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## **Antiphospholipid antibodies: crossroads between autoimmunity and infections?**

Sciascia Savino<sup>1,2</sup>, Radin Massimo<sup>1</sup>, Bazzan Mario<sup>3</sup>, Roccatello Dario<sup>1,2</sup>

1.Center of Research of Immunopathology and Rare Diseases- Coordinating Center of Piemonte and Valle d'Aosta Network for Rare Diseases, Department of Clinical and Biological Sciences, S. Giovanni Bosco Hospital and University of Turin, Turin, Italy.

2. SCU Nephrology and Dialysis, S. Giovanni Bosco Hospital, Turin, Italy.

3. UOSD Hematology and Thrombosis Unit, S. Giovanni Bosco Hospital, Turin, Italy.

### **Corresponding Author:**

Savino Sciascia, MD, PhD;

Center of Research of Immunopathology and Rare Diseases- Coordinating Center of Piemonte and Valle d'Aosta Network for Rare Diseases, and SCU Nephrology and Dialysis, S. Giovanni Bosco Hospital

Piazza del Donatore di Sangue 3, 10154, Turin, Italy.

Email [savino.sciascia@unito.it](mailto:savino.sciascia@unito.it) Tel +390112402056 Fax +390112402052

We read with interest the letter by Ponzetto and co-workers [1] commenting on our recently published Point of View paper [2]. They propose the hypothesis that testing for *Helicobacter pylori* (HP) and, when positive, managing that underlying infection might improve the current management of some patients with antiphospholipid syndrome (APS). Of note, they highlighted that HP's eradication might result in loss of antiphospholipid antibodies (aPL)[3]. There are several mechanisms through which pathogens can initiate or perpetuate autoimmunity and, while the factors causing production of aPL remain undefined, there is evidence that molecular mimicry is one of the mechanisms by which experimental APS can occur in association with certain pathogens [4]. However, when reviewing the available data on the topic, Abdel-Wahab et al. showed that, although the development of aPL with all traditional manifestations of APS were observed after variety of infections (mainly chronic viral infections including HIV and HCV), the causal relationship between infections and APS is still on debate [5]. Clinical evidences support that infections can trigger the development of catastrophic APS [6]; nevertheless, the possible contribution of various infections in the pathogenesis of APS need further longitudinal and controlled studies to establish the incidence, and to better quantify the risk and the outcomes of aPL-related events after infection.

Finally, an important question still remains: is the aPL negativization enough to change the therapeutic approach in a patient with APS? Recent data suggest that persistent negative aPL profile is not an indication to interrupt oral anticoagulant therapy as patients are still at high risk of recurrences [7].

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