Purpuric Pityriasis Rosea Associated with Acute Myeloid Leukemia: Case Report

Akut Myeloid Lösemiyle İlişkili Purpurik Pitriyazis Rozea

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ABSTRACT Pitriazis rosea (PR) may present in atypical clinical forms. Purpuric pityriazis rosea (PPR) is characterized by pruritic annular patches. The course of the disease is same as the typical PR. The histopatologic hallmark of the disease is erythrocyte extravasation. Few cases have been reported and none of them had no systemic associations.We have been reporting PPR associated with acute myeloid leukemia with abnormal clotting parameters. The disease itself or clotting abnormalities are causing to rise the purpuric nature of the disease. Diagnose of the PPR is based on clinical and histopathological findings.

Key Words: Pityriasis rosea; leukemia, myeloid

ÖZET Pitriyazis rozea atipik klinik formlarda görülebilir. Purpurik pitriyazis rozea; kaşıntılı, annüler plaklarla karakterizedir. Hastalığın seyri tipik pitriyazis rozea gibidir. Histopatolojik bulgusu eritrosit ekstravazasyonudur. Sınırlı sayıda olgu bildirilmiştir ve hiçbirinde sistemik hastlalık ilişkisi yoktur. Burada akut myeloid lösemiyle ilişkili purpurik pitriyazis rozea olgusu sunulmaktadır. Sistemik hastalığın kendisi veya anormal kan parametreleri hastalığın purpurik görünümüne neden olmaktadır. Purpurik pitriyazis rozeanın tanısı klinik ve histopatolojik bulgulara dayanmaktadır.

Anahtar Kelimeler: Pityriasis rosea; leukemia, myeloid

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Pityriasis rosea (PR) is a relatively common disease with self limited course. It occurs worldwide and racial suspectibility has not been documented. It usually affects teenagers and 10-35 year old young adults. Classically a herald patch pre-dates the eruption characteried by smaller erythematous ovoid patches with collaret like scaling. These lesions locate on the trunk in Christmas tree patern,in which their long axes follow Langer'slines of clevage. It has been reported that clinical distrubution and morphological features of PR may vary and diverse atypical variants.[1] PPR is one of these atypical forms and only 13 cases have been reported. Although exact etiology of PR is unknown, there is some evidence related viral infection. Mildly increased prevalence in patients with decreased immunity, such as bone marrow transplant recipients, support this theory. On the other hand, certain drugs, psychogenic factors and stress having a dep-

ressant effect on the immune system,has also been considered as etiologic factors.[1] Atypical forms of PR associated with hematological malignancies have rarely been reported.[2] Herein we report a case of PPR in a patient with acute myeloid leukemia (AML).

CASE REPORT

A 30 year-old female previously diagnosed as acute myeloid leukemia (AML M2) in May 2005, hospitalized for the 4th chemotherapy in January 2006. Chemotherapy regimen was including idarubicin plus cytosine arabinoside. She was also on antibiotic therapy including amicacin, vancomycin, tazocin and amphotericin B for the neutropenic fever and allopurinol for possible hyperurisemia. On the last day of the chemotherapy, she noticed an annular lesion on her breast, thereafter new and smaller ones started to occur on her chest and groin and then spread to trunk and arms. On dermatologic examination, therewere ovoid-annular and erythematous patches, measuring 1-5 cm in diameter, located on the breast and upper trunk. There were barely visible collarette like scaling and purpuric, cayenne pepper like spots on the annular magrin of the lesions. The one located on the breast was the largest and preceded the others (Figure 1 and Figure 2). Oral mucosal petechia was not detected. There were no prodromal or subjective symptoms. Four milimeter skin biopsy obtained from the herald patch revealed compact hyperkeratosis, epidermal spongiosis, superficial perivascular lymphohistiocytic infiltrate and erythrocyte extravasation (Figure 3). Laboratory examination revealed some abnormalities compatible with her primary disease and medication. White blood cell count was 131 x 10^3 mm^3, with 30% lymphocyte count and platelets were 27x10^3. There were 90% blastic lymphocytes in peripheralblood smear. Protrombin time, partial thromboplastin time and INR (International normaliation ratio) were higher than normal limits. Bone marrow aspiration revealed; 42% lymphocyte, 31% monocyte, 26% graulocyte and reduced amount of megacaryocytes. Diagnosis of PPR is based on clinical and histopathological findings. In a one week period, lesions cleared spontaneously without any treatment.

