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Case Review

The Clinico-Pathological and Dermascopic Study on Pityriasis Rosea

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Abstract

Background: Pityriasis Rosea is a papulosquamous disorder, more common during autumn and winter with a decreased incidence in summer. Experiments by various workers to determine the cause of the disease have been unsuccessful. Fungus, bacteria, spirochetes, drugs, contact with new garments, psychogenic and neurogenic factors were all implicated in the causation of the disease. The prodromal illness, generalised exanthem accompanied by constitutional reactions, spontaneous resolution and life long immunity, all point towards a viral cause.

Objectives: To determine various morphological patterns and distribution of skin lesions, the histopathology, duration and complication of the disease.

Materials and Method Used: All the patients attending the outpatient Department of Dermatology, Venereology, and Leprology, at SS Hospital attached to SS Institute of Medical Sciences and Research Centre, Davangere for a period of 24 months (October 2018 to October 2020).

Results: Study showed male preponderance and age group of 11-20 years were affected which were mostly the population spending more time outdoors. The lesions were seen more commonly in winter and rainy season measuring 2 - 5 cm and was seen over trunk, abdomen, back and arm with shape of herald patch varying from oval to round shape with peripheral collarette of scales; distributed in Christmas tree pattern which were symmetrical in distribution. Neutrophilia, lymphocytosis, eosinophilia and raised ESR was noted in few patients.

Histopathological examination of the herald patch showed flaky hyperkeratosis, patchy parakeratosis, thinned out granular layer, irregular acanthosis, focal spongiosis, dilated blood vessels and sparse inflammatory infiltrate around blood vessels in upper dermis.

Keywords: Dermascopic Study; Pityriasis Rosea (PR); Skin Lesions

Introduction

Pityriasis Rosea (PR) is a common dermatoses seen in dermatology out- patients department. It is defined as an acute, self-limiting papulo-squamous disorder affecting mainly children and young adults, and characterised by a distinctive skin eruption and minimal constitutional symptoms [1] or a self-limiting disorder

characterised by the development of asymptomatic erythematous scaly macules on the trunk.

The eruption is preceded by a solitary patch termed "herald patch", mainly located on the trunk. Few days later, a secondary eruption appears, with little pink, oval macules, with a grayish peripheral scaling collarette around them [2,3].

The disease has been given many names, such as Erythema annulatum (Rayer), Herpes tonsurans maculosus et squamosus (Hebra), Lichen annulatus serpiginosus (Wilson), Pityriasis circine (Horand), Pityriasis dissemine (Hardy), Pityriasis marginee et circinee (Vidal), Pityriasis rubra aigu dissemine (Bazin) [3], Pseudoxantheme erythemato desquamatif (Besnier), Roseola annulata (Willan), Roseola furfuracea herpetiformis (Behrend) and Roseola squamosa (Nicolas and chapard).

Pityriasis Rosea has been reported in all races, with varying incidence between 0.3 and 3 percent. It is more common between 10 and 35 years of age, with equal sex distribution or a slight female preponderance. The disease is more common during autumn and winter with a decreased incidence in summer. It has been reported to occur among persons in the same intimate environment.

The prodromal illness, generalised exanthem accompanied by constitutional reactions, spontaneous resolution and lifelong immunity, all point towards a viral cause. Extensive research has been carried out in patients with PR with respect to the newly identified Human Herpes Virus 6 and 7.

The results from different studies however are found contradictory. It is thus possible and remains an unproved fact that HHV-6 and 7 may play a role extensively in some patients with PR.

Search for this agent led to evaluation of a number of organisms in this disorder such as cytomegalovirus (CMV), Epstein-Barr virus, parvovirus B19, picornavirus, influenza and parainfluenza viruses, *Legionella* spp., *Mycoplasma* spp., and *Chlamydia* spp. infections; however, evidence exists that PR is not associated with them [4].

Etiology

The evidence to support an infectious aetiology for PR is its distinct clinical course. There is a primary skin lesion followed by a secondary eruption, with complete remission mostly within eight weeks.

This course of the disease is similar to most of the viral infections. Moreover, many patients do not have a second attack, a phenomenon which is commonly seen in many viral diseases [26,27].

