The clinical manifestations of metastatic skin cancer are classified broadly into nodular, inflammatory and sclerodermoid.[4] Some such cancers are well known to present unique clinical manifestations, such as carcinoma en cuirasse, sister Mary Joseph's nodule, and so on.[5,6] Annular purpuric eruption is an extremely rare condition, and only one other case has been reported of annular purpuric eruption with skin metastasis as a unique clinical manifestation. Watanabe *et al.*[7] suspected that the purpuric colour was related to the colour of the tumour cells in lymphatic vessels in association with skin metastasis of SCC.

Our case was histopathologically proved to have cutaneous metastasis of apocrine carcinoma. Furthermore, it was clinically humorous to present giant annular purpuric eruption. Histopathological examination found no obvious extravasation of red blood cells, but there were many tumour cells infiltrating into lymphatic vessels. We speculate that the obstruction of the lymphatic vessels might have caused centrifugal spreading to make the annular form, and that dilated vessels in the deep dermis caused the clinically observable purpuric eruption.

In conclusion, this is the first report of cutaneous metastasis in apocrine carcinoma with the unique clinical condition of giant annular purpuric eruption. When we deal with giant annular purpuric eruption, we should consider skin metastasis of apocrine carcinoma as a differential diagnosis.

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Loss of alpha-dystroglycan expression in cutaneous melanocytic lesions

Dear Sir,

The dystroglycan complex (DG), originally characterized in muscle and in genetic muscle diseases, links the epithelial cell cytoskeleton to the basement membrane.[1] DG has been shown to be involved in skin morphogenesis and in epithelial carcinogenesis, modulating cell differentiation and adhesion, assembly of the epithelial basement membrane and interactions with the extracellular matrix (ECM).[2] DG comprises an alpha (α-DG) and a beta (β-DG)-subunits, with the α-DG subunit being the extracellular, functional part of the complex and binding several ECM components. Loss of α-DG function has been reported in several epithelial- and neural-derived tumours and correlated with tumour grading, progression and clinical outcome.[3] In the skin, DG is localized at the dermo-epidermal junction and is produced by epidermal keratinocytes and dermal fibroblasts.[4] Tissue expression of the α-DG subunit has not been previously characterized in cutaneous melanocytic nevi and in malignant melanoma. In this study, expression of the DG complex was analysed by Western-blot in cell extracts from human normal melanocytes (hMel) and three melanoma cell lines (BLM, M14, IF6). Furthermore, tissue expression of α-DG was assessed by immunohistochemistry in a panel of archival melanocytic lesions, including melanocytic nevi ($n = 20$), cutaneous melanoma ($n = 51$) and melanoma metastases ($n = 53$). A polyclonal antibody to alpha-DG (clone VIA4-1) from Upstate Biotechnology and a monoclonal antibody to β-DG (clone 43DAG/8D5) from Novocastra were used for the analyses. β-DG molecule was detectable in both melanocytes and melanoma cells displaying a higher expression in IF6 melanoma cells compared to normal melanocytes. On the other hand, a decreased expression of α-DG was observed in all three melanoma cell lines compared to normal melanocytes (Fig. 1). At the tissue level, in benign melanocytic nevi α-DG was expressed by basal melanocytes at the dermo-epidermal junction and by epithelioid melanocytes, organized in nests, in the superficial dermis (Fig. 2a,b). In primary cutaneous melanoma, α-DG expression appeared to be lost in almost all cases. Loss of α-DG expression in

melanoma was observed at all Clark invasion levels, both in the epidermis and dermo-epidermal junction as well as in the superficial to deep dermis (Fig. 2c–e). In melanoma metastases, expression of α-DG was evident in 20.8% of cases, being mainly focal and limited to spindle-cell like melanoma cells (Fig. 2f). Previous studies have only characterized the *in-vitro* expression of DG subunits by melanocytes and melanoma cells, as well as tissue expression of β-DG in melanocytic nevi.[5,6] We report a differential pattern of α-DG expression between benign and malignant melanocytic tumours, suggesting a peculiar role of the DG complex in melanocyte biology and melanomagenesis. In benign melanocytic nevi, α-DG displayed a consistent expression pattern at the dermo-epidermal junction, where interactions with basal keratinocytes and ECM components are prominent in the epidermal melanin unit. In primary cutaneous melanoma, a loss of α-DG tissue expression was observed across all stages of progression. This finding is compatible with the loss of anchorage to the basement membrane in the early intraepidermal proliferation of transformed melanocytes due to an altered expression of ECM binding proteins.[7] Loss of the α-DG subunit in melanoma is likely to be caused by several post-translational mechanisms, such as an altered glycosylation pattern and/or proteolytic degradation of the membrane complex.[8] Loss of α-DG function results in a disruption of cell-to-ECM interactions and might promote

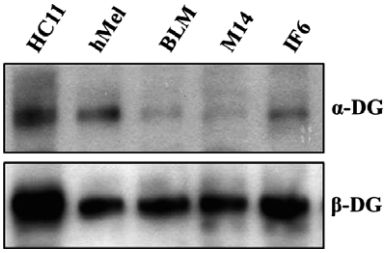

Figure 1 Levels of expression of α- and β-dystroglycan (DG) in normal human melanocytes (hMel) and melanoma cell lines (BLM, M14, IF6), as assessed by western-blot analysis.