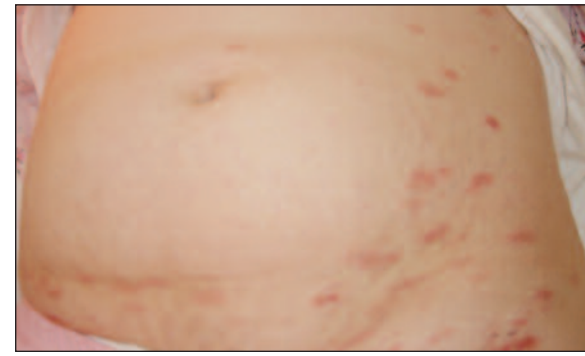

FIGURE 1

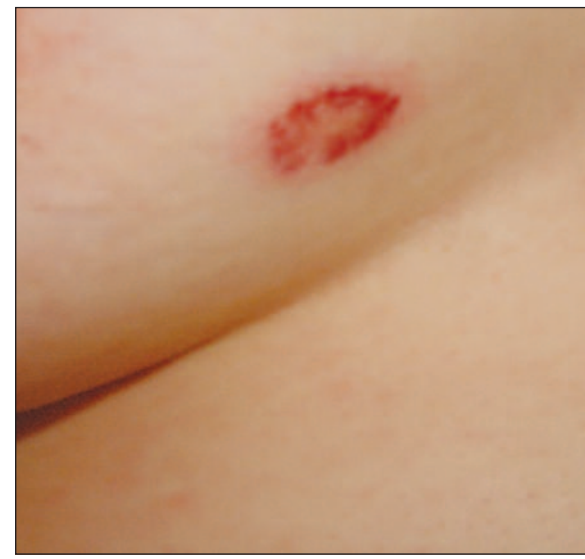

FIGURE 2

FIGURE 1, 2: Annular and purpuric patchs located on the breast and trunk.

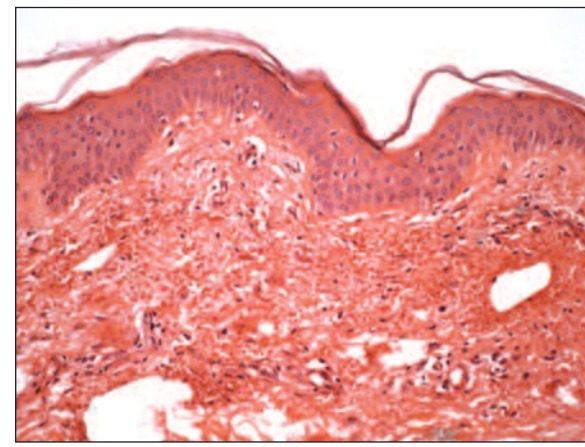

FIGURE 3: Epidermal spongiosis, superficial perivascular lymphohistiocytic infiltrate and extravasated erythrocytes (HE, x 200) .

DISCUSSION

Atypical variants of PR are rare and occur in only 20% of all cases. Atypical presentation can be ob-

served in its morphology, size, site, dispersion, location, number of lesions, severity and in the course of the disease. Morphologically vesicular-pustular, purpuric or hemoragic and urticarial-papular forms might be observed.[3] PPR was first described by Hartman presenting with hemorrhagic herald patch and smaller purpuric lesions.[4] Hemorrhagic and PPR are different terminologies describing the same condition. We could find the reports of 13 cases in the literature, clinical characteristics of these cases are summarized in Table 1. All of the reported cases of PPR had similar clinical and histopathological morphology. It usually presents with macular purpuric lesions located on the trunk and extremities. Although palatal petechia has reported to accompany cutaneous lesions, none of the reported cases had mucosal lesions. None of them had systemic symptoms neither. Clinical course of PPR is that of typical PR. The typical histological findings, shared by all of the reported cases, were focal parakeratosis, spongiosis, and superficial lymphocytic perivascular infiltrate and erythrocyte extravasation without vasculitis.[1,5-10] Specific and nonspecific cutaneous lesions might be associated with hematologic malignanicies. These lesions may precede, occur concomitantly or develop after the diagnosis of malignancies. Association of atypical form of PR with hematologic malignancies have rarely been reported. Garcia-F-Villalta et al. reported a case of eczematous PR in a patient with systemic symptoms and lymphadenopathies,subsequently diagnosed as Hodgkin's

TABLE 1: Summary of reported cases of PPR.

