CASE REPORT

A case of vitiligo that followed the path of a varicose vein in the lower leg

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Abstract : Vitiligo is an acquired chronic depigmenting disorder of the skin and is characterized by the destruction of melanocytes. One of the clinical features of vitiligo is that damage to normal skin frequently results in the formation of depigmented macules, which is known as Köebner's phenomenon (KP). Here, we presented a case of vitiligo, in which depigmented macules followed the course of a dilated varicose vein. Dilatation of blood vessels was considered to contribute to the development of the vitiliginous lesions as a trigger for KP. Any kind of skin injury can trigger KP, but this is only the second case in which a dilated blood vessel caused KP in vitiligo. J. Med. Invest. 71 : 177-178, February, 2024

Keywords **:** *vitiligo, varicose vein, Köebner's phenomenon*

INTRODUCTION

Vitiligo is an acquired chronic depigmenting disorder of the skin and is characterized by the destruction of melanocytes. Its estimated prevalence is 0.5-2% of the population in worldwide (1). Vitiligo affects both genders equally and commonly begins in childhood or young adulthood but it can develop at any age. Approximately 20% of patients with vitiligo have a positive family history of vitiligo (2). The clinical manifestation is hypopigmented and asymptomatic macules that appear chalk- or milk-white in color, with sharply demarcated margins. These lesions usually arise in areas exposed to chronic trauma, especially the hands or arms. The distribution of the lesions is usually symmetrical, although sometimes it is unilateral and may have a dermatomal arrangement. Histopathologically, there is a marked absence of melanocytes and melanin in the epidermis. But diagnosis is mainly based on clinical features and a skin biopsy is rarely required (3). The precise pathogenetic mechanisms of vitiligo are complex, and multiple mechanisms, including genetic mechanisms, autoimmunity, oxidative stress, and neurological dysfunction, have been proposed. However, there is now consensus on the autoimmune nature of vitiligo (1, 4). Vitiligo could coexist with other autoimmune disorders such as autoimmune thyroid disease, particularly Hashimoto thyroiditis and Graves disease, pernicious anemia, diabetes mellitus, and Addison's disease. Treatment for vitiligo includes potent topical corticosteroids and tacrolimus (0.03% or 0.1%). Narrow-band ultraviolet B (UVB) light therapy can be used in patients not responding to topical treatment or in patients with extensive disease. However, the treatment of vitiligo is often unsatisfactory and there is as yet no consistently reliable therapy.

One of the clinical features of vitiligo is that damage to normal skin frequently results in the formation of depigmented macules, which is known as Köebner's phenomenon (KP). KP is a well-known phenomenon in dermatology and is defined as the development of new dermatosis lesions in normal skin after trauma or other stimuli. Any kind of skin injury can trigger KP, but trauma is the most common stimulus (5).

Here, we report a rare case of vitiligo, in which depigmented macules followed the path of a dilated varicose vein. We suggest that this pattern may have been attributable to KP induced by extension of the skin due to the underlying varicose vein. Various stimuli are known to induce KP, but this is only the second case in which a dilated blood vessel caused KP in vitiligo.

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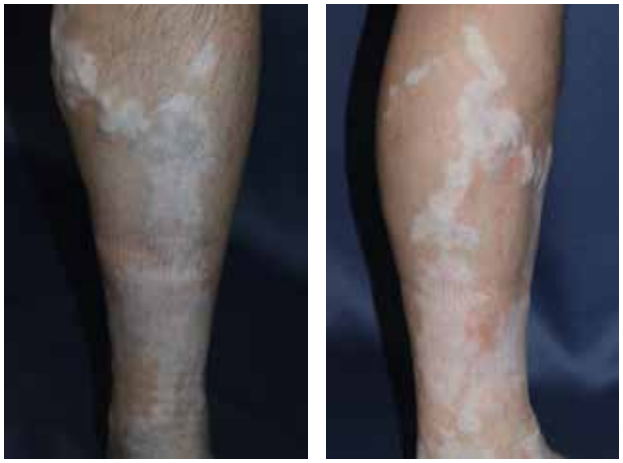
CASE REPORT

