Prurigo pigmentosa: A Case with Nickel Sensitivity

Prurigo Pigmentoza: Nikel Duyarlılığı Olan Bir Olgu

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ABSTRACT Prurigo pigmentosa is a distinctive inflammatory dermatosis characterized by recurrent, pruritic erythematous papules and reticulate hyperpigmentation. The eruption typically shows a symmetrical distribution on the trunk and tends to localize mostly on the neck, upper back, clavicular region and chest. The pathogenesis of prurigo pigmentosa is still unknown however it has been suggested that ketosis related to diabetes mellitus, dieting or anorexia nevrosa; friction, sensitivity to sunlight, and contact allergy might be a triggering factor. There are some prurigo pigmentosa cases reported to be associated to contact allergens like para-amino compounds, trichlorophenol, chromium and nickel in the literature. We report a Turkish young female prurigo pigmentosa patient with nickel sensitivity and we discuss the role of contact allergens in the etiology of prurigo pigmentosa.

Key Words: Dermatitis, contact; hyperpigmentation; nickel; prurigo

ÖZET Prurigo pigmentoza rekürren, pruritik eritematöz papüller ve retiküler hiperpigmentasyonla karakterize kendine özgü inflamatuar bir dermatozdur. Erüpsiyon tipik olarak gövdede simetrik bir dağılım gösterir ve çoğunlukla boyun, sırtın üst kısmı, klaviküler bölge ve göğüste lokalize olmaya eğilimlidir. Patogenezi halen bilinmemekle beraber diabetes mellitus, diyet ya da anoreksia nevroza ile ilişkili ketoz; friksiyon, güneş hassasiyeti ve kontakt allerjinin tetikleyici birer faktör olabileceği öne sürülmüştür. Literatürde para-amino bileşikleri, triklorofenol, krom ve nikel gibi kontakt allerjenlerle ilişkili olduğu rapor edilmiş bazı prurigo pigmentoza vakaları vardır. Biz burada nikel sensitivitesi olan genç bir kadın prurigo pigmentoza hastasını rapor ediyor ve prurigo pigmentoza etyolojisinde kontakt allerjenlerin rolünü tartışıyoruz.

Anahtar Kelimeler: Dermatit, temas; hiperpigmentasyon; nikel; prurigo

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Prurigo pigmentosa (PP) is an idiopathic inflammatory skin disease characterized by recurrent eruption of pruritic erythematous macules and papules.[1] The lesions appear suddenly and regress in a short time leaving a reticular pigmentation. The eruption typically shows a symmetrical distribution on the trunk and tends to localize mostly on the neck, upper back, clavicular region and chest.[2] The forehead, arms and abdomen are rarely involved locations. Papules of PP tend to recur at the same location.[1] Most of the reported PP cases are of Japanese origin and a signifi-

cantly high number of non-Japanese PP patients are reported from Turkey.[3] Herein we report another Turkish PP patient who is a young female with nickel sensitivity.

CASE REPORT

A 21-year-old female patient presented with a 7-year history of a pruritic eruption, usually recurring in the spring. According to the history of the patient the eruption had started on her arms and was later localized to the trunk. Dermatological examination of the patient revealed erythematous, excoriated papules and plaques on a background of reticular, brown hyperpigmentation localized on the back and extending from epigastric region to the umblicus. Urticarial plaques were observed on the upper extremities and on the areas outside the hyperpigmented patches of the back and abdomen (Figures 1, 2).

There was no history of diabetes, photosensitivity, eating disorder or strict diet. The patient had a contact dermatitis history with metal accessories. A patch test was performed with the European Standard test series and a vesicular-pustular reaction was observed for nickel.

A skin biopsy from an erythematous plaque on the back was reported as allergic contact dermatitis. The lesions regressed spontaneously then during the follow-up the eruption recurred with intense itching. This time upper extremities were normal but there were additional excoriated papules and plaques on the breasts bilaterally, nape and interscapular region. Histopathological examination of the skin biopsies from nape and back revealed regular acanthosis, hyperpigmentation, lymphocyte exocytosis, neutrophilic spongiosis in the epidermis, focal epidermal microabscess formation and focal vacuolar changes in the dermoepidermal junction. Perivascular and interstitial mixed type inflammatory cell infiltrate composed of eosinophils, lymphocytes, neutrophils; and multiple melanophages with melanin pigment were observed in the dermis (Figure 3). The patient was diagnosed as PP with these histopathological findings and typical clinical features.

