□ LETTERS TO THE EDITOR □

Transient Eosinophilia with Catastrophic Vessel Occlusion

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To the Editor We read with interest the case of a 67 year old man who developed cerebral venous thrombosis under conditions of transient eosinophilia in the absence of any other genetic or acquired pro-thrombotic factors (1). We herein report the case of a 34 year old woman who presented with the most catastrophic vascular syndrome amongst types of transient eosinophilias reported to date (reviewed in 1).

The patient was admitted for the treatment of digital ischaemia and breathlessness and was found to have a thrombus in the superior and inferior vena cava, pulmonary arteries and right atrium, both femoral veins and right hepatic and portal vein. An inferior vena cava filter was inserted; however she became haemodynamically unstable and received systemic thrombolysis. Nevertheless Doppler ultrasounds showed persistent clots in the bilateral popliteal veins and right superficial femoral vein and she subsequently received intravenous methylprednisolone at a dose of 500 mg for two days which was then switched to oral therapy at a dose of 50 mg daily and slowly tapered down over four months. The eosinophil count at presentation was $2.1 \times 10^9/L$, with a peak of $3.0 \times 10^9/L$ observed after two weeks followed by normalisation after eight months. The platelet count was $43 \times 10^9/L$ at presentation and normalised within two weeks. The results of bone marrow biopsy were normal, and cytogenetics for FIP1L1-PDGFR α , Bcr-Abl and JAK2V617F were negative. Moreover, antinuclear antibodies, P-antineutrophil cytoplasmic antibodies (ANCA) and C-ANCA were negative, as was serology and a stool examination for tissue and intestinal parasites. A full thrombophilia

screen was repeatedly negative. Why some patients with transient eosinophilia of unknown cause develop thrombosis remains a mystery. It is known that eosinophilic cationic protein (ECP) neutralizes negatively charged heparin, thus inhibiting the anticoagulant effect of antithrombin on factor X and factor II (reviewed in 2). ECP and membrane basic protein (MBP) may interfere with thrombomodulin and thereby prevent the thrombin-dependent activation of anticoagulant protein C (2). Furthermore, eosinophils may release tissue factor (2), whilst eosinophil peroxidase possibly upregulates the levels of tissue factor on endothelial cells (2) and jointly promotes the activation of factor X via factor VII (2). Because there is no definite threshold level above which eosinophils are more pro-thrombotic, the detection of activated peripheral blood eosinophils may be helpful, as CD66b is expressed on the eosinophil surface (3), where it regulates adhesion and binding to endogenous and exogenous carbohydrate ligands (4). However, this marker has been neither validated nor introduced into clinical practice. Therefore, it would be wise to offer thromboprophylaxis in patients with transient eosinophilia until their condition resolves.

The author states that he has no Conflict of Interest (COI).

Ames PRJ

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