

Remission of Delayed Pressure Urticaria after Eradication of *Blastocystis hominis*

Nicoletta Cassano^{1,2}, Bianca Maria Scoppio², Maria Carmela Loviglio² and Gino A. Vena^{2*}

¹Istituto Dermatopatico dell'Immacolata, IDI, IRCCS, Rome and ²2nd Unit of Dermatology, Department of Internal Medicine, Immunology and Infectious Diseases, University of Bari, Policlinico, Piazza Giulio Cesare, 11, IT-70124 Bari, Italy. *E-mail: g.vena@dermatologia.uniba.it

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Sir,

Blastocystis hominis is one of the most common human intestinal protozoans in the world, especially in developing countries. Nevertheless, current knowledge about this parasite is incomplete and, despite recent progresses, several issues are still controversial and much debated, such as the clinical relevance and pathogenicity (1–4). *B. hominis* has been found in patients with gastrointestinal symptoms as well in asymptomatic individuals. It has long been considered a harmless commensal but growing evidence seems to support its role as a pathogenic parasite. In the absence of conclusive data, it could be hypothesized that the pathogenic potential and virulence of *Blastocystis* are conditioned by other concomitant factors, such as the host response, and depend on the number of organisms or the type of strains, considering that a remarkable antigenic and genetic heterogeneity among isolates has been demonstrated (4). Extra-intestinal manifestations of *B. hominis* infection have rarely been reported and include infectious or reactive arthritis (5–7) and skin disorders (8–11), such as palmoplantar or diffuse pruritus and chronic urticaria.

A 32-year old woman with a history of allergic rhinitis had since October 1998 suffered from chronic recurrent urticaria which was almost controlled by treatment with H1-receptor antagonists. Whealing subsided after 4 years, when cutaneous manifestations were replaced by painful swellings in pressure sites. For this reason, she was examined in November 2002, where a delayed

pressure urticaria (DPU) was diagnosed. Treatment with systemic corticosteroids gave only partial benefit. Subsequently, dapsone was introduced at a dose of 25 mg/day. Symptoms gradually ameliorated over a 3-month period without disappearing. After dapsone discontinuation, her symptoms became severe and disabling within 1 month, despite regular treatment with H1-receptor antagonists. Skin lesions on the feet were sometimes associated with marked pain, which was so intense that it induced the patient to wear larger shoes than usual. Whealing of common urticaria or angioedema never occurred. The only concomitant symptoms reported by the patient were mild ponderal loss and persistent asthenia. Pressure challenge tests confirmed the diagnosis of DPU. Laboratory investigations failed to disclose any systemic diseases, including malabsorption, endocrinological, autoimmune and rheumatological disorders. Full blood count, including eosinophil count, erythrocyte sedimentation rate, C-reactive protein, cryoglobulins, circulating immune complexes, C3, C4, C1-INH, IgE and other immunoglobulins were all within the normal range. Chest radiography, radiographic examination and laser Doppler flowmetry of the limbs revealed unremarkable findings, as well as serological investigations, oropharyngeal swabs and faecal antigen test with *Helicobacter pylori*. Stool examinations on three consecutive samples were positive for *B. hominis*. The patient was then treated with metronidazole 1.5 g/day for 10 days. DPU began to improve 2 weeks after

the start of therapy and disappeared 4 weeks later. Three faecal analyses were performed 4 weeks after the end of treatment and gave negative results. No recurrence of DPU was observed during 6 months of follow-up.

The successful unexpected response of DPU to *B. hominis* eradication seems to suggest a possible role in DPU in our patient, although DPU has never been associated with infectious diseases.

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