Doxycyclin treatment of 100 mg/day was commenced. Ten days later her pruritus resolved and after 2 weeks the eruption disappeared. In the sixth week of treatment just a reticular brown pigmen-

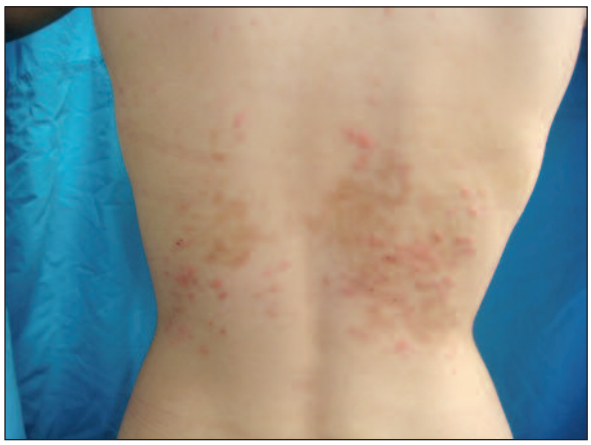

FIGURE 1: Erythematous, excoriated papules and plaques on a background of reticular, brown hyperpigmentation on the back.
(See for colored form http://dermatoloji.turkiyeklinikleri.com/)

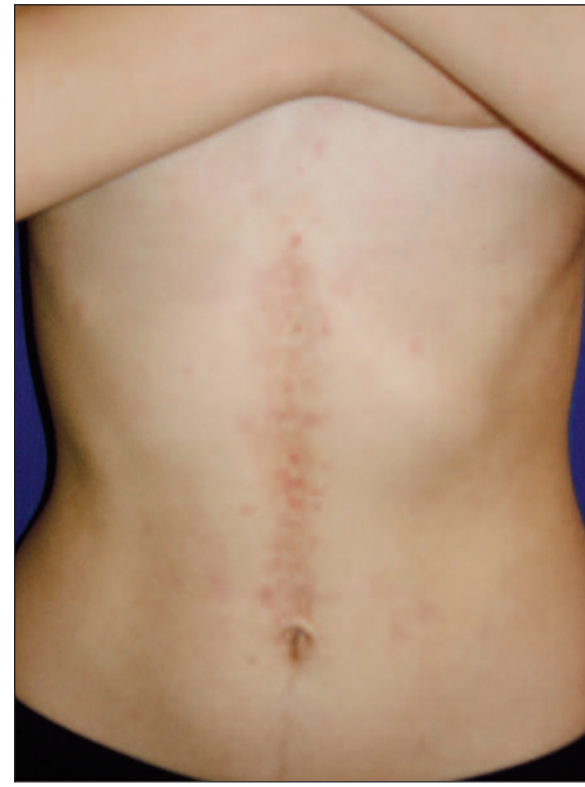

FIGURE 2: A plaque with brown hyperpigmentation and erythematous excoriated papules extending from epigastric region to the umblicus in a linear form.
(See for colored form http://dermatoloji.turkiyeklinikleri.com/)

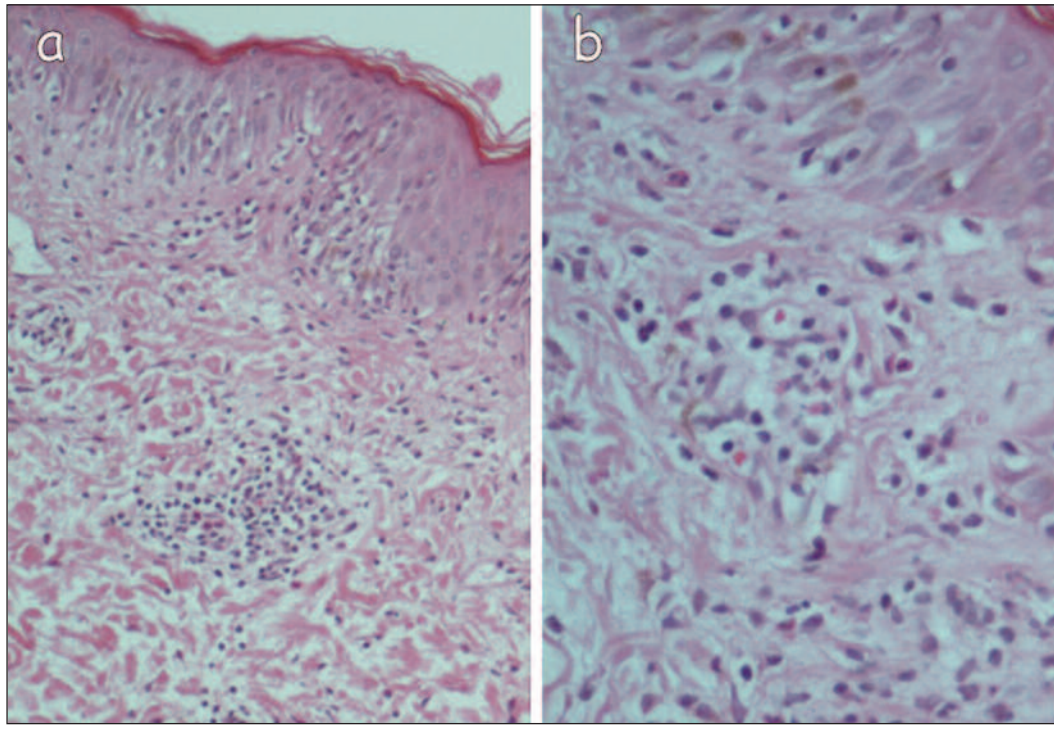

FIGURE 3: a) Epidermal acanthosis, exocytosis of lymphocytes and interface dermatitis (Hematoxylin-eosin stain; original magnification, x10). **b)** Perivascular and interstitial mixed inflammatory cell infiltration composed of lymphocytes, eosinophils and polymorphonuclear leucocytes; melanophages (Hematoxylin-eosin stain; original magnification, x20).
(See for colored form http://dermatoloji.turkiyeklinikleri.com/)

tation was observed. The treatment was completed at the end of the third month. No recurrences occured during 6 months follow-up.

