

Extravasation can occur accidentally. The symptoms such as pain and redness develop immediately after the injection. Normally, muscle tissue remains intact. Treatment modalities have been suggested to limit the skin damage caused by hydroxyzine. Hot pack application and topical steroids are recommended. In patients with deep and large ulcers, debridement and plastic surgery such as skin flaps and grafts might be required.

In conclusion, hydroxyzine is widely used; however, it may cause severe tissue damage.

Therefore, the use of hydroxyzine should be restricted. Namely, it should be used only when other sedating drugs or antihistamines are not available or are contra-indicated. In such cases, we should avoid intradermal and/or subcutaneous injections, as well as avoiding strongly massaging the injection site. ■

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Atopic dermatitis with CD30-positive anaplastic large cell lymphoma

The association between long-term severe atopic dermatitis (AD) and lymphoma is a matter of debate. However, a substantial number of cases of severe AD with CD30+ anaplastic large cell lymphoma (ALCL) have been reported. We present the case of a 39-year-old female patient with long-term AD whose ALCL became systemic and nearly took a fatal course. She had been diagnosed with AD in early childhood and had been suffering from it ever since. Her disease severity had been mild and she was treated with topical steroids and oral antihistamines without any additional therapies such as oral steroids, cyclosporine, topical calcineurin inhibitors or phototherapy. From age 32, she

had repeatedly developed multiple small nodules on her scalp, face, trunk, and extremities. The nodular lesions ulcerated easily and became infected with methicillin-resistant *Staphylococcus aureus* but healed spontaneously. At age 36 years, the patient developed a huge ulcerated mass in her oral cavity. She attended a cancer center with the suspicion of malignancy but the mass disappeared spontaneously. The pathological findings of the mucosal lesion showed no atypical cells but the skin lesion contained atypical large lymphoid cells with mitotic figures and she was diagnosed as lymphomatoid papulosis. At age 39 years, she developed a reddish nodule on her right cheek. It rapidly expanded to form an ulcerative mass 10 cm in diameter (figure 1A); there was extensive cervical and subclavicular lymphadenopathy and disseminated cutaneous small nodules. On histology, large atypical lymphocytes with cerebriform nuclei formed nests in the upper to mid-dermis (figures 1B-C). These large anaplastic cells were positive for CD30 (figure 1D), CD3, CD4, and CD8 (CD4>CD8) and negative for CD20, CD79a, ALK, EMA, TIA-1, granzyme B, EBER, and p53. Cervical lymph node biopsy gave similar histological and immunophenotypical findings, with large atypical lymphocytes. Monoclonal rearrangement of the T-cell-receptor γ -chain was identified in the CD4+ CD30+ subpopulations in the tumor, leading to a diagnosis of CD30+ ALCL. A computed tomographic scan of the chest, abdomen, and pelvis did not show any other visceral abnormalities except for bilateral neck and axillary lymphadenopathy. Because the growth of cervical lymph nodes was aggressive, the patient received 2 cycles of CHOP (cyclophosphamide, hydroxydaunorubicin, oncovin, prednisolone) chemotherapy followed by radiotherapy. However, multiple hepatic metastases were found. Additional ESHAP (etoposide, solumedrol, high-dose cytosine arabinoside, cisplatin) and modified DeVIC (dexamethasone, mitoxantrone, ifosfamide, carboplatin) chemotherapies were performed without any benefit, and massive paraaortic and intraperitoneal adenopathy developed. The patient finally received an allogenic peripheral blood stem cell transplant, which brought her into complete remission.

