EOSINOPHILIC FASCIITIS SECONDARY TO RANIBIZUMAB INJECTIONS

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Background: EF is a rare disorder characterized by fasciitis and peripheral eosinophilia; it is differentiated from scleroderma by the pattern of skin involvement with sparing of the digits, fascial sparing of dermis and absence of RP. It can be regarded as scleroderma-variant with progression to scleroderma in some cases. It has been postulated that the disturbed vascularization found in scleroderma skin is due to reduced expression of the potent angiogenic factor vascular endothelial growth factor (VEGF). We report a case of EF, possibly secondary to the use of ranibizumab intravitreal injection (anti-VEGF mAb) for wet age-related macular degeneration (AMD).

Methods: A 72 year old woman receiving intravitreal injections of ranibizumab for wet AMD presented with a history that 6 months previously she had received her first injection of ranibizumab after which she noticed swelling of her legs and forearms. After the second injection, the swelling increased and spread from feet to thighs. The treatment was suspended, with gradual settling of swelling but she was left with very tight skin over her forearms with loss of movement at wrists and flexion deformities of her fingers developing an early prayer sign. She had tight skin over her feet, calves and thighs with some residual lumpiness. There were no telangiectasiae, sclerodactyly or mucosal involvement. She was not taking any regular medications including health supplements. She was treated with prednisolone 20 mg daily and Mycophenolate 1 g daily had regular monitoring blood tests and was reviewed monthly. At follow up after 3 months she reported feeling well in herself, was walking long distances and experienced no further spreading of skin tightening. She is maintained on 20 mg prednisolone and 2.5 mg daily dose of MMF and 9 months after the initial injections of ranibizumab she received two further intravitreal injections as her sight was deteriorating with no worsening of her condition.

Results: There was evidence of normochromic anaemia, eosinophilia (1.9 x 109), thrombocytosis, polyclonal increase in immunoglobulins IgG (58.7 g/l) with elevated ESR (102 mm/h) and CRP (56 mg/l), ANA and ANCA negative, ENA positive for Ro but negative for ScL70. Her albumin was low but with normal liver function, renal function tests with negative serum free light chain analysis. There was no evidence of pulmonary fibrosis on high-resolution CT scan of the chest, pulmonary function tests and echocardiography were normal with no evidence of pulmonary hypertension.

Conclusion: EF is a rare disorder, the cause of which remains unknown. We consider that in this woman’s case it was possibly triggered by the use of ranibizumab, an inhibitor of VEGF.

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