

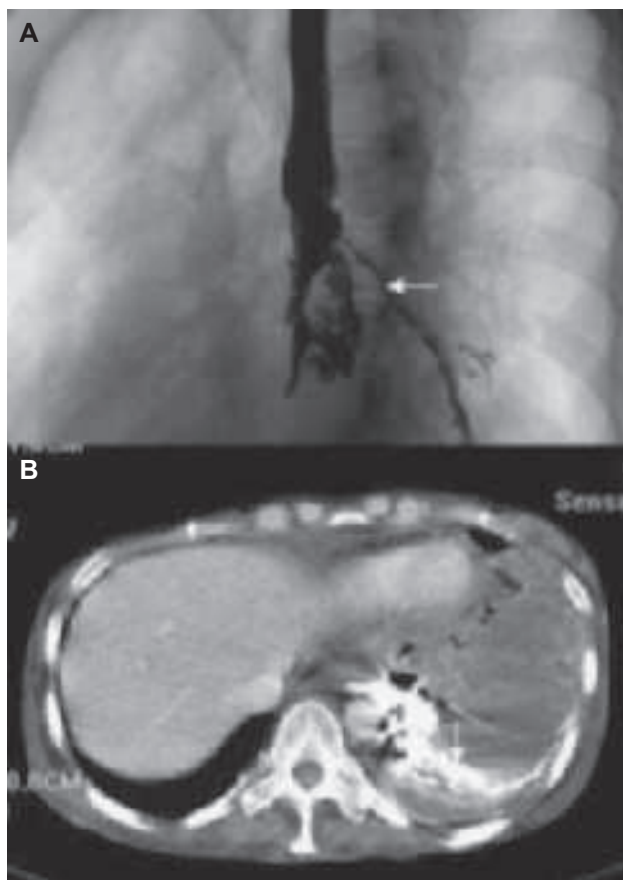


Figure 1. A) A barium swallow study revealed that barium leaked into the left main bronchus (white arrow), suggesting the presence of a tracheoesophageal fistula. **B)** Thoracic computed tomography disclosed left lung infection, left pleural effusion and air inclusions, left atelectasis, and barium in esophagus and left pleural cavity (white arrow), indicating an esophagus rupture.

were always co-administrated with steroids in our patient. Therefore, TEF was not simply considered as a complication of steroids. The diagnosis of TEF in our patient was particularly difficult. She ate almost nothing, which could partially explain why respiratory symptoms were absent. Meanwhile use of corticosteroids may contribute to a delay in diagnosis. Her conditions went rapidly downhill after the barium swallow study. Therefore, when causes for dysphagia are not clear, physicians should always keep a high index of suspicion for TEF, and be especially cautious when choosing barium swallow studies. ■

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Is there a link between chronic urticaria and atopy?

We read with great interest the report entitled "Is chronic urticaria an atopic condition?" by A Nassif, suggesting that chronic urticaria (CU) is part of the atopic diathesis since > 90% of CU patients examined by the author had a personal or familial history of atopic diseases [1].

We have just carried out a prospective study in 42 CU patients that does not support the hypothesis that CU affects mainly atopic patients. The prevalence of atopy in our CU patients was defined by two criteria: 1) personal and/or familial history of atopic diseases including, as in Nassif's study, asthma and/or eczema and/or allergic rhinitis/conjunctivitis; 2) sensitization to at least one common allergen, confirmed by positive skin prick tests. Indeed, atopic status is a propensity to develop immunoglobulin E antibodies in response to natural exposure to environmental allergens, as assessed by skin prick tests [2]. With this definition, the prevalence of atopy has been estimated as between 20 and 32% in Western European countries [3-5].

Results show that 57% of the CU patients had a familial history of atopic diseases and that only 43% were personally affected by these conditions. Altogether, 74% had such personal and/or familial medical histories. These results are lower than Aude Nassif's (82%, 70.5% and 96.5% respectively). More importantly, when the atopic status of the CU patients was defined by the positivity of prick tests to at least one of 13 pneumallergens and 16 trophallergens, the figures were much lower. Indeed, only 29.5% of the 34 patients who were prick tested had at least one positive prick test, while 23.5% had more than one positive test.

In conclusion, this study does not support the hypothesis that CU affects mostly atopic patients. In this respect, CU has been associated to autoimmunity [6]. Future multicentric studies investigating a higher number of patients will give more precise data on the respective roles of atopy and autoimmunity in the development of CU. ■

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Acute onset disseminated superficial porokeratosis heralding diffuse large B-cell lymphoma

We report the case of a 45-year-old Caucasian woman affected by a sudden and widespread eruption of papular lesions with a hyperkeratotic border (*figure 1A*). The lesions were distributed on the trunk, upper and lower extremities. The patient complained of pruritus [1]. Clinical, dermatoscopic and histological features supported the diagnosis of disseminated superficial porokeratosis (DSP). The dermatoscopic evaluation disclosed the presence of a white scar-like area, surrounded by a round brown rim, mimicking the outlines of a volcanic crater (*figure 1B*) [2]. Microscopic examination of the punch biopsy revealed a thin epidermis with the presence of distinctive cornoid lamellae and a discrete perivascular lymphohistiocytic infiltrate in the dermis (*figure 1C*) [1, 2]. Immunohistochemistry revealed an increase of p53 expression in keratinocytes of the basal layer under the cornoid lamella (not shown) [3].