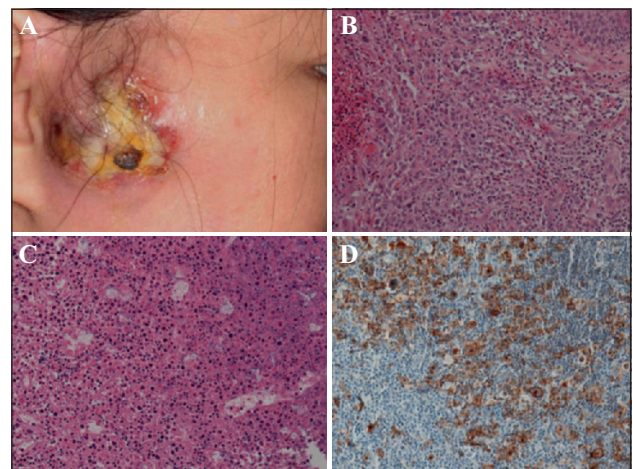


Figure 1. A) Clinical appearance of huge tumor on right cheek at age 39. B) The pathological finding of a skin lesion at age 36. C) High-power view of a biopsied specimen from the tumor (B), showing medium-sized to large atypical lymphocytes. D) These cells were CD30 positive.

We experienced a case with chronic AD who developed lymphomatoid papulosis which turned into a systemic lymphoma. The relationship between AD and lymphoma is still a matter of debate. However, so far, 10 cases of AD patients developing CD30+ primary cutaneous ALCL have been reported [1-3, 5-7]. The average age of these patients (male/female: 8/2) was 38.5 years. Seven of the 10 patients had been treated with systemic steroids or other immunosuppressive medications, including oral cyclosporine, or with phototherapy. One of them died in spite of radiation and chemotherapies [2]. Two other patients received chemotherapies and bone marrow or blood stem cell transplants [2]. Cyclosporine arguably increases the risk of developing lymphoma [1, 3-6] and ALCL occurrence has been reported in a psoriatic patient treated with cyclosporine [8]. Phototherapy with UV-B or PUVA is suggested to cause p53 mutations leading to lymphoma [2]. Of note, three patients, including ours, had no past history of immunosuppressive therapy. Considering that CD 30+ lymphocytes are harbored in skin lesions in AD [4] and that patients with AD are susceptible to impetigo and staphylococcal infections from early childhood [9], it can be hypothesized that persistent (super)antigenic stimulation of T cells may trigger neoplastic transformation [2, 6]. Finally, it should be mentioned that the association of CD30+ ALCL with AD may be fortuitous; indeed, CD30+ ALCL is a rather rare condition but AD is one of the commonest skin diseases. ■

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Mean platelet volume variation after biologic therapy in psoriasis and psoriatic arthritis

Psoriasis (Pso) is a chronic inflammatory, immune-mediated skin disease affecting 2% of the general population [1]. Pso patients may also present with psoriatic arthritis (PsA). Pso has been suggested as an independent risk factor for metabolic syndrome, diabetes mellitus, deep venous thrombosis, coronary artery disease and acute myocardial infarction [2]. Platelet activation may be a link in the pathophysiology of diseases prone to thrombosis and inflammation [3]. Mean platelet volume (MPV) is an indicator of platelet function and activation, which is a determinant feature of atherosclerosis [4]. Elevation of MPV values has been shown to be an independent risk factor in acute myocardial infarction, hyperlipidemia and diabetes mellitus [5, 6]. However, the association between higher MPV value and markers of systemic inflammation and disease activity in psoriasis are controversial.

Some authors [7, 8] have shown that, compared to healthy subjects, MPV values are significantly higher in patients with Pso and PsA, and positively correlated with PASI score [8]. More recently, however, Saleh *et al.* found no differences in MPV values between PsO patients and controls [9].

Ours is the first report analyzing MPV changes after anti TNF α therapy in a psoriatic population.

Clinical characteristics and blood values from 59 patients (27 with PsA and 32 with Pso) without conditions that could alter platelet activation (such as known cardiac diseases and cardiovascular risk factors such as smoking and obesity) were collected before and after 6 months of anti-TNF therapy. Mean age of patients was 45.7 ± 12.2 years. Diagnosis of PsA was assessed according to the CASPAR criteria. Severity of psoriasis was assessed using the Psoriasis Area and Severity Index (PASI) score. Patients started biologic therapy accordingly to standard schedules after an adequate washout period of at least 2 weeks from the previous systemic treatment: about 40% were treated with adalimumab; 50% with etanercept and 10% with infliximab. The control group consisted of 20 healthy sex- and age-matched subjects without cardiovascular risk factors. Blood samples were obtained after a fasting period of 12 h,