follow up. Me [25,75 percentile]. (n=39).

	Visit II (1 month)	Visit III (3 Month)	Visit IV (12 month)	Visit V (24 month)
RA patients	2,33* [1,6;3,8]	3,55* [2,32;6,23]	2,64* [2,03;6,39]	3,07* [1,56;6,18]

*p < 0,05

Conclusions: Thus, all given prove the sufficient immunogenicity and safety of 23-valent pneumococcal vaccine in RA patients, getting different therapeutic regimens during the 2-year follow-up.

Disclosure of Interest: None declared

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THU0133 SERUM PENTRAXIN-3 IN THE ASSESSMENT OF CARDIOVASCULAR RISK IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is associated with the increased cardiovascular (CV) morbidity and mortality due to accelerating, progressive atherosclerosis. The chronic, systemic inflammatory process is responsible for both joint damage and increased CV risk in RA patients. Pentraxin 3 (PTX3) is an inflammatory marker, a member of long pentraxin superfamily, supposed to be involved in inflammatory process as well as in atherosclerosis.

Objectives: The goal of the study was to assess the role of PTX3 as an inflammatory marker in patients with RA and to evaluate the relationship between PTX3 and CV risk markers [carotid intima-media thickness (cIMT), QTc distance (dQTc), lipid profile].

Methods: The study group consisted of 72 consecutive RA patients, 60 (83,3%) female and 12 male (16,7%), with the mean (SD) age 53,4 (10,29) (range 21–71) and disease duration 16,8 (10,3) years (range 2–49). The activity of RA was estimated by clinical examination with the disease activity score in 28 joints (DAS28). Remission or low disease activity was observed in 35 (48,6%) patients; moderate or high disease activity (DAS28 >3,2) in 37 (51,4%) patients. Disease modifying antirheumatic drugs (DMARDs) used in the treatment included: methotrexate 61 (84,7%) patients, chloroquine or hydroxychloroquine 9 (12,5%), leflunomide 4 (5,6%), cyclosporine 1 (1,4%) patient. The majority of patients 54 (75%) were treated with biological DMARDs, currently or in the past.

Results: The mean (SD) PTX3 concentration in RA patients was 4,57 (2,83) ng/ml (range 1,43–16,07). The mean (SD) cIMT value was 0,86 (0,2) mm (range 0,43–1,77). There were 19 (26,4%) RA patients with advanced atherosclerosis (presence of atherosclerotic plaques).

The positive, significant correlations were found between PTX3 concentration and other inflammatory markers: C-reactive protein (CRP) (R=0,5), ESR (R=0,46) and white blood cell count (WBC) (R=0,41). PTX3 concentration was also correlated with clinical disease activity markers: DAS28 value (R=0,41), as well as with tender joint count (TJC) (R=0,01), swollen joint count (SJC) (R=0,009), patient's global assessment of the disease activity (R=0,02).

The mean (SD) PTX3 concentration was significantly higher in patients with moderate/high RA activity in comparison with remission/low disease activity [5,56 (3,29) vs 3,48 (1,71) ng/ml, $p=0,001$] and in patients anti-CCP positive compared with anti-CCP negative [4,57 (2,58) vs 3,02 (0,85) ng/ml, $p=0,04$].

The mean (SD) PTX3 concentration was significantly higher in patients with definite atherosclerosis (cIMT >0,9 mm) than in patients with subclinical or no atherosclerosis [5,77 (3,02) vs 3,99 (2,58) ng/ml, $p=0,04$], as well as in patients with atherosclerotic plaques in comparison with no plaques (6,18 (2,83) vs 4,02 (2,64) ng/ml, $p=0,0006$).

There was a negative correlation between PTX3 and dQTc (R= -0,33, $p=0,007$).

Conclusions: The results of the study suggest a twofold role of PTX3:

1. an inflammatory marker of the joint disease activity
2. a biomarker indicating intensity of atherosclerosis, estimated by greater cIMT value and the presence of atherosclerotic plaques. The negative correlation between PTX3 and dQTc suggests the increased risk of sudden cardiac death due to shortening of dQTc.

Disclosure of Interest: None declared

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THU0134 INTERSTITIAL LUNG DISEASE AND RHEUMATOID ARTHRITIS. MULTICENTER STUDY WITH TOCILIZUMAB

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Background: Interstitial Lung Disease (ILD) is a severe extraarticular manifestation of rheumatoid arthritis (RA). Anti-TNF α drugs and conventional disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate (MTX) have been involved in the development of ILD. IL6 has been implicated in the pathogenesis of ILD (Kobayashi J et al). However, a fatal case of exacerbation of ILD has been described with tocilizumab (TCZ) (Kawashiri SY et al).

Objectives: Our aim was to assess the efficacy and safety of TCZ in ILD associated with AR.

Methods: Multicenter study of RA patients with ILD treated with TCZ. ILD was diagnosed by high-resolution computed tomography (HRCT). TCZ was used at standard dose (8 mg/kg/iv/4 weeks). We have analyzed the following variables: a) 1-point change in the degree of dyspnea according to the Modified Medical Research Council (MMRC); b) Forced Vital Capacity (FVC) improvement $\geq 10\%$; and improvement $\geq 10\%$ in DLCO; c) HRCT, and d) joint assessment (DAS28 score).

Results: We studied 12 patients (9 women/3 men) with ILD related to RA.