Most experiments at transmitting PR to human beings have failed. But, Wile UJ in his study has shown that PR can be transferred using blister fluid or extract of scales of PR patients. He also showed that bacterial cultures from the scales were negative [28].

An electron microscopic study on lesional biopsy of the herald patch reported virus-like spherical particles with size of 70nm in the intercellular spaces and the cytoplasm of Langerhans cells [29]. Virus-like particles in the dyskeratotic keratinocytes were also reported another study [30].

Evidence supporting an infectious aetiology

a) **Concurrent cases:** There have been many case reports of two or more patients with PR in the same family or intimate environment [31].

b) **Associations:** Epidemiological studies reported associations of PR with prodromal history of respiratory tract infections [32], unfavourable social and economic background[15], and contact with patients with PR [33].

c) **Immunology:** Immunohistologic data shows perivascular aggregates of predominantly active CD4 T- helper cells in the superficial dermis. There is also an increase in Langerhans cells, which is an antigen presenting cell suggesting an infectious aetiology for PR [34,35].

d) **Human Herpes virus (HHV) 6 and human Herpes virus 7:** The viral DNA is reported to be present in peripheral blood mononuclear cells in the lesional and unaffected skin of majority (80-100%) of individuals with acute PR. HHV7 is detected slightly more frequently than HHV6, but often both viruses are found. However, its role as a causative agent is yet to be proved [34,35,37,38].

e) **CMV, EBV and Parvovirus:** Most recent studies have proved that there is no association between PR and these viruses [40].

f) **Enterovirus:** Enteroviruses tend to produce a variety of exanthem. A case has been reported with PR like skin eruption with a typical Christmas tree pattern and the demonstration of a monoclonal antibody that identified enteroviruses, sug-

gests that an unusual enterovirus could be the possible cause of the rash [43].

Autoimmunity

An autoimmune element in the pathogenesis of PR has been suspected by some investigators [45]. They proposed that PR is an autoaggressive disease affecting a small, genetically susceptible subset of the population. They believed that an infectious agent may be the trigger factor in the pathogenesis.

It has been reported that 28% of patients with PR have T lympho-cytotoxic Antibody, an autoantibody present in 82% of patients with systemic lupus erythematosus (SLE) [46].

PR has also been reported to occur in a patient with Behçet's disease. Whether the PR eruption is related to the disease process, the interferon treatment is totally coincidental is not known [47].

Atopy and genetic predisposition

In a case control study, patients with PR and their relatives were reported to have a higher incidence of asthma and eczema [45].

Other factors

a) Pschychogenic aetiology: It has also been proposed in highly stressed individuals. Though the pschycosomatic theory is considered to be unlikely, it may the depressant effect of stress on the immune system that makes these individuals more susceptible to PR.

b) New Clothing: The distribution of the skin lesion in PR sometimes coincides with the location of various garments of the body, it has been thought that these may precipitate or affect the course of the disease.

c) Pregnancy: The slightly increased prevalence of PR in pregnant women is possible, but this fact has not been confirmed.

d) Bone Marrow transplantation, Administration of BCG/ Hep B/ Pneumovac, Insect Bite, Wasp sting have all been implicated in the causation of the disease, but none has been proved [16,23].

Clinical features and diagnosis

Classic pityriasis rosea

The characteristic feature of a Classical pityriasis rosea is Herald Patch, seen in 80% of all the patients (Syn: Mother patch, Primary Plaque or Plaque Primitive, Primary Medallion).

The herald patch is the first lesion to appear in a PR patient. It is a solitary round or oval lesion with a central wrinkled salmon coloured area and a darker red peripheral zone separated by a collarette of fine scaling.

It may vary from 1 - 10 cm in diameter. The herald patch may occur anywhere on the body, although the trunk and upper arms are its predilected sites i.e. in the areas covered by clothes [26].

The onset of Herald Patch may be preceded by general malaise, nausea, loss of appetite, fever, joint pain and lymphadenopathy.

Figure 1: Lesions aligning along Langer lines in Pityriasis Rosea.

Secondary eruption

The herald patch is followed by the secondary eruption. The range of the interval between primary and secondary eruptions can be as wide as 2 days to 2 months, but is predominantly around 5 to 15 days.

