INDIVIDUALS WITH ACPA POSITIVE RA PRESENTING INITIALLY WITH PALINDROMIC SYMPTOMS ARE SIGNIFICANTLY LESS LIKELY TO BE RHEUMATOID FACTOR POSITIVE THAN CLASSICAL ACPA POSITIVE RA; COULD THE AETIOPATHOGENESIS DIFFER BETWEEN THESE TWO DISTINCT TYPES OF ACPA POSITIVE RA?

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Background: It remains unclear whether ACPA positive RA presenting initially with palindromic symptoms (PRA) has the same underlying pathophysiology as ACPA positive RA presenting without palindromic symptoms (classical ACPA positive RA). The lung is an initiating site in RA development and rheumatoid factor (RF) is generated within bronchial associated lung tissue (1). RF in combination with ACPA immune complexes can induce TNFα from joint macrophages and potentially induce synovitis (2). Unsurprisingly a positive RF is a risk factor for RA development. Alternatively, palindromic rheumatism in the absence of RA occurs commonly in RF negative individuals (3) (4). Therefore this form of explosive synovitis often occurs independently of RF, but is commonly associated with ACPA positivity (5-6) (3).

Objectives: To identify whether classical ACPA positive RA differs in terms of RF positivity compared to ACPA positive PRA.

Methods: A retrospective case note observational study was undertaken to identify ACPA positive RA patients with or without a palindromic onset. All patients fulfilled the RA 2010 EULAR/ACR classification criteria (4). Patients were stratified for a history of smoking. RF was measured with Tina-quant Rheumatoid Factors II Test System, by Roche Diagnostics Corporation at the time of diagnosis. A value of <14 IU/ml was considered as negative, weakly positive (14-42 IU/ml) and strongly positive (>42.1 IU/ml) as per EULAR/ACR guidelines (4).

Results: 99 classical ACPA positive RA and 35 ACPA positive PRA were identified. The median level of RF for PRA was significantly lower than the non-PR onset RA group (25 vs 86 IU/ml, p < 0.001). Comparing classical ACPA positive RA with ACPA positive PRA revealed 0/99 (0%) vs. 8/35 (23%) to be RF positive, 23/99 (23%) vs. 11/35 (31%) to be RF weakly positive and 68/99 (69%) vs. 18/35 (46%) to be strongly RF positive. Classical RA patients were significantly less likely to be RF negative, odds ratio 0.25 (95% CI 0.09-0.73), p < 0.001. RA patients were significantly more likely to be strongly RF positive OR 4.32 (1.88-9.98), p = 0.0005. Classical ACPA RA patients were more likely to be smokers than ACPA PRA patients 60/99 (61%) vs. 18/35 (51%), however this was not significant OR 1.48 (95% CI 0.88-2.32) p = 0.15. Classical ACPA RA patients accumulated a greater number of pack years, median of 17 vs 5, however, the difference was not significant P = 0.12.

Conclusion: The majority of ACPA RA patients presenting with palindromic symptoms have either a negative or weakly positive RF (54%). This contrasts with classical ACPA RA (31%) and suggests that a strongly positive RF is more important in the pathogenesis of classical ACPA RA than ACPA PRA. We suggest that a strongly positive RF and palindromic rheumatism are likely to be independent risk factors for ACPA positive RA.

REFERENCES

[1] Pecani A et al, Reumatismo 2017

Disclosure of Interests: None declared

REFERENCES