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Letter to the Editor

Co-occurrence of Psoriatic Arthritis with Venous Thromboembolism: Genetic Aspects

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TO THE EDITOR

In a recent article, it was investigated for the first time an increased risk of venous thromboembolism (VTE) occurrence among psoriatic arthritis (PsA) patients compared to controls in the general population without PsA [1], who demonstrated for the first time In the framework of this study, the authors conducted an elegant study involving 5.276 newly diagnosed PsA patients and 21,011 controls without PsA and they suggested that the increased risk of VTE in PsA patients appeared to be related to the underlying comorbidities and not independently associated with PsA. Particularly, PsA patients of older age and previous history of VTE only were found to be associated with increased risk of VTE. This data [1], raised some interesting points and we appreciate the opportunity to extend further this piece of novel information, considering that there is a well-studied genetic component in the pathogenesis of both diseases. VTE is a multifactorial disease, representing a worldwide health problem affecting people of all ages, sexes and races, and has 2 major subtypes: deep vein thrombosis and pulmonary embolism [2]. PsA is a seronegative chronic inflammatory joint disease with prevalence rates ranging between 0.3% and 1% worldwide that is associated with psoriasis (PS), characterized by inflammatory changes in attachments of articular capsules, tendons, and ligaments to bone surface [3].

The study of Gazitt et al. [1], posed the intriguing question concerning the putative role of a shared genetic background as regards with the co-occurrence of VTE with PsA. Recently, we reported various gene polymorphisms that are associated with an increased susceptibility for VTE and two more autoimmune diseases, rheumatoid arthritis (RA) as well as VTE and ankylosing spondylitis (AS) [4]. Aiming to provide a comprehensive update on the current understanding of the potential shared genetic component of VTE and PsA, we proceeded in an extensive search of the current literature aiming to find gene polymorphisms associated with both diseases. Thus, it was found that the rs1801133 single nucleotide polymorphism (SNP) of methylenetetrahydrofolate reductase (MTHFR) [5,6], the insertion/deletion (ID) polymorphism of the angiotensin-converting enzyme (ACE) (ACE I/D) [6,7], the rs1800629 of (TNF-a) [8,9], the rs7975232 of vitamin D receptor (VDR) [6,10], as well as the rs2853550 and rs16944 SNPs of interleukin 1 beta (IL1B) [6,11], genes are associated with the development of both diseases [7,8]. Taking into account that the majority of VTE susceptibility factors are related to haemostatic system and coagulation cascade, while the development of PsA is associated with a high number of inflammatory factors, it is unlikely an extended overlap between these categories of gene loci to be observed.

In conclusion, although the underlying molecular mechanisms involved in the development of VTE are still elusive, we managed to find a number of gene polymorphisms pointing out that certain genes can be considered as potential risk factors for developing both VTE and PsA, thus suggesting a shared genetic predisposition in limited cases. Therefore, based on the data collected from previously published genetic studies, we can assume that a "cross-talk" exists between the coagulation and inflammatory cascades, given that gene polymorphisms involved in inflammation may also result in an increased susceptibility towards VTE. Altogether, apart from the data presented by Gazitt et al. [1], who suggested that the increased risk of VTE in PsA patients is related to the underlying comorbidities and not independently associated with PsA, we pointed out a potential effect of some genetic factors on this co-occurrence. Further identification of shared genetic loci associated with VTE and PsA is of emerging interest and may help to delineate the mechanisms leading to a stronger clinical association between these diseases and ultimately result in the detection of novel therapeutic targets for new therapies.

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Authors' contribution

MIZ and GNG designed the work, analyzed the data, wrote the letter and approved the final manuscript.

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