Lesions usually fade in two weeks but new lesions will continue to appear in crops at 2 - 3 days interval over a week or 10 days [23,26].

Collarette scaling

The word pityriasis comes from the Greek meaning bran.

In PR, it describes the fine desquamation of the lesions. Collarette means collar- like which denotes two characteristics of the peripheral scaling pattern in PR:

1. The scaling is circinate or oval.
2. The morphology of scaling is such that fine fragments of scales are attached only at the periphery, reflecting a tendency of peeling from the centre towards the edge. The whole lesion can also be covered with a fine scale initially, then desquamating to leave collarette scaling around each lesion. When stretched across the long axis the scales tend to fold across the lines of stretch which is called as the "Hanging Curtain" sign [49].

Truncal distribution

In classical cases, only the trunk and proximal aspects of the extremities are involved. This distribution pattern is traditionally termed as T-shirt-and-shorts, high-necked short sleeved vest or bathing suit pattern. However, it has been reported that lesions can be distal to the elbow in 4.8% of cases, and distal to the knees or the elbows in 15.3% of cases [18].

The face is usually spared, although sometimes a few patches may spread to the cheeks. Palms and soles are spared in most cases [18]. This fact is usually taken as one of the differentiating features from secondary syphilis.

Symmetry

The secondary rash in Classic Pityriasis Rosea is very symmetrical [5].

Orientation

On the anterior and posterior aspect of trunk the characteristic orientation of the secondary eruptions has been described in various terms as Christmas-tree pattern, inverted Christmas-tree pattern, fir tree pattern, parallel to the ribs or along the skin cleavage lines, that is, on the anterior trunk, the rash seems to be radiating medially and inferiorly, while on the posterior trunk, the rash seems to be radiating laterally and inferiorly. This orientation along the skin cleavage line is most characteristic at the anterior and posterior axillary folds and supraclavicular areas. However, the underlying mechanism for this orientation pattern is unknown [6].

Pruritus

It may vary from no pruritus at all to severe pruritus. Pruritus is severe in 25% of patients; mild to moderate in 50% and absent in 25% of patients. When PR is irritated, itching is usually prominent.

Spontaneous remission

The duration of the rash varies from 2 to 12 weeks, but may last for as long as five months which is known as PR perstans.

PR has been termed as Doctor's Delight, owing to spontaneous remission with no complication.

Relapse

The incidence of relapse is very low, only about 2 - 3% of patients will have a relapse. The eruption is usually less severe in relapse.

Persistence of lesion

PR usually lasts 6 - 8 weeks, although durations as short as 2 weeks have also been reported. A persistent form of PR (PPR) has been reported only in 1 patient so far.

Atypical pityriasis rosea

An epidemiological study estimated that up to 39% of patients with PR may have some atypical features. Atypical features include atypical rash morphology, rash size, rash distribution and site of the lesions [15].

Atypical size of lesions

Pityriasis rosea gigantea of Darier is rare characterised by large sized plaques which can have sizes up to the patient's own palm. In one patient, the herald patch was of the size and the shape of a pear over the right scapular region. The secondary lesions were of sizes about 5 cm by 6.3 cm.

Papular PR is the other extreme in the size of PR lesions. It is more often seen in children. The primary eruption consists of a coalescence of papules which represents the herald patch. The secondary eruption, numerous small papules 1 - 2 mm in diameter may be seen together with the classical oval PR patches.

Atypical morphology of lesions

Atypical Lesion	Typical Feature
Follicular	Lesions here are typically follicular and present in groups or isolated fashion. Differential diagnosis: follicular lichen planus, keratosis pilaris and atopic dermatitis
Vesicular	A generalized eruption of 2 - 6 mm vesicles or as a rosette of vesicles. May be severely pruritic, is most commonly seen in children and young people, and may affect the head, palms, and soles. Differential diagnosis: varicella and dyshidrosis.
Purpuric	Presents as macular purpura on skin and sometimes over the oral mucosa.
Urticarial	Presents with lesions similar to urticarial wheals often accompanied by intense pruritus.
Generalized Papular	Rare form; more common in young children, pregnant women, and Afro -Caribbeans. It presents as multiple small 1 - 2 mm papules which may occur along with classic patches and plaques.
Lichenoid	Observed in the course of atypical PR but is more commonly caused by drugs such as gold, captopril, barbiturates, D-penicillamine, and clonidine.
Erythema Multiforme- Like	Present with targetoid lesions along with the classical lesions of PR. Differential diagnosis: dermatophytic infection.
Giant	It consists of plaques ranging from 5 cm to 7 cm.

