AN ANALYTICAL STUDY OF CLINICAL FEATURES IN 100 CASES OF PITYRIASIS ROSEA

DISSERTATION

Submitted To the Tamilnadu Dr.M.G.R. Medical University In Partial Fulfilment Of The Requirements For The Award Of Degree Of

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CERTIFICATE

This is to certify that this dissertation titled **"AN ANALYTICAL STUDY OF CLINICAL FEATURES IN 100 CASES OF PITYRIASIS ROSEA"** is a bonafide work done by **DR.N.Saravanan,** Post Graduate in M.D. Dermatology, Venereology and Leprosy, Coimbatore Medical College, Coimbatore- 641014, during the academic year 2007-2010. This work was done under my direct guidance and supervision and submitted for the M.D.BRANCH XII A Examination in March 2010 to the Tamil Nadu Dr.M.G.R. Medical University, Chennai.

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DECLARATION

I, **DR.N.SARAVANAN**, solemnly declare that this dissertation titled **"AN ANALYTICAL STUDY OF CLINICAL FEATURES IN 100 CASES OF PITYRIASIS ROSEA"** is a bonafide work done by me at Coimbatore Medical College during 2008-2009 under the guidance and supervision of Prof. Dr. DR.V.Somasundaram, M.D., D.D., Professor and Head, Department of Dermatology, Coimbatore Medical College, Coimbatore-641014.

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, towards partial fulfilment of requirement for the award of M.D. Degree in Dermatology, Venereology and Leprosy (Branch XII A).

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CONTENTS

INTRODUCTION

Pityriasis rosea is an acute self-limiting disease, probably infective in origin, affecting mainly children and young adults, and characterized by distinctive skin eruption and minimal constitutional symptoms.[1] The initial lesion, Herald patch, is followed several days to weeks later by the appearance of numerous similar-appearing smaller lesions, located along the lines of cleavage of the trunk (Christmas tree pattern).[2]

SYNONYMS[3]

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),

Pseudoexantheme erythemato desquamatif (Besnier),

Roseola annulata (Willan),

Roseola furfuracea herpetiformis (Behrend),

Roseola squamosa (Nicolas and Chapard).[3]

Pityriasis Rosea has been reported in all races, the average annual incidence at one center was reported to be 0.16 percent.[2] In temperate regions, it is more frequent during the winter months, whereas in tropical areas, there is some seasonal variation.[1] Usually it is more common between 10 and 35 years of age, with equal sex distribution or a slight female preponderance[2]. It has been reported to occur among persons in the same intimate environment with a higher incidence among dermatologists.[4, 32]

Experiments by various researchers to determine the cause of the disease have been unsuccessful. Various etiological factors that have been attributed are fungus, bacteria, spirochetes, drugs, contact with new garments, psychogenic factors and neurogenic factors.

Currently a virus is believed to play an etiological role. The prodromal illness, generalised exanthema accompanied by constitutional reactions, spontaneous resolution and life long immunity, all points towards a viral cause. Extensive research has been carried out in patients with Pityriasis rosea with respect to the newly identified human herpes virus 6 and 7. It is thus possible and remains an unproved fact that human herpes virus 6 and 7 may play a role extensively in some patients with Pityriasis rosea. [4]

In its classical form, Pityriasis rosea is a distinctive dermatoses which is readily

diagnosed clinically. The initial lesion is a Herald patch or primary plaque, followed by a generalised secondary rash after one or two weeks with a typical distribution parallel to the cleavage lines of the skin, resembling a “Christmas tree”.[1, 2, 4]

The other clinical presentations are macular, papular, vesicular, lichenoid, pustular, purpuric, urticarial and erythema multiforme like.[3, 4,] The lesions can occur rarely over the scalp, palms and soles. After a period of about six to eight weeks the lesions resolve spontaneously leaving a residual hypo or hyper pigmentation without any complications.[5]

REVIEW OF LITERATURE

DEFINITION

Pityriasis Rosea of Gibert is defined as an acute, self-limiting disease, probably infective in origin, affecting mainly children and young adults, and characterised by a distinctive skin eruption and minimal constitutional symptoms.[4] or a self-limiting disorder characterised by the development of asymptomatic erythematous scaly macules on the trunk. *Pityriasis* means fine scales; *rosea* denotes rose coloured or pink.[2]

Pityriasis Rosea is a clinical diagnosis. The typical form is relatively well defined and easily recognised. But, variants or atypical forms including atypical morphology and atypical distribution are fairly common, and even a trained eye may find it difficult to differentiate these atypical forms of Pityriasis rosea.[5]

HISTORICAL BACKGROUND

The term *pityriasis* was first coined by the great Greek physician Claudius Galen (AD129 - 216) to describe dandruff.[6] Robert Willan (1757-1812), an Edinburgh graduate, was regarded by many as the father of modern dermatology. He devised the first modern classification of skin diseases.

