effects on the cardiovascular system and blood lipids. To our knowledge, only two cases of anastrozole-related Henoch Schönlein purpura (HSP) have been published so far in the literature [2, 3]. We report of a third case in a postmenopausal estrogen receptor-positive woman with early breast cancer.

A 55-year-old postmenopausal woman was referred to our department in June 2006 after undergoing a right breast lumpectomy and ipsilateral axillary node dissection for a 1.3 cm, HER-2 negative, node positive and hormone receptor-positive breast cancer. Postoperatively, she received four cycles of adjuvant chemotherapy followed by radiotherapy to the right breast and endocrine therapy with anastrozole. In July 2007 she presented with an episode of gross hematuria associated with abdominal pain followed, two days later, by the occurrence of several small palpable and burning purpuric lesions in both legs (figure 1A). The patient was then admitted to the hospital where a CBC, a serum chemistry panel including antinuclear antibody, hepatitis markers, cryoglobulin, p- and c-ANCA and coagulation studies were all within the normal range. A punch biopsy of the skin showed infiltration of dermal vessels by neutrophils, red blood cell extravasation and fibrinoid necrosis, consistent with a leucocytoclastic vasculitis (figure 1B). The immunofluorescent staining revealed IgA deposits in the dermal vessels suggestive of HSP. The cutaneous lesions resolved spontaneously about 2 weeks after anastrozole was withdrawn and the patient was then restarted on endocrine treatment with tamoxifen. Her subsequent skin examinations have been unremarkable.

Henoch-Schönlein purpura is an acute small vessel leucocytoclastic vasculitis which is prevalent in young children [4]. Adults may also be affected and often present with a more severe clinical picture. Although the cause is still controversial, IgA1 deposition in vessel walls and renal mesangium are responsible for the major clinical manifestations. Typically, patients present with a palpable purpuric rash, usually concentrated on the buttocks and lower extremities [5]. Painful arthritis, most often affecting the ankles and knees, may precede the onset of purpura in about one third of the cases. Gastrointestinal, renal, peripheral and central nervous system involvement may sometimes occur and be responsible for a delay in diagnosis. HSP has been linked to infectious agents as well as environmental factors, malignancy and various pharmacological agents. Tamoxifen, a selective estrogen receptor modulator (SERM), has also been reported to induce vasculitis but the underlying biological mechanism is still unclear. Paraneoplastic vasculitis always needs to be ruled out in a patient presenting with palpable purpura and history of cancer, though it usually parallels the course of the underlying malignancy [6]. In our case, somewhat similarly to the previous reports, prompt resolution of the skin changes after discontinuation of anastrozole and without a specific treatment with steroids or immunosuppressive agents, led us to exclude a paraneoplastic form.

In conclusion, anastrozole-related HSP may be a potential, though rare and completely reversible short-term adverse effect of aromatase inhibitor treatments and, as such, it needs to be recognized if a cutaneous vasculitis develops in patients on these medications.

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Lymphomatoid papulosis after allogenic stem cell transplantation

Post-transplantation lymphoproliferative disorders (PTLD) are relatively common lymphoid proliferation or lymphomas that develop in a recipient of a solid organ or bone marrow allograft. However, patients undergoing allogeneic stem cell transplantation have a PTLD incidence of about 1%. Moreover most examples are of B cell origin, and CD30+ T cell PTLDs are very rare [1]. We report a case of lymphomatoid papulosis (LyP) in an immunosuppressed patient who had received an allogenic stem cell transplantation. To our knowledge, only a few cases of posttransplantation LyP have been reported [2-4]. Our patient was a 49-year-old woman with a history of aplastic anemia who had received an allogenic stem cell transplantation six months before her visit to our clinic. The patient had received cyclosporine 75 mg for GVHD

Figure 1. A) single and confluent purpuric lesions on the anterior aspect of patient’s lower leg; B) skin biopsy specimen of the purpuric lesion showing leucocytoclastic vasculitis with neutrophil infiltration, red blood cell extravasation and fibrinoid necrosis.
prophylaxis after stem cell transplantation for about six months. About one month before, the medication was changed to cyclosporine 50 mg with prednisolone 10 mg, because of elevated liver enzyme levels. She had prophylactic antiviral treatment just before and after she had stem cell transplantation. The patient had no history of previous malignancy. She presented with multiple zosteriform erythematous papules on her left arm, which had been about one month previously (figure 1A). Because of her clinical presentation and immunosuppression state, we initially suspected herpes zoster; however, the lesions were not improved with sufficient oral antiviral treatment. We performed a biopsy and the specimen showed a wedge shaped perivascular lymphoid infiltrate with cytologic atypia (figure 1C). In the immunohistochemical study, the atypical lymphocytes expressed CD30 and CD3 antigens, but not CD20, CD34, CD68, and myeloperoxidase (figure 1D). Immunohistochemical staining to herpes simplex virus (HSV) and varicella-zoster virus were all negative. Polymerase chain reaction analysis for HSV DNA was also negative. Serologic tests for human immunodeficiency virus types 1 and 2, Epstein-Barr virus (EBV), and hepatitis viruses were all negative. The histopathological and immunophenotypical findings established the diagnosis of LyP. She was treated with topical steroids without discontinuation of the immunosuppressive agent and the cutaneous lesion showed significant improvement after four weeks. After six months, she had no recurrence, but showed post-inflammatory hyperpigmentation (figure 1B).

The pathogenesis of PTLD is complex and probably multifactorial. Drug-induced immunodeficiency and chronic antigenic stimulation exerted by the recipient’s tissue play an important role. Other risk factors include the type of transplanted organ, the recipient and donor EBV serological status, the type of disease leading to transplantation, and the type, length, and intensity of immunosuppressive drug treatments [1]. The incidence of PTLDs varies from 1% to 11% in solid organ transplant recipients. In bone marrow transplanted patients, the PTLD incidence is lower than 1% [1]. It is possible that patients with organ transplants as opposed to bone marrow transplants simply require prolonged immunosuppressive treatment and that this is a possible cause of an increasing risk of PTLD. Recently, the possibility of preventing transplant rejection has been radically improved by the introduction of new immunosuppressive drugs such as cyclosporine, tacrolimus, muromonab, and mycophenolate mofetil [1]. Ciancio et al. [5] investigated PTLD incidence in transplant recipients treated with different types of immunosuppressive regimens over 18 years and observed a greater prevalence of PTLDs in patients treated with new immunosuppressive drugs than in those treated only with corticosteroids. There are several CD 30+ lymphoproliferative disorder cases who received cyclosporine therapy.

[6] Therefore, in our case, the immunocompromised status and cyclosporine treatment itself could be possible causes of PTLD.

In conclusion, we present a rare case of a PTLD presenting as LyP in an allogenic stem cell transplantation patient. Dermatologists should consider LyP in the differential diagnosis of cutaneous neoplastic and infectious conditions that arise in immunosuppressed patients after transplantation.

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Skin infection masquerading as lupus erythematosus profundus

A 55-year-old woman was admitted to our department with low grade fever and an erytho-violaceous, sharply demarcated plaque on her lower limb of 8 weeks’ duration. Firstly, it had been diagnosed as cellulitis and she received antibiotic treatment ampicillin/clavulanic acid, 625 mg per os tid for 7 days, without any improvement. Her medical history was unremarkable for other illnesses and medications. On admission she was febrile (37.5 °C) and she could not relate her cutaneous lesion with any specific presumptive cause (animal, insect bite or trauma).