The patient denied excessive sun exposure in the past and there was no family history of similar skin lesions. One month after the diagnosis, the patient manifested chest symptoms indicative of a superior vena cava syndrome. A biopsy was performed, resulting in a diagnosis of mediastinal diffuse large B-cell lymphoma (DLBCL) (*figure 1D*) with an immunohistochemical profile of CD20+, CD30+, CD10-, bcl-2+, bcl-6+, Ki-67+. Epstein-Barr virus IgG serum antibodies were positive, while hepatitis C virus RNA resulted negative. The patient received six cycles

of cyclophosphamide, doxorubicin, vincristine and prednisone chemotherapy associated with rituximab, followed by mediastinal radiotherapy. After two years, the lymphoma was dramatically reduced, as documented by positron emission tomography/computed tomography. The overall extent of the cutaneous lesions and also the itching improved in parallel.

To our knowledge, this is the first report of DSP in association with DLBCL, as a pre-neoplastic disease. The close temporal relationship of these manifestations seems to support a common pathogenetic link, as reported for other forms of premalignant porokeratosis, e.g. punctate porokeratotic keratoderma [4].

In fact, the aetiopathogenesis of DSP is multifactorial, comprising both genetic and exogenous triggering factors, such as ultraviolet light, trauma, infectious agents or immunosuppressive conditions [1]. In the literature, the development of DSP is reported after organ transplants [2] and in patients affected by various hematological disorders such as Hodgkin's disease [5], myelodysplastic syndrome [5], multiple myeloma [5], leukemia [2] and, more recently, non-Hodgkin's lymphoma [6]. In our patient, DSP appeared four weeks before the diagnosis of DLBCL and then ran parallel with the neoplasm. DLBCL, a subentity within the group of B-lineage non-Hodgkin's lymphoma in the World Health Organization classification, is a high-grade malignancy accounting for 30-40% of all adult non-Hodgkin's lymphomas. The etiology of this lymphoma is not entirely clear, but recent studies have described an important pathogenetic link between p53, a tumor-suppressor gene, and the bcl-6 gene, which controls B-cell proliferation and differentiation. Bcl-6 activation inhibits p53 transcription, leading to a disruption of the p53 pathway and to the development of bcl-6 expressing B-cell lymphoma [7], as observed in our patient. As reported by Arranz-Salas [3], the p53 partial function may induce others malignancies, such as premalignant cutaneous lesions and skin tumors. Recently, immunohistochemical analyses have revealed an over-expression of p53 protein in up to 40% of the porokeratosis cases studied [8]. The growth of a clone of epidermal cells bearing p53 alterations, located at the base of the parakeratotic column, could contribute to the development of this precancerous condition. Indeed, p53 overexpression was detected also in our patient.

Alexis and colleagues also recently described a patient with a relapse of non-Hodgkin's lymphoma associated with the sudden appearance of porokeratosis, suggesting the hypothesis of this cutaneous disorder as a paraneoplastic disease in course of non-Hodgkin's lymphoma [6]. In our case, the close temporal link between DLBCL and DSP, taken together with the immunohistochemical analyses, led us to speculate that the tumor-suppressor gene p53 could be involved in disease pathogenesis. ■

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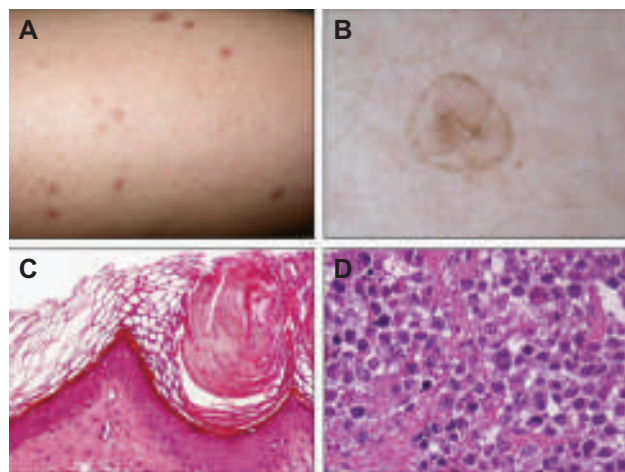


Figure 1. Disseminated superficial porokeratosis: clinical features (A), dermatoscopic aspect (B); histopathology (C, H&E 250 $\times$). Diffuse large B-cell lymphoma: histopathology (D, H&E 400 $\times$).

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