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See case report on page 81

Antiphospholipid Syndrome: A complex disease

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In the last three decades, a variety of clinical manifestations involving almost all organs and tissues (cardiac, pulmonary, neurological, renal, cutaneous, haematologic, gastrointestinal, ocular, skeletal and endocrinologic), have been described associated with antiphospholipid antibodies (aPL) . Nevertheless, the exact mechanism underlying the pathogenesis of aPL-mediated damage has been poorly recognized. Inflammatory mechanisms beyond thrombosis have been proposed in some clinical presentations, suggesting a role for immunomodulation in therapeutic strategy.

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Commentary

In the last three decades, a variety of clinical manifestations involving almost all organs and tissues (cardiac, pulmonary, neurological, renal, cutaneous, haematologic, gastrointestinal, ocular, skeletal and endocrinologic), have been described associated with antiphospholipid antibodies (aPL) (1). Nevertheless, the exact mechanism underlying the pathogenesis of aPL-mediated damage has been poorly recognized. Inflammatory mechanisms beyond thrombosis have been proposed in some clinical presentations, suggesting a role for immunomodulation in therapeutic strategy (2).

This interesting case-report by Ardalan et al. (3) definitely illustrates the multiple expressions of this disease. Although the classical criteria included in the revised classification for antiphospholipid syndrome (APS), pregnancy morbidity or thrombosis and persistence of positive aPL (4) were not present, clinical features such as thrombocytopenia, renal dysfunction and mitral valve regurgitation, undoubtedly support the diagnosis of APS. Motor neuropathy, a rare and recently described presentation (5), added complexity to the diagnosis and highlights the importance of a multidisciplinary approach of these patients. Antiphospholipid syndrome is really a disease with protean faces.

Conflict of interest

The author declared no competing interests.

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