Table 1

Atypical herald patch: Herald patch may be absent in 20% of patients or present with secondary eruptions or may occur at unusual sites such as face, scalp, genitalia, or other sites.

Sometimes, the two atypical variants may coexist in the same patient as reported by Sinha., *et al*. where a 16-year-old girl presented with two atypical morphological variants-generalized papular and EM-like.

Atypical site of lesions

Site	Typical feature
Inverse	Lesions are predominantly present in acral and flexural areas involving axilla, groin, and face.
Acral	Located on wrists, palms, lower legs, feet, and sole with sparing of trunk and proximal parts of limbs. Differential diagnosis EM, syphilis, necrolytic acral erythema, and drug eruptions should be excluded
Unilateral	Extremely rare variant reported in both children and adult where the lesions were located on one side of body and patient had herald patch with classical secondary lesions.
Blaschkoid	Lesions follow the Blaschko's line.
Limb- Girdle	Also known as PR of Vidal; the eruption is limited to shoulders or pelvic girdle, thus involving axilla and groins. Lesions are usually larger and more annular.
Oral Mucosa	Involved in 16% of patients, and the lesions may be punctuate, erosive, bullous, or hemorrhagic but are usually asymptomatic in nature.
Localized	The eruptions are localized to one part of the body.

Table 2

Pityriasis rosea in dark-skinned

Dark-skinned individuals have been shown to have certain atypical features such as face may be involved frequently, papular lesions are more common, eruption tends to be more itchy, and subsequent post-inflammatory hyperpigmentation is frequent.

Drug induced pityriasis rosea-like rashes

Barbiturate	Diphtheria Toxoid
Isotretinoin	Levamisole
Bismuth	D-Penicillamine
Interferon α	Metronidazole
Ketotifen	Captopril
Clonidine	Methopromazine
Lithium	Gold

Table 3

Pityriasis Rosea due to drug is now considered as a separate condition distinct from PR in ICD-10.

Pityriasis rosea and vaccination

PR like eruptions has been reported after vaccinations such as Bacillus Calmette-Guerin, influenza, H1N1, diphtheria, smallpox, hepatitis B and pneumococcus.

Recurrent pityriasis rosea

Recurrences of PR are believed to be rare and studies have reported the second episode in 1 - 3% of patients. Multiple recurrences (> 2) are considered a very rare presentation of this very common condition.

Characteristics of drug induced PR

The lesions in a Drug induced PR are generally less numerous, larger and more scaly than usual. The Herald Patch and the Christmas tree distribution are frequently absent.

PR and related physical conditions

In rare cases enanthema may occur with haemorrhagic macules and patches, bullae on the tongue and cheeks or lesions that resemble aphthous ulcer.

Pitting of nails and onychodystrophy after PR has also been reported.

Lymphadenopathy may occur in patients in PR, especially early in the course of the disease and in association with flu like symptom.

Other associated skin diseases more commonly found along with PR are atopy and seborrheic dermatitis and acne vulgaris.

Pityriasis rosea in pregnancy

Pregnant women are more susceptible to pityriasis rosea because of their altered immune response.

Complication

No complications have been reported except for flu like symptom.

Differential diagnoses

For Christmas Tree Eruptions	1. Erythema dyschromicum perstans (Ashy Dermatosis) 2. Lichen planus and lichenoid reactions 3. Pityriasis lichenoides et varioliformis acuta or chronica 4. Kaposi's sarcoma
For Annular eruptions and Herald patch	1. Pityriasis alba 2. Nummular eczema 3. Seborrheic dermatitis 4. Dermatophytic infection
For Papular Eruptions	1. Drug Eruptions and Erythema Multiforme 2. Scabies 3. Guttate Psoriasis 4. Secondary syphilis
For Purpuric PR	1. Vasculitis 2. Haematological diseases [5]
For Inverse PR	Papular Acrodermatitis of Childhood (Gianotti-Crosti Syndrome)
Others	Hodgkins disease, Mycosis Fungoides, Gastric Carcinoma and Bronchogenic Carcinoma are associated with PR like eruption [26].

