

## SHORT COMMUNICATION

### Rejection-mediated Regression of Melanocytic Naevi in an Immunosuppressed Organ Transplant Recipient

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Eruptive melanocytic naevi and/or excess of melanocytic naevi have been reported in several groups of immunosuppressed patients. The eruption of melanocytic naevi after immunosuppression is a peculiar phenomenon indicating that the immune system may play a major role in limiting proliferation of melanocytes (1). In this article we describe a patient with excess of post-transplant melanocytic naevi that spontaneously disappeared after graft rejection.

#### CASE REPORT

In May 2007, a 50-year-old woman underwent pancreas-kidney transplant for a diabetic nephropathy. Her immunosuppressive maintenance therapy consisted of tacrolimus (7 mg/day), cortone acetate (25 mg/day) and mycophenolic acid (720 mg/day). In June 2008, on physical examination, multiple acquired pigmented lesions were observed, especially on the patient's back (Fig. 1a). She had no personal history of longstanding sun exposure or sunburns and there was no family history of melanoma. Dermoscopic examination revealed the presence of brown globules throughout melanocytic naevi, especially peripherally. In July 2011, she suffered an acute rejection of the renal allograft treated, with good response, with steroid bolus. Kidney rejection was avoided, but 2–3 months later several melanocytic naevi disappeared or became smaller (Fig. 1b).

#### DISCUSSION

Eruptive melanocytic naevi and/or excess of melanocytic naevi have been reported in several groups of immunosuppressed patients, notably HIV-positive patients, organ transplant patients (2, 3), patients affected by chronic myelocytic leukaemia (4), patients treated with chemotherapy for cancer (5) or with immunosuppressive agents including biological therapies (6, 7). This phenomenon appears to be consistent with the idea that an intact host immune system may normally limit the proliferation of melanocytes. We described a case of eruptive post-transplant melanocytic naevi phenomenon and the spontaneous involution of the melanocytic naevi after graft rejection and restoration of complete immune responsiveness (2).

We observed a regression of the excess of melanocytic naevi after the acute kidney transplant rejection although the increase of the immunosuppressive therapy should have induced melanocytic proliferation. It is plausible that this event has been the result of the immune response against the kidney-graft which has probably involved antigens exposed on both kidney and melanocytes. The clinical effect of melanocyte fading can be considered as the result of this exaggerated immune response.

This case is a further clinical example of the role played by the host immune system in controlling melanocyte proliferation.



Fig. 1. Excess of melanocytic naevi of the patient's back before acute kidney rejection (a). The same area after the episode of rejection (b).

The authors declare no conflict of interest.

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