DISCUSSION

PP is a recurrent pruritic eruption of erythematous papules that unite to form a reticulate pattern. Intensely pruritic lesions of sudden onset last approximately a week, then the rash tends to cool down only to recur in full blown fashion again.[2] PP is generally seen among young females in the spring and summer.

The disease shows consistent stage-dependent histopathologic features. Early lesions of PP, like a smooth-surfaced reddish macule or an urticarial papule present with a superficial perivascular infiltrate of neutrophils. Then the neutrophils are scattered in the papillary dermis and enter the slightly spongiotic epidermis including necrotic keratinocytes. In a fully developed lesion such as a crusted red papule, a smooth-surfaced papulovesicle or a vesicle, a patchy lichenoid infiltration with a predominance of eosinophils and lymphocytes appear. Intraepidermal vesiculation due to spongiosis and ballooning or a subepidermal vesiculation as a result of extensive vacuolar alteration at the dermo-epidermal junction may develop. In a late lesion, that is, a pigmented macule, perivascular infiltrate consists of lymphocytes mainly and the epidermis becomes hyperplastic, parakeratotic and slightly hyperpigmented. Melanophages appear in the dermis. Immunofluorescence studies are negative invariably.[4] The histopathological findings of our patient were consistent with the histopathological findings of the fully developed and late lesions of PP.

The pathogenesis of PP remains unknown but some mechanisms have been proposed. Ketosis related to diabetes mellitus, dieting or anorexia nervosa; *Helicobacter pylori* infection, atopic diathesis, pregnancy are the systemic conditions that have been reported to be associated to PP.[3,5-8] Teraki et

al. described a relation between PP and ketosis. They also suggested that previous cases of PP that were accompanied by fasting, dieting, or diabetes mellitus may also be related to ketosis.[5]

Some exogenous factors have also been associated with PP such as physical trauma, friction from clothing and sweating, contact allergy, and sensitivity to sunlight.[1] Stress is another factor that was reported to be associated with PP.[9-11] Lu et al. reported stress as an aggravating factor in one of their patients.[10] Choi et al. reported another PP patient skin lesions of whom were aggravated by emotional stress and clothing-associated mechanical irritation.[11]

An allergic pathogenesis has been proposed as there are PP cases reported to be associated to contact allergens like trichlorophenol, para-amino compounds, chromium and nickel in the literature.[12-16] Contact with trichlorophenol and para-amino compounds used in manifacturing clothing have been implicated by Cotterill et al. and Yamasaki et al., respectively.[12,13] Kim et al. reported a PP case with contact allergy to chrome in detergent left in clothes after washing and Tanii et al. reported a case of PP with an allergic reaction to the chromium component in acupuncture needles.[14,15] In another case bismuth-subsalisilate containing antiacid ingestion was described.[17] Since the eruptions primarily involve a covered area of the body, it was suggested that the contact allergy might be triggered by the friction of clothing especially in the spring and summer seasons when increased sweating occurs.[13,14]

Our patient had a contact dermatitis history with metal accessories and a positive reaction was observed for nickel in the European standard patch test.

The only PP patient reported to have nickel sensitivity in the literature; was also a young Turkish female with a similar clinical presentation, a history of contact dermatitis with metal jewelery and a European standard patch test positive for nickel.[16] No additional endogenous or exogenous factors were documented and nickel sensitization was accepted as a triggering factor or an associated condition by the authors.

Nickel can induce various clinical pictures, of either eczematous or non-eczematous type. Unlike most other allergens, nickel can give rise to a pathognomonic clinical picture, of erythemato-micropapulo-vesicular lesions, that are in fact the most common type of nickel-induced eruption. Dry contact dermatitis of the fingertips of the hands and nummular eczema are also relatively common clinical presentations. Airborne contact dermatitis, dyshidrosiform contact dermatitis, and lichenoid contact dermatitis are the other clinical forms of allergic dermatitis due to contact with nickel.[18] PP may be another presentation of allergic dermatitis due to nickel sensitivity. Even though their association may be coincidental, nickel may be a triggering factor for the development of PP.

Dapsone and minocyclin are the most commonly used therapeutic agents in PP. However doxycyclin is also reported to be effective in PP.[3,6,19,20] Dapsone is known to inhibit release of lysosomal enzymes from neutrophils and tetracyclines inhibit chemotaxis of neutrophils; namely medications that have proven to be helpful to patients with prurigo pigmentosa have capability for inhibiting migration and/or function of neutrophils.[4] Our patient responded very well to treatment with doxycycline.

In conclusion, we suggest that although the relationship is not clear, contact allergy must be investigated as an etiological factor in the evaluation of PP patients.

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