A 55-year-old Japanese male with a 20-year history of vitiligo was referred to our department due to the recent enlargement of depigmented macules on his lower legs. Otherwise, he was healthy and had no remarkable medical history, including of thyroid disease. He had been a civil servant and had never been exposed to chemicals that could induce depigmented lesions occupationally. There was no family history of similar symptoms. He had been intermittently treated with prednisolone valerate acetate cream, diflucortolone valerate cream, and narrow-band UVB treatment for the depigmented macules on his face, hand, and foot. Several years ago, he noticed a varicose vein on his left lower leg, and whitish lesions had appeared in the same area in the past 2 years. A physical examination showed that the depigmented macules which appeared chalk-white in color, with sharply demarcated margins followed the course of the varicose vein (Figs. 1a, b). The depigmented lesions were not restricted to the top of the enlarged blood vessel, but covered the entire surface of the skin above the enlarged blood vessel. There were also several small depigmented macules on his face and dorsal of the hands. Based on these clinical findings, we made a diagnosis of vitiligo. We treated these lesions with topical fluocinonide cream (40 g/month) and 0.1% tacrolimus hydrate ointment (10 g/month). Although pigmentation was gradually observed in the lesions on the face and hand, depigmented lesions on the lower leg were intractable. Sixteen months later, since the patient gradually developed numbness of the lower leg, he underwent endogenous laser ablation therapy. We continued the same topical treatment after this procedure but no remarkable changes were observed for the first couple of months. However, when the patient returned to the hospital 4 months after the procedure, some

pigmentation was seen in the lesions on his lower leg (Fig. 2).



1a 1b

Figure 1a, b. Linear hypopigmented lesions were detected along a varicose vein.

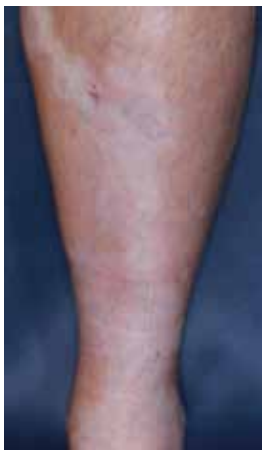


Figure 2. Four months after endogenous laser ablation therapy. Slight pigmentation was observed in the lesion.

DISCUSSION

Vitiligo is a well-known type of dermatosis that can induce KP (5). The incidence of KP in vitiligo ranges between 21% and 62% (6). Any kind of skin injury can trigger KP : physical wounds, scratching, mechanical friction, burns, chronic pressure, inflammatory dermatoses, and radiotherapy. Among them, trauma is the most common stimulus. Since not all injuries induce KP in the same patient, the depth, width, duration, and type of the injury may be relevant. Chronic superficial minor damage can also induce KP in vitiligo patients. Vitiligo lesions frequently develop on the hands, wrist, and waist, where repeated friction due to daily activities occur, which can be interpreted as delayed KP. The pathogenesis of KP in vitiligo remains unclear, but several theories, including immunological mechanisms, deficient melanocyte adhesion, and increased oxidative stress, have been suggested (5).

In our case, depigmented macules were observed along the line of a dilated varicose vein. It may have been coincidental, but the shape of the depigmented lesions, which clearly followed the path of the varicose vein, suggested that the dilated blood vessel contributed to the development of the vitiligous lesions as a trigger for KP. Chronic friction from clothes due to the prominence of the blood vessel may have been involved. However, the vitiliginous lesions covered the entire surface of the dilated blood vessel, rather than being localized to the top of the enlarged blood vessel's surface. Previously, a similar case of vitiligo that followed the path of a varicose vein was reported (7). This previous case and our case suggest that blood vessel dilation due to varicose veins could be a triggering factor for KP in vitiligo. In addition, cases of vitiligo in which the vitiliginous lesions coincided with keloid plaques and striae distensae have also been reported (8, 9). These cases indicate that continuous stretching tension could be a triggering factor for KP in vitiligo. Defective melanocyte adhesion is considered to be one of the mechanisms of KP in vitiligo (5). It has been reported that E-cadherin, which is the primary adhesion molecule linking melanocytes and keratinocytes, is significantly altered in the non-lesional skin of patients with vitiligo (10). As Eun SH *et al*. suggested (8), continuous stretching tension may weaken the adhesion between melanocytes and keratinocytes, resulting in the development of new vitiligo lesions.

Although it is not life-threatening or symptomatic, vitiligo is psychologically devastating and frequently intractable to treatment. Since KP plays an important role in the development of vitiligo lesions, understanding the factors that can trigger KP is important. Our case indicates that varicose veins may be another cause of KP in vitiligo.

CONFLICT OF INTEREST

None declared

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