Table 4

Investigations

Skin scraping for potassium hydroxide smear and fungal culture, serological test to exclude secondary syphilis, complete hemogram.

Pathology

Lesional pathology

The epidermal findings include focal parakeratosis; however in rare cases it may be diffuse. The granular cell layer is reduced or absent. Slight acanthosis, focal spongiosis which may progress to vesiculation can be seen. In the dermis, a superficial perivascular infiltrate of lymphocyte, histiocytes and occasionally eosinophils may be observed. There is papillary dermal oedema and a variable number of extravasated red cells.

Dermascopic features: Peripheral collarette scale followed by central yellow with peripheral reddish background, peripheral dotted vessels with patchy distribution.

Treatment

Though self limiting, water, soap, wool and sweating may cause irritation and should be avoided in acute stages. For patients with mild pruritus zinc oxide or calamine lotion will suffice. For patients with pruritus severe enough to disturb the quality of life, along with topical preparation like zinc oxide or calamine lotion, a course of Erythromycin can be given 250 mg QID for 2 weeks; for children 25 - 40 mg/kg/day in four divided doses for 2 weeks [49], along with antihistamines and mid potency topical corticosteroid for symptomatic relief of pruritus.

For patients with associated flu like symptoms and/or extensive skin diseases, oral Acyclovir 800mg, 5 times daily for 1 week or equivalent Acyclovir derivatives may hasten recovery from disease. Phototherapy has been found to be useful. Dapsone has been used in severe vesicular PR.

Case 1

Classical pityriasis rosea. Exanthematous eruptions with erythemato-squamous lesions, sharply defined, round or oval, following cleavage lines on the trunk.

Figure 2

Case 2

Herald patch in atypical location. Lesion noted over proximal thighs.

Figure 3

Case 3

Inversus pityriasis rosea. Lesion distributed on face and neck; the trunk is not affected.

Figure 4

Case 4

Hypopigmented pityriasis rosea. Round to oval hypopigmented lesions during the whole course of the eruptions.

Figure 5

Case 5

Inversus pityriasis rosea. Lesions distributed on the face and neck.

Figure 6

Dermascopic findings

Figure 7: Dermascopic picture shows: peripheral collarette scale followed by central yellow with peripheral reddish background, peripheral dotted vessels with patchy distribution.

Figure 8: Dermascopic picture shows: Peripheral collarette scale, followed by central yellow with peripheral reddish background.

Histopathological features

Figure 9

Epidermis shows mild acanthosis with mild hyperkeratosis and focal areas of parakeratosis. There are foci of spongiosis with lymphocyte infiltration.

Lymphocytes are extending on to epidermis (lymphocytic exocytosis). Dermis perivascular infiltration (coat sleeve pattern) seen.

Figure 10

Epidermis shows moderate acanthosis with foci of lymphocytic spongiosis. There is parakeratosis, focal to diffuse. Dermis shows perivascular lymphocytic infiltration, and extravasation of erythrocytes, extending focally into epidermis. No eosinophils seen.

Discussion

The present study showed young adults (21 - 30 years) to be commonly affected. Male preponderance was observed in the study.

Most of the population belonged to working class population, 35 patients (41.2%) followed by students, 34 patients (40%) in the present study.

Higher incidence has been similarly recorded in the winter and rainy seasons, from September to January. But sporadic cases were seen throughout the year.

No familial occurrence was present in the study.

In the present study only 3 patients gave a history of wearing new garments prior to the onset of the disease.

Prodromal illness like upper respiratory tract infection and malaise were seen in 2 patients (2.4%).

The herald patch was seen to occur over the covered areas of the body in majority (88%) of the patients.

Christmas tree pattern was noted in 79 patients (92.9%).

Patients with fair skin were found to have herald patch with pinkish tinge were 8 patients (9.4%) as compared to dark skinned patients who had Herald patch with erythematous tinge, 77 patients (90.6%).

No complications were seen in our study.

