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Dermatology

PITYRIASIS ROSEA WITH A TWIST

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ABSTRACT Pityriasis Rosea(PR) is a common acute, self-limited papulosquamous disorder that favours otherwise healthy adolescents and young adults. A 13 year old boy presented with a cutaneous vesicular eruptions over palms followed by its extention over other parts of trunk and extremities of 2 weeks duration. A provisional diagnosis of extensive pityriasis rosea, pityriasis lichenoid et varioliformis acuta(PLEVA) and drug eruption was made. Skin biopsy revealed features consistent with vesicular pityrisis rosea. Patient was given oral prednisolone which resulted in resolution of lesions. There are a paucity of reports of vesicular pityriasis rosea with palmar involvement.

KEYWORDS : Pityriasis Rosea, PLEVA , Vesicular

INTRODUCTION:

Pityriasis rosea(PR) literally 'rose-colored' scales, was named by French physician Camille Melchoir Gibert in 1860,but recognized first by Willan in 1798.[1] It is an acute inflammatory dermatoses of unknown etiology with a self limited course. A history of herald patch and a few characteristic lesions in a 'Christmas tree' pattern aid the diagnosis of typical PR. It may present with atypical features in around 20% cases[1]. Here we present an unusual presentation of vesicular pityriasis rosea.

CASE HISTORY:

A 13 year old boy presented with a mildly pruritic rash of 2 weeks duration on palms, soles and trunk. According to the mother, the lesions first appeared on palms and soles followed by extension to extremities [Figure1] and other parts of the body within a period of few days. There was no history of associated fever, abdominal or joint pains. On enquiry, patient denied taking any drugs prior to appearance of rash. On examination the palms [Figure2] and soles demonstrated deep seated tense fluid filled vesicle, they were extensive on palms and few on soles. The lesions on trunk [Figure1] were papules and plaque, many of which were topped with vesicles and few showed excoriation and crusting. Oral cavity and other mucosal sites were spared. There was no involvement of face, scalp and nails. A provisional diagnosis of PR, PLEVA, drug eruption was kept. A biopsy was performed from lesion on trunk which showed [Figure4] focal parakeratosis of startum corneum with mild pityriasiform hyperplasia, moderate spongiosis with intraepidermal vesicles. Papillary dermis shows RBC extravasation with moderate lymphohistiocytic infiltrate around superficial vessels. On clinicopathological correlation, a final diagnosis of vesicular pityarisis rosea was reached. A full blood count and blood chemistry profile yielded normal results. The patient was started on prednisolone 30mg. The lesions almost disappeared [Figure3] after 7 days after which the dose of prednisolone was tapered. He is still on follow up in our department and no new lesions have appeared.

DISCUSSION:

In 1798 Robert Willan described “roseola infantum” as a self limited eruption. Camille Melchior Gibert first named word Pityriasis Rosea also called Pityriasis rosea of Gibert[2] which is an acute self-limiting papulosquamous disease reported in all races with an incidence of 6.8 per 1000 dermatological patients. PR may occur in patients of all ages, mainly occurring between the age of 10 and 35years[4]The exact cause of PR is uncertain but Human herpes Virus 6 and 7 have been suggested the most likely cause.[2] In classical PR solitary oval skin colored to erythematous plaque with collaratte of scale appears usually on trunk and less commonly on neck.This solitary patch is called herald patch which was first described by Loius-Anne-Jean Brocq in 1887,it is skin colored to salmon colored annular plaque with a raised advancing edge which measures 2-4cm.Most common sites are chest, back, neck, extremities. The lesions form a Christmas tree or fir tree pattern.

The histopathology of PR was first described by Unna in 1894.[3] Small mounds of angulated parakeratosis(tea pot tlid sign), focal spongiosis, acanthosis and a mild lymphocytic perivascular (coat sleeve) and interstitial papillary dermal infiltrate is seen. Focal interface changes may also be seen. Extravasation of RBCs in papillary dermis and partly in epidermis (Sabourav sign) and absence or decreased granular layer (lowenbach sign) histological signs of PR, though they are not specific for this condition.

The incidence of atypical PR is 20%.The atypicality may be in morphology , size, distribution, course or symptoms. The various atypical morphological types includes vesicular, purpuric, urticarial, generalized papular, lichenoid, erythrodermic and EM-like PR.

Vesicular PR presents as a generalized eruption of 2-6mm vesicles or as a rosette of vesicles. It may be severely pruritic, most commonly seen in children and young people and may affect head, palms and soles, as an initial presentation. In our patient also the lesions first occurred over palms and soles which then spread to involve other parts of body. On histopathology the lesions of vesicular PR usually demonstrate spongiosis with intraepidermal vesicles along with focal basal cell vacuolar degeneration. Papillary dermis shows RBC extravasation with moderate lymphohistiocytic infiltrate around superficial vessels.[5]

On clinical examination, sometimes the lesions of vesicular PR may resemble PLEVA and pityriasiform drug eruption as in our case. The common drugs implicated in pityriasiform drug erution are ACE inhibitors, hydrochlorothiazide, captopril, barbiturates, metronidazole and allopurinol. The lesions usually appear immediately on taking the offending drug and resolve on its withdrawal. Such cases usually do not show a herald patch with subsequent appearance of other lesions. All the lesions appear at the same time. On histopathological evaluation drug induced PR all the features with or without presence of eosinophils in the dermal infiltrate. The possibility of drug reaction was ruled out in our case as there was no history of any drug intake prior to onset of eruptions and absence of eosinophils in the dermal infiltrate.[7]

Pityriasis lichenoids et varioliformis acuta (PLEVA) also referred as Mucha-Habermann disease is an acute variant of pityriasis lichenoides. It is a papular clonal T cell disorder in which crops of spontaneously regressing erythematous to purpuric papules occur which heals with varioliform scars. On histopathological evaluation overlying cornified layer shows parakeratosis, perivascular and dense bandlike predominantly lymphocytic infiltrate in the papillary dermis that extends into reticular dermis in a wedge-shaped pattern. The infiltrate obscures the dermal epidermal junction with pronounced vacuolar alteration of basal layer, marked exocytosis of lymphocytes and erythrocytes, intercellular and extracellular edema leading to variable degree of epidermal necrosis, these findings were not present our patient.[6]

PR is a self-limiting disorder; therefore most patients would require

symptomatic treatment in form of emollients, antihistaminics, and topical steroids to control pruritus. Antibiotics like erythromycin and azithromycin are said to be effective due to their anti-inflammatory and immunomodulatory actions. Antivirals like acyclovir have also been used. In severe form of the disease patients can be given oral steroids for short duration to control progression. Phototherapy has also been used in extensive PR in few cases.

On extensive search of available literature we found fewer case reports of this entity. [Table 1][9][10][11][12][13][14][15][16] Out of them only 3 cases showed palm and sole involvement. Therefore we report this entity for its rarity and atypical presentation.

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