

Vogt-Koyanagi-Harada Syndrome Associated with Psoriasis and Autoimmune Thyroid Disease

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Accepted January 21, 2008.

Sir,

Vogt-Koyanagi-Harada syndrome (VKH) is a bilateral granulomatous panuveitis associated with cutaneous (poliosis, alopecia, and vitiligo), neurological (aseptic meningitis) and auditory (dysacusis, tinnitus, vertigo) manifestations related to a cell-mediated autoimmune process against melanocytes (1). We report here a case associated with psoriasis and Hashimoto's thyroiditis. Cyclosporin A (CyA) treatment was efficient for the psoriasis and VKH symptoms. Although this association may be coincidental, we discuss the potential links between these 3 conditions, which support the hypothesis of a common genetic basis with specific variants of susceptibility genes.

CASE REPORT

A 49-year-old Italian woman was referred for the management of an extensive vitiligo, which had been evolving for the past 6 months. Her medical history was notable for: (i) psoriasis restricted to the elbows and the knees since the age of 23 years, without any familial history; (ii) Hashimoto's thyroiditis; (iii) dysacusis, both diagnosed in 1997; and (iv) bilateral anterior uveitis with bilateral hyalitis in 2000. Bilateral

cataract developed subsequently to the chronic ocular inflammation and was treated by surgery in 2001. At that time, the panuveitis was considered as idiopathic and responded dramatically under local and systemic corticosteroid therapy (prednisone 1 mg/kg/day). However, attempts to taper corticosteroid doses resulted in recurrences of ocular inflammation at a dose of 10 mg/day. Moreover, psoriasis flared-up while tapering corticosteroids.

At presentation, vitiligo affected the face, the neck, the trunk, the limbs and trauma and friction sites. Psoriasis patches were strictly confined to the vitiliginous areas. Poliosis, frontal non-cicatricial alopecia and eyebrows depilation were also noted (Fig. 1). The patient mentioned daily tinnitus. Audiometry revealed presbycusis. Moreover, she presented chronic headaches with nuchal rigidity without any other focal neurological signs. Examination of the cerebrospinal fluid showed pleocytosis and an increase in protein content (0.57 g/l). Ocular examination and angiography confirmed that the disease was VKH at the chronic phase. HLA I and II typing were A*02 A*30 B*13 B*18 and DRB1*07 DRB1*13, respectively. Oral corticosteroid therapy was inefficient on VKH symptoms and triggered psoriasis flare. Therefore, treatment with CyA was initiated



Fig. 1. Psoriasis restricted to the vitiliginous area on: (A) the abdomen and (B) the limbs. (C) Poliosis of the eyebrows and diffuse frontal alopecia. (D) Close-up of poliosis of the left eyebrow with a psoriatic lesion underneath.

(300 mg/day) with a complete clearance of psoriatic lesions and improvement of the auditory and neurological symptoms. Later on, repigmentation of the vitiliginous lesions was noted by the patient. However, increased serum creatinine levels prompted us to diminish the dose of CyA and vitiligo relapsed.

DISCUSSION

An association between VKH and psoriasis has been reported in 4 cases in the literature (3 women out of 4, aged between 39 and 68 years) (2–5). In most cases psoriasis preceded the onset of VKH by 1–25 years (2–4). Lesions were strictly restricted to the vitiliginous areas in one case (2). It is noteworthy that one patient did not present any dermatological sign of VKH (3).

CyA is known to be an effective drug for the treatment of both psoriasis (6) and refractory inflammatory eye diseases, including VKH (1). Nowadays, it is used at a dose of 5 mg/kg/day for the latter indication (1). We observed here an excellent efficiency on skin symptoms, with a complete clearance of psoriatic lesions and obtained a good control of the most disabling symptoms of VKH, such as headaches. Interestingly, the patient reported spontaneous improvement of vitiliginous lesions. CyA has seldom been used for vitiligo. It has proved efficiency on a vitiliginous line of chickens (7), but failed to show similar effects in humans (8). Moreover, vitiligo seemed to worsen after tapering the doses of CyA, as observed in chickens (7). Therefore, the real effect of CyA on vitiligo in our case remains unclear.

VKH has been reported with other auto-immune diseases (5, 9, 10), including autoimmune thyroid diseases (AITD) such as Grave's disease (10) or hypothyroidism (5, 9, 11, 12). To our knowledge, the association of VKH, psoriasis and AITD has been reported in one case (5). Moreover, the patient also developed corneal melting; a rare autoimmune ocular disease characterized by corneal thinning and elevated risk of perforation (5).

This triple association remains coincidental with regard to the number of reports. However, in all 3 diseases there is evidence of underlying immunological mechanisms. Specific HLA genotypes are associated with an increased risk of VKH among individuals (1). A T-cell lymphocyte-mediated autoimmunity is observed against pigmented cells of the uvea, the skin, the inner ears and choroids plexus (1, 13). On the other hand, some cases of association between psoriasis and AITD or vitiligo have been reported (14). In our case, one could speculate on the possible existence of a shared eye-thyroid-skin autoantigen explaining such an association. However, this antigen remains to be identified. A more interesting hypothesis in our patient would be the existence of a susceptibility gene predisposing to autoimmune diseases in general (14, 15). Indeed, autoimmune diseases share

common underlying mechanisms, such as reactivity to self-antigens by the humoral or cellular immune system and genetic associations with HLA (15). Some specific variants may confer susceptibility to epidemiologically associated autoimmune and auto-inflammatory diseases, as illustrated, for instance, by the occurrence of vitiligo, AITD and psoriasis in several members of families with multiple auto-immune diseases associated with vitiligo (14).

Conflict of interest: None to declare.

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