



## Erratum

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Niels Lomborg, Department of Rheumatology, Odense University Hospital, 5000 Odense C, Denmark.

E-mail: niels.lomborg@rsyd.dk

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## Assessment of arterial stiffness in patients with inactive and active Behçet's disease: comments on the article by Yilmaz et al

We read with great interest the article by Yilmaz et al on 'Assessment of arterial stiffness in patients with inactive and active Behçet's disease' (1). The authors investigated the relationship between central blood pressures, pulse wave velocity (PWV) measurements, and biochemical parameters in patients with inactive and active Behçet's disease (BD) and control subjects. They concluded that PWV values were higher in patients with active BD than in patients with inactive BD. We believe that these findings will be guides for further studies about the effect of inflammation on arterial stiffness parameters in patients with BD.

BD is a chronic, multisystemic, inflammatory process with clinical features of mucocutaneous lesions, and ocular, vascular, articular, gastrointestinal, neurological, urogenital, pulmonary, and cardiac involvement (2). Measuring disease activity in BD is difficult because the disease presents with a fluctuating course, and none of the available laboratory tests reflect overall disease activity. However, numerous BD study groups worldwide have attempted to develop a standardized disease activity index. The BD Current Activity Form (BDCAF) presents an easy, practical, and reliable method of assessing disease activity in patients with BD in the Turkish population (3). In this context, the results presented by Yilmaz et al may have been more useful had they mentioned this disease activity index.

This multisystemic disorder primarily affects the vascular system. BD is commonly related to morbidity and mortality accompanied by the vascular system presenting with vasculitis, thromboembolism, and pulmonary artery aneurysm. In addition, male gender, a younger age of onset, and human leucocyte antigen (HLA)-B51 positivity in BD are associated with vascular involvement and may predict morbidity and mortality in BD (4). All of these factors may be associated with arterial stiffness parameters, and it might have better if Yilmaz et al had described these factors.

Arterial stiffness indicates the viscoelastic properties of the vessel wall. It represents vascular damage and is a measure of the degree of atherosclerosis. Increased arterial stiffness is a common indicator of atherosclerotic involvement of the vascular structure indicating coronary artery disease,

cerebrovascular disease, and peripheral arterial disease. BD is associated with endo-thelial dysfunction and chronic inflammation. Endo-thelial dysfunction is an important early step in the process of atherogenesis, and is commonly investigated by measuring arterial stiffness. We previously investigated the relationship between increased arterial stiffness in patients with BD without cardiovascular involvement and known cardiovascular risk factors. We concluded that arterial stiffness parameters were associated with BD without significant cardiovascular involvement (5). We also reported that arterial stiffness was significantly higher in psoriasis patients compared to controls. In addition, we concluded that, in patients with psoriasis, arterial stiffness correlated positively with age, sex, body mass index, diastolic blood pressure, and high-sensitivity C-reactive protein (hsCRP) level (6). Thus, it would have been better if the Yilmaz et al had mentioned some inflammatory diseases such as psoriasis that can be related to the arterial stiffness parameters.

Hypothyroidism plays a key role in cardiovascular disease pathogenesis by increasing total peripheral vascular resistance and inflammatory condition (7). Previous work by Obuobie et al showed increases in augmentation indexes and central aortic pressures of hypothyroid patients (8). Central aortic pressure had statistically significant relationships with serum calcium levels and 24-h urinary microalbumin excretion rates in patients. When arterial stiffness parameters were investigated with respect to their relationships with other studied parameters in patients, central aortic pressure showed significant relationships with serum vitamin B12 and phosphorus levels while augmentation indexes had significant correlations with albumin and magnesium levels (9).

Given that arterial stiffness is a non-invasive method for evaluating endothelial dysfunction in clinical practice and that, without other inflammatory markers, arterial stiffness alone may not provide information to clinicians about the activity index in patients with BD (10), it would have been better if these factors had been included in the paper by Yilmaz et al (1).

I Balta<sup>1</sup>, S Balta<sup>2</sup>, S Demirkol<sup>2</sup>, M Demir<sup>2</sup>, C Ozturk<sup>2</sup>

<sup>1</sup>Department of Dermatology, Kecioren Training and Research Hospital, and <sup>2</sup>Department of Cardiology, Gulhane Medical Academy, Ankara, Turkey

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Sevket Balta, Department of Cardiology, Gulhane School of Medicine, Tevfik Saglam Sreet, 06018 Etlik-Ankara, Turkey.

E-mail: drsevketb@gmail.com

## Erratum

In the article 'Disease-modifying anti-rheumatic drugs for refractory severe knee synovitis in patients with peripheral spondyloarthritis: efficacy and predictors of response' by GT Sakellariou, FE Sayegh, AD Anastasilakis, I Bisbinas, GA Kapetanios (*Scand J Rheumatol* 2013;42:369–72), the authors report three errors in Table 1.

The fourth line of the second column should be blank

The fifth line of the second column should read 4 (28.6)

The sixth line of the second column should read 10 (71.4)

The correct table is printed below. We apologize for this error.

Table 1. Demographic and clinical characteristics of the studied patients.

Parameter	All patients (n = 45)	Knee synovitis responders (n = 14)	Knee synovitis non-responders (n = 31)	Knee synovitis responders vs. non-responders, p-value
Age at disease onset (years)	33.2 ± 1.7	29.2 ± 1.8	35.0 ± 2.3	0.054
Time interval between disease onset and DMARD initiation (years)	4.6 ± 0.7	5.8 ± 1.7	4.1 ± 0.6	0.376
Male gender	33 (73.3)	10 (71.4)	23 (74.2)	0.846
Disease subtype				0.011
AS	5 (11.1)		5 (16.1)	
PsA	22 (48.9)	4 (28.6)	18 (58.1)	
unSpA	18 (40.0)	10 (71.4)	8 (25.8)	
Psoriasis ever	22 (48.9)	4 (28.6)	18 (58.1)	0.067
HLA-B27*				0.023
Positive	9 (47.4)	2 (20.0)	7 (77.8)	
Negative	10 (52.6)	8 (80.0)	2 (22.2)	
Pattern of arthritis at DMARD therapy onset				0.577
Monoarthritis	14 (31.1)	4 (28.6)	10 (32.3)	
Oligoarthritis	29 (64.5)	10 (71.4)	19 (61.3)	
Polyarthritis	2 (4.4)		2 (6.4)	
Pattern of DMARD therapy				0.681
Monotherapy	37 (82.2)	12 (85.7)	25 (80.6)	
Combination	8 (17.8)	2 (14.3)	6 (19.4)	

DMARD, Disease-modifying anti-rheumatic drug; AS, ankylosing spondylitis; PsA, psoriatic arthritis; unSpA, undifferentiated spondyloarthritis.

\*Included only the patients with known HLA-B27 status (n = 19).

Values given as mean ± standard error of the mean (SEM) or n (%).