Blood investigations for 43 patients (50.58%) showed increased level of neutrophils while, 42 patients (49.41%) had neutrophils under normal range.

26 patients (30.6%) showed increased level of lymphocytes, while 59 patients (69.4%) had normal level.

Histopathological findings like flaky hyperkeratosis, patchy parakeratosis, thinned out granular layer, irregular acanthosis, fo-

cal spongiosis, dilated blood vessels and sparse inflammatory infiltrate around blood vessels in upper dermis were seen which is similar to that reported in literature.

Summary and Conclusion

In this clinical and etiopathological study on Pityriasis Rosea done in Department of Dermatology, SSIMS & RC, Davangere, Karnataka the following points were observed.

Clinical diagnosis of Pityriasis Rosea was easy, based on the presence of Herald Patch, characteristic morphology and distribution pattern of the lesions.

The present study has revealed male preponderance (62.4%).

The age incidence was found to be high in young adults (11 - 20 years age) which comprises 41.8% of population.

Most of the patients were working population- 35 patients (41.2%) and students- 34 patients (40%).

Familial incidence was not observed.

The disease was more frequent in the winter and rainy months (September to January) with sporadic cases occurring throughout the year.

Factors like wearing of new garments (3.5%), pregnancy (1.2%), pain abdomen (1.2%) and upper respiratory tract infections (2.4%) were found to precipitate the disease.

The Herald Patch was observed to occur in the covered areas of the body, mostly over the trunk. The secondary eruptions had a wide spectrum of morphological forms with a variable distribution.

Herald Patch was seen to occur more frequently over the trunk in 41 patients (48.2%) followed by the abdomen and back- 11 patients each (12.9%) and 7 patients (8.2%) on the arm.

The size of the herald patch varied from 2 to 5 cm in diameter. The largest sized herald patch was 5 cm in diameter.

The shape was oval and round with peripheral collarette of scales in 81 (95.3%) cases.

Christmas tree was present in maximum number of patients that is 79 patients (92.9%).

The central area of the patch was light brown to erythematous to pink coloured and appeared wrinkled in majority of the cases.

Erythematous color appeared maximum in the population that is in 77 patients (90.6%) and pink in 8 patients (9.4%).

82 patients (96.5%) presented with the symmetrical distribution of the lesion.

And 3 patients (3.5%) presented with asymmetrical distribution of the lesion.

Acne vulgaris was associated in 3 patients (3.5%), atopy was associated in 2 patients (2.4%) and seborrheic dermatitis was associated in 1 patient (1.2%).

Drug intake history with metronidazole in 2 patients (2.4%), NSAIDS in 1 patient (1.2%), omeprazole in 1 patient (1.2%) and terbinafine in 1 patient (1.2%) was associated with onset of Pityriasis Rosea lesion.

Localised Pityriasis Rosea in 1 patient (1.2%), Inverse Pityriasis Rosea in 2 patients (2.4%) and Papular Pityriasis Rosea in 1 patient (1.2%) were also observed in this study.

Most of the patients had complete resolution of the lesions in 9 weeks.

In most of the patients the lesions vanished without trace, and post inflammatory hypopigmentation was noted in few patient.

There was no complication in any of the patients.

Recurrence of the disease was also observed in 2 patients (2.4%).

Patients with increased level of neutrophils were 43 (50.58%) while patients with neutrophils under normal range were 42 (49.41%).

Patients with increased level of lymphocytes were 26 (30.6%), while patients with normal level were 59 (69.4%).

Patients with increased eosinophil count that is more than 5% were 2 (2.4%).

Maximum number of patients that is 83 patients (97.6%) had eosinophils in normal range.

Patients (3.5%) with increased monocyte count were 3 (3.5%), while 82 patients (96.5%) had normal range.

Erythrocyte sedimentation rate (ESR) was increased in 16 patients (18.8%) while 69 patients (81.2%) had normal ESR range.

Histopathological examination of the herald patch showed flaky hyperkeratosis (22.4%), patchy parakeratosis (61.2%), thinned out granular layer (51.8%), irregular acanthosis (71.8%), focal spongiosis (48.2%), dilated blood vessels and sparse inflammatory infiltrate around blood vessels in upper dermis.

