Eosinophilic Cellulitis (Wells’ Syndrome): Treatment with Minocycline

Sir,

Tetracyclines have been widely used in dermatology as antibiotics in acne. More recently, they have also been used as immunomodulators in non-infectious skin disorders (1). Especially those dermatoses that are mediated by eosinophilic granulocytes (2), like bullous pemphigoid, appear to respond favourably to tetracyclines. Here, we report a case of eosinophilic cellulitis (Wells’ syndrome) that responded well to oral minocycline.

CASE REPORT

A 75-year-old woman presented with a history of recurrent itchy and painful swellings, which had started in the summer. The lesions occurred on the hands, wrists, face and lower legs and slowly resolved in 2–4 weeks, leaving slate grey pigmentation. She noticed a relation with stripping of plants (Salvia) in her garden. There was no history of insect bites. Her medical history included a low-grade non-Hodgkin’s lymphoma, which had responded well to chemotherapy 7 years earlier and had been in stable partial remission since then. She had also had herpes zoster, thrombosis, recurrent urinary tract infection and adverse skin reactions to diclofenac and terbinafine.

Physical examination of the skin showed up to 6 cm large, dome-shaped, erythematous, firm oedematous swellings on the right jaw and the forehead, with vesicles discharging a clear exudate. The eyelids were swollen. On her hands and lower legs erythematous-squamous lesions of previous nodi were present. In the neck and inguinal area reactive lymph nodes were palpable.

Histological examination of lesional skin showed perivascular and interstitial infiltrates, consisting of eosinophilic granulocytes and lymphocytes reaching the septa of subcutaneous adipose tissue. The dermis showed oedema and basophilic degeneration of collagen bundles, some covered by eosinophilic material (“flame figures”). Immunofluorescence microscopy of lesional skin showed intraperiodal vesicles filled with numerous eosinophils, and in the dermis discrete granular deposits of fibrin and IgM, with clusters of IgG and fibrin-coated collagen bundles. In the vessel walls granular deposits of complement C3c were found. In normal skin no immune deposits were present. There were no circulating auto-antibodies detectable with monkey oesophagus substrate.

Laboratory investigations of peripheral blood showed increased levels of eosinophilic granulocytes 1.57 × 10E9/L and 19.7%. ESR 79 mm/1h, IgE > 2000 E/ml, IgG 17.5 g/l with an IgG lambda paraprotein and C-reactive protein 16 mg/l. The following laboratory studies were either negative or within normal limits: haemoglobin, platelet count, serum IgA and IgM, alpha-1-antitrypsin, liver- and kidney screening test, antinuclear antibody, antineutrophil cytoplasmic antibodies, serologic tests for Borrelia burgdorferi, cytomegalovirus, Toxoplasma gondii, Echinococcus ascaris, toxocara, Entamoeba histolytica, Taenia solium, Trepomonas pallidum, Epstein-Barr virus and human immunodeficiency virus. Urinalysis showed leukocytes. Parasitologic examination of faeces was negative. CT-scan revealed a diffuse lymphadenopathy in the neck.

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Lars Detlev Köhler, MD, Matthias Möhrenschlager, MD, and Johannes Ring, MD
Department of Dermatology and Allergy Biederstein, Technical University of Munich, Biedersteiner Str. 29, D-80802 Munich, Germany.

REFERENCES


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Els B. Stam-Westerveld, M.D.¹, Simon Daenen, M.D., Ph.D.², Jan B. Van der Meer, M.D., Ph.D.³ and Marcel F. Jonkman, M.D., Ph.D.¹* Departments of ¹Dermatology, and ²Hematology, Groningen University Hospital, Groningen, Hanzeplein 1, P.O. Box 30.001, NL-9700 RB Groningen, The Netherlands.

* Corresponding author

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