Author/yea r	Age/sex	Location	Mucosal examination	Laboratory	Prognosis	Treatment
*Hartman M, 1944	?	?	?	?	?	?
*Rinaldi VG, 1954	?	?	?	?	?	?
Verbov J, 1980	20/F	Anterior trunk	NA	N	Resolution in 6 weeks	-
	23/M	Trunk/lower extremities	NA	N	Resolution 4 weeks	-
	10/M	Anterior trunk/ upper extremities	NA	NA	Resolution in 18 days	-
Paller A, 1982	7/M	Upper anterior tighs, buttocks, groin, axilla	NA	NA	Resolution 8 weeks	Topical hydrocortisone tid
	4,5/ F	Buttocks, trunk axilla, Lower extremities	NA	N	NA	Topical hydrocortisone
	10/ F	Posterior thigh	NA	N	Complete clearance in 4 week	Emolients
	25/F	Right breast, lower abdomen, proximal thighs	NA	N	Complete resolution in 8 weeks	-
Pearson J, 1993	11/F	Neck, trunk, proximal extremities	NA	N	Resolution in 6 week	-
Sezer E, 2003	17/M	Trunk and arms	Normal	N	Spontaneous resolution in 4 weeks	-
Chuh A, 2005	33/M	Trunk, extremities	Normal	N	Complete resolution 4 weeks	Topical calamine lotion, antihistamines
Aliagaoglu C, 2006	16/F	Trunk, upper arms, neck	Normal	N	Spontaneous resolution in 4 weeksSummary of reported cases of PPR	-
Present case	30/F	Upper trunk	Normal	Clotting abnormalities	Resolution in ten days	-

*Full texts of these articles were not available.

N: Normal

NA: Not available.

disease.[2] They proposed that until the cause of PR has been established, an explanation for the relationship between HD and PR remains speculative. Brazzelli et al. reported three patients with chronic myeloid leukemia who were all on Imatinib mesylate (IM) therapy .[11] These patients have characteristic clinically erythematous, slightly pruritic and macular skin eruption. Lesions had a peripheral collarette of desquamation with parallel dispersion to Langer's skin lines and located on the trunk limbs, and arms suggesting the diagnosis of PR. But on the base of histopathological findings, strong correlation between the time course of the reaction and the administration of IM, and inducing of the lesions by re-challenge of the IM led them have further evidence of the link between the two events. There are drug eruptions mimicking PR. Angiotensin converting enzyme inhibitors alone or in combination with hydrochlorothiazide, allopurinol, nimesulide, acetyl salicylic acid have been reported as causative agents.[12] Although our patient was on allopurinol therapy, characteristic findings of drug induced PR such as absence of herald patch, bright violet-red color of the lesions, severity of itching and lack of response to antihistamines, presence of eosinophils in the blood and in the skin were not observed. For the current case, disappearing of the lesions while on allopurinol treatment, and the presence of dusky-red color of the lesions with purpura, lack of pruritus, skin and tissue eosinophilia helped us to rule out the etiologic role of drugs. Our case is different from the previously reported ones in terms of associated hematologic malignancy and clotting abnormalities. All of the reported cases of PPR had normal clotting parameters and patients were otherwise healthy. For the present case altered immunologic reactivity, caused by the disease itself or medications may be responsible for atypical presentation of the disease. On the other hand, the role of abnormalities in clotting parameters can not be ruled out. Although atypical presentations of PR associated with hematologic malignancies have been rarely reported, we could not able to find any report of PPR with hematologic malignancy. In conclusion, to be aware of wide spectrum of PR is important for clinicians. Atypical forms may be associated with certain malignancies. Histopathologic examination and clinical clues may help to establish definitive diagnosis.

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