Histopathology of the secondary eruptions showed a similar picture to the herald patch like flaky hyperkeratosis, patchy parakeratosis, thinned out granular layer in some areas, focal spongiosis, dilated blood vessels, and patchy inflammatory infiltrate around the blood vessels in upper dermis.

Culture of scales from the lesions did not show any bacterial growth.

In conclusion most of the clinical and histopathological features were consistent with the findings reported in the literature.

Bibliography

1. Champion RH., *et al*. "Viral rashes". In: Rook's Textbook of Dermatology. 6th edition. Oxford: Blackwell Sciences (1998): 1092-1095.
2. Urbina F., *et al*. "Clinical variants of pityriasis rosea". *World Journal of Clinical Cases* 5.6 (2017): 203-211.
3. Percival GH. "Pityriasis rosea". *British Journal of Dermatology* 44 (1932): 241-253.
4. Chuh A., *et al*. "Pityriasis rosea – Evidence for and against an infectious aetiology". *Epidemiology and Infection* 132 (2004): 381-390.
5. Truhan AP. "Pityriasis rosea". *American Family Physician* 29 (1984): 193-196.
6. Weiss L. "Pityriasis rosea – an erythematous eruption of internal origin". *The Journal of the American Medical Association* 41 (1903): 20-28.
7. Crissey JT. "Pityriasis rosea". *Pediatric Clinics of North America* 3 (1956): 801-809.
8. Parsons JM. "Pityriasis rosea of Gibert". *Journal of the American Academy of Dermatology* 16 (1987): 1260-1261.
9. Chuang TY., *et al*. "Pityriasis rosea in Rochester, Minnesota,1969-78". *Journal of the American Academy of Dermatology* 7 (1982): 80-89.
10. Klauder JV. "Pityriasis rosea with particular reference to its unusual manifestations". *The Journal of the American Medical Association* 82 (1924): 178-183.
11. Jacyk WK. "Pityriasis rosea in Nigerians". *International Journal of Dermatology* 19 (1980): 397-379.
12. Jackson R. "The lines of Blaschko: a review and reconsideration: Observations of the cause of certain unusual linear conditions of the skin". *British Journal of Dermatology* 95 (1976): 349-360.
13. Vollum DI. "Pityriasis rosea in the African". *Transactions of the St. John's Hospital Dermatological Society* 59 (1973): 269-271.
14. Ahmed MA. "Pityriasis rosea in the Sudan". *International Journal of Dermatology* 25 (1986): 184-185.
15. Traore A., *et al*. "Pityriasis rosea in secondary schools in Ouagadougou, Burkina Faso". *Annales de Dermatologie et de Vénéréologie* 128 (2001): 605-609.
16. Spelman LJ., *et al*. "Pityriasis Rosea like eruption after bone marrow transplant". *Journal of the American Academy of Dermatology* 31 (1994): 348.
17. Hendricks AA and Lohr JA. "Pityriasis Rosea in infancy". *Archives of Dermatological* 115 (1979): 896-867.
18. Hyatt H. "Pityriasis rosea in a three month old". *Archives de Pédiatrie* 77 (1960): 364.
19. Cohen EL. "Pityriasis rosea". *British Journal of Dermatology* 79 (1967): 533-537.

20. Messenger AG., *et al*. "Case clustering in pityriasis rosea: support for role of an infective agent". *British Medical Journal* 284 (1982): 371-373.

21. Olumide Y. "Pityriasis rosea in Lagos". *International Journal of Dermatology* 26 (1987): 234-236.

22. Harman M Aytekins. "An epidimological study of Pityriasis Rosea in the eastern Anatolia". *European Journal of Epidemiology* 14 (1998): 495-497.

23. Chuh AA., *et al*. "Pityriasis Rosea – an update". *Indian Journal of Dermatology, Venereology and Leprology* 71.5 (2005): 311-314.

24. Okamoto H., *et al*. "Dyskeratotic degeneration of epidermal cells in pityriasis rosea: light and electron microscopic studies". *British Journal of Dermatology* 107 (1982): 189-194.