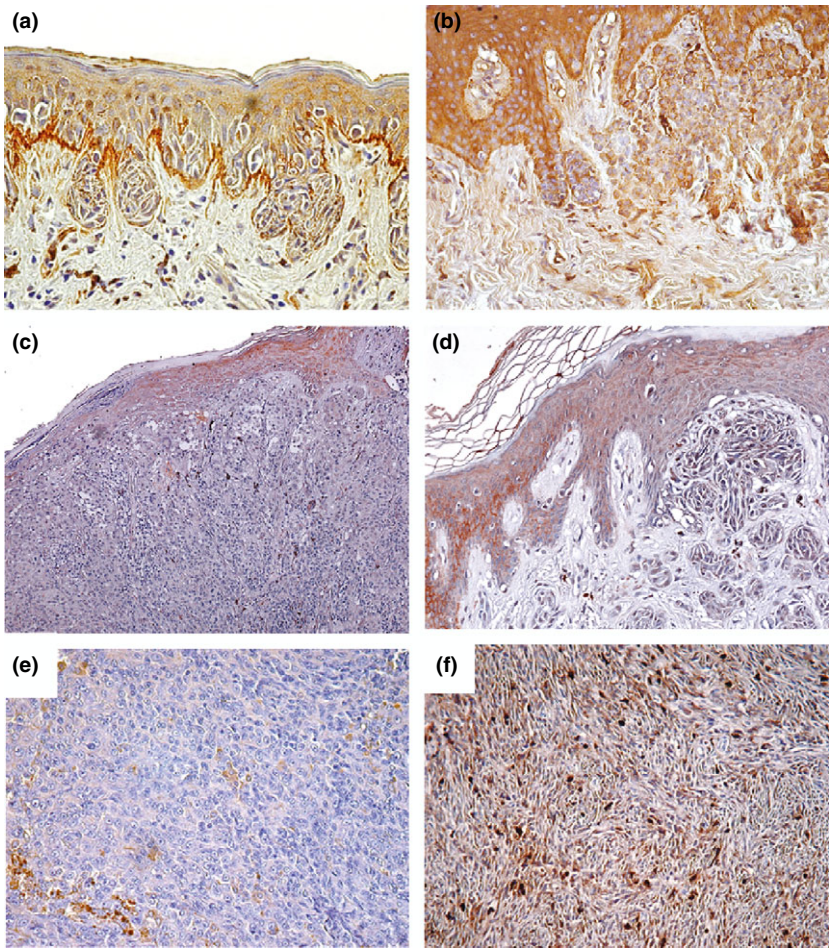

Figure 2 α–Dystroglycan (DG) staining in representative benign melanocytic nevi (a, b), primary cutaneous melanoma (c, d) and melanoma metastases (e, f). (a) staining of junctional melanocytic nests in relation with the basement membrane (internal control) and at the tip of the rete ridges; (b) diffuse intracytoplasmic and intercellular staining of melanocytic nests at the dermo-epidermal junction and of scattered epithelioid melanocytes in the upper dermis; (c, d) complete loss of staining in the irregular nests at the dermo-epidermal junction and dermis; (e) loss of staining of confluent sheets of epithelioid melanoma cells; (f) diffuse positive, intercellular staining of atypical spindle cells.

tumour invasion and metastasis. However, the observed expression of α-DG on cells with spindle-cell like morphology in melanoma metastasis suggests that its restoration could favour the implant of cells in metastatic sites, as previously reported in other cancers.[9] Further *in vitro* and *in vivo* studies are warranted to assess the role of the DG complex in melanocyte-keratinocyte interactions and in human melanomagenesis.

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Isolated unilateral facial nerve palsy: an unusual manifestation of recurrent diffuse large B-cell lymphoma

Editor

Relapse of diffuse large B-cell lymphoma (DLBCL) after 5 years of clinical remission is uncommon and can pose a significant diagnostic challenge for practitioners.[1,2] Herein is described only the second reported case, to our knowledge, of DLBCL relapse presenting as unilateral facial palsy with associated focal cutaneous involvement, and the first case to occur during a period of prolonged remission.

A 79-year-old man was referred by his otolaryngologist to the dermatology clinic for evaluation of left facial paresis of 1-year duration, initially treated with corticosteroids for presumed Bell's palsy. Despite treatment, the patient's facial weakness worsened and sensation also decreased. Head and neck MRIs revealed no definitive abnormalities along the courses of cranial nerves V and VII. Temporal bone CT scan and chest X-ray were unremarkable. Laboratories including LDH, CRP, ANCA, ANA, Lyme antibodies, VDRL and ACE levels were all negative or normal. Prior to our evaluation, the patient had had a skin biopsy performed on a cutaneous lesion on the left zygoma. Initial histopathologic review was interpreted as granulomatous dermatitis consistent with a ruptured follicle or cyst. Outside medical records documented the patient had been diagnosed seven years prior with stage IIA diffuse large B-cell lymphoma, initially manifesting as a paratracheal mass and treated with six cycles of R-CHOP chemotherapy with apparent remission.

Physical exam was notable for significant left-sided lower eyelid ectropion, incomplete eyelid closure, incomplete closure of the left side of the mouth, hypoesthesia of the left face, as well as an ill-defined, skin-coloured to pearly pink, depressed papule on the left lateral cheek. No submandibular or cervical lymphadenopathy was appreciated. Skin biopsy of the left cheek papule exhibited histopathology consistent with squamous cell carcinoma *in situ* (SCCIS), and an occult neurotropic invasive squamous cell carcinoma was considered.

Upon secondary histopathologic review of the outside skin biopsy of the lesion from the left zygoma, sections showed a diffuse, dense, sheet-like atypical lymphoid infiltrate occupying the dermis with periadnexal predilection (Fig. 1a,b). The atypical lymphoid infiltrate was largely comprised by pleomorphic, large lymphocytes featuring an open chromatin pattern and