25. Antonio AT Chuh., *et al*. "Case Clustering in PR". *Archives of Dermatology* 139 (2003): 489-493.

26. Parsons JM. "Pityriasis rosea update: 1986". *Journal of the American Academy of Dermatology* 15 (1986): 159-167.

27. Allen RA., *et al*. "Pityriasis rosea". *Cutis* 56 (1995): 198-202.

28. Wile UJ. "Experimental transmission of pityriasis rosea. A preliminary report". *Archives of Dermatological Research* 16 (1927): 185-188.

29. Aoshima T., *et al*. "Virus-like particles in the herald patch of pityriasis rosea". *Dermatologica* 162 (1981): 64-65.

30. El-Shiemy S., *et al*. "Light and electron microscopic studies of pityriasis rosea". *International Journal of Dermatology* 26 (1987): 237-239.

31. Miller TH. "Pityriasis rosea: report of three cases in one family with clinical variations in two of them". *Archives of Dermatological Research* 44 (1941): 66-68.

32. Chuang TY., *et al*. "Recent upper respiratory tract infection and pityriasis rosea: a case-control study of 249 matched pairs". *British Journal of Dermatology* 108 (1983): 587-591.

33. McPherson A., *et al*. "Is pityriasis rosea an infectious disease?" *Lancet* 2 (1980): 1077.

34. Sugiura H., *et al*. "Evolutionary changes of immunohistological characteristics of secondary lesions in pityriasis rosea". *Archives of Dermatological Research* 280 (1988): 405-410.

35. Watanabe T., *et al*. "Pityriasis rosea is associated with systemic active infection with both human herpesvirus-7 and human herpesvirus-6". *Journal of Investigative Dermatology* 119 (2002): 793-797.

36. Werner Kempf and Volker Adams. "Pityriasis Rosea is not associated with HHV 7". *Archives of Dermatological* 135 (1999): 1070-1072.

37. Antonio AT Chuh., *et al*. "Is HHV-7 the causative agent of PR- a critical review". *Indian Journal of Dermatology* 43 (2004): 870-875.

38. Drago F., *et al*. "Human herpesvirus 7 in patients with pityriasis rosea. Electron microscopy investigations and polymerase chain reaction in mononuclear cells, plasma and skin". *Dermatology* 195 (1997): 374-378.

39. Watanabe T., *et al*. "Pityriasis rosea is associated with systemic active infection with both human herpesvirus-7 and human herpesvirus-6". *Journal of Investigative Dermatology* 119 (2002): 793-797.

40. Antonio AT Chuh. "The association of Pityriasis Rosea with CMV, EBV, Parvovirus B19 Infections". *The European Journal of Dermatology* 13.1 (2003): 25-28.

41. Hudson LD., *et al*. "Pityriasis rosea. Viral complement fixation studies". *Journal of the American Academy of Dermatology* 4 (1981): 544-546.

42. Raskin J. "Possible dermatrophic virus associated with Pityriasis Rosea". *Acta Dermato-Venereologica* 48 (1968): 474-481.

43. John KS Chia., *et al*. "Enterovirus infection as a possible cause of Pityriasis Rosea: demonstration by immunochemical staining". *Archives of Dermatology* 142 (2006): 942-943.

44. Antonio AT Chuh and Henry HL Chan. "Prospective case control study of chlamydin, legionella, Mycoplasma Infection in patients with PR". *The European Journal of Dermatology* 12.2 (2002): 170-173.

45. Burch PRJ and Rowell NR. "Pityriasis rosea – an auto-aggressive disease?" *British Journal of Dermatology* 82 (1970): 549-560.

46. Hosokawa H., *et al*. "Naturally occurring T lymphocytotoxic antibody in viral and related skin diseases". *Acta Dermato-Venereologica* 64 (1984): 275-280.

47. Tay YK and Goh CL. "One-year review of pityriasis rosea at the National Skin Centre, Singapore". *ANNALS Academy of Medicine Singapore* 28 (1999): 829-831.

48. Sharma PK., *et al*. "Erythromycin in pityriasis rosea: A double-blind, placebo-controlled clinical trial". *Journal of the American Academy of Dermatology* 42 (2000): 241-244.

49. Labro MT. "Anti-inflammatory activity of macrolides: a new therapeutic potential?" *Journal of Antimicrobial Chemotherapy* 41 (1998): 37-46.

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