Willan described a rash which he termed as *roseola annulata* in 1798. Pierre François Olive Rayer (1793-1867), a French dermatologist, described a very similar rash

and termed it as *erythema annulatum* in 1828[6]. Erasmus Wilson (1809-1884), the first professor of dermatology at London University, wrote about *lichen annulatus serpinginosus* in 1857. He described the lesion as *small, flat, erythematous discs, bounded by a sharp and distinct margin… and converted into rings.*[7] These rashes were named as *pityriasis rosea* later. [7]

The term *pityriasis rosea* was introduced by Camille Melchoir Gibert. [6, 8, 9] He was given the credit for the first accurate description of the rash of Pityriasis rosea. Also, he was the first to report that Pityriasis rosea is subject to recurrences and that Pityriasis rosea was not caused by fungus .[6]

Pierre-Antoine-Ernest Bazin (1807-1878) in 1862 described the annular type of Pityriasis rosea and he was the first to report prodromal malaise in Pityriasis rosea. [6, 10]

Jean Baptiste Emile Vidal, a French dermatologist, described a similar condition which he termed as *pityriasis circiné et marginé.* He thought that Pityriasis rosea and *pityriasis circiné et marginé* were different conditions as the latter runs a longer course. [6]

Some dermatologists consider *pityriasis circinata et marginata of Vidal* as a special form of Pityriasis rosea, with fewer and larger lesions often localised at the axillae or groins. [10, 11] Other dermatologists are of the opinion that *pityriasis circinata*

and *pityriasis circinata et marginata* should be synonyms of Pityriasis rosea. [6] Other synonyms of Pityriasis rosea are of historical interest only, and include *herpes tonsurans maculosus*, *pityriasis disséminé*, *pityriasis rubra aigu*, *roseole squameuse of Chapard* and *pityriasis maculate et circinata of Bazin*.

Herald patch and centripetal scaling

Louis-Anne-Jean Brocq (1856-1928) was the first to describe *plaque primitive* or *primitive patch* as a distinctive diagnostic sign of Pityriasis rosea in 1887, 27 years after Gibert's description of Pityriasis rosea.[7]

In 1899, Alfred Blaschko (1858-1922), a German dermatologist, famous for his description of *Blaschko's lines,*[12] pointed out that *in Pityriasis rosea there is exfoliation from the centre to the periphery, while in psoriasis desquamation takes place from the periphery to the centre.* [7]

Modern nomenclature and classification

In the International Classification of Diseases, ninth Revision, clinical modification (ICD-9-CM), *Pityriasis rosea* was coded under 696, *Psoriasis and similar disorders* as 696.3. *Pityriasis circinata (et maculata)* was listed as a synonym of Pityriasis rosea under code 696.3.[13]

In ICD-10, *pityriasis rosea* is coded under chapter XII - Diseases of the skin and subcutaneous tissue as a separate entity L42.[14]

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EPIDEMIOLOGY

Pityriasis rosea is seen worldwide. All races are equally susceptible to Pityriasis rosea.[11] The clinical presentation of Pityriasis rosea is also similar in all races. African patients tend to have more extensive rash and involvements of the face and scalp have been reported to be more frequent than Caucasians. [11, 15] It was reported that an erythematous or *rosea* picture is seldom seen in black patients.[16] Asian patients tend to have similar rash morphology and extent of involvement as Caucasian patients.[11]

Incidence and prevalence

The average annual incidence at one centre was reported to be 0.16 percent (158.9 cases per 100,000 person-years) [2]. Traore A *et al* in his cross sectional study calculated the prevalence of Pityriasis rosea to be 0.6%. [17]

There is a slightly increased prevalence in patients with decreased immunity such as bone marrow transplant recipients. [18]

Age and sex distribution

Pityriasis rosea affects all ages from infants to the elderly.[19] It is uncommon in young children(less than 2 years) and elderly (more than 65 years). [2, 4] Most patients are between the years of 10 and 35. [2, 4, 5] The youngest reported patient was three months old

and the oldest reported patient was 83 years old. [5, 20]

Camille Melchoir Gibert stated that females are affected more often than males [21]. Many studies have reported a slight female preponderance, the most frequently reported male: female ratio being 1: 1.2 to 1: 1.5.[2, 3, 4, 9,15,11,16, 17, 79] The reason for the apparent female predominance is unknown.

Seasonal Variation

The data on seasonal variation in Pityriasis rosea have been conflicting.[22] In temperate zone, Pityriasis rosea is more common during winter, whereas in tropical regions it has been found to be more common during hot and dry seasons. [1, 9, 22]

Case clustering

Significant temporal clustering independent of seasonal variation occurred in a series of patients with Pityriasis rosea. This may be indicative of an infectious cause. [23]

AETIOLOGY

The evidence to support an infectious aetiology for Pityriasis rosea is its distinct clinical course. There is a primary skin lesion followed by a secondary eruption, with complete remission mostly within about eight weeks. This course of the disease is similar to most of the viral infections. Moreover, many patients do not have a second

attack, due to life long immunity a phenomenon which is commonly seen in many viral diseases. [2, 24, 25]

Most experiments at transmitting Pityriasis rosea to human beings have failed. But, Wile UJ in his study has shown that Pityriasis rosea can be transferred using blister fluid or extract of scales of Pityriasis rosea patients. He also showed that bacterial cultures from the scales were negative. [26]

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 [27]. Virus-like particles in the dyskeratotic keratinocytes were also reported in another study. [28] The viral
DNA is reported to be present in peripheral blood mononuclear cells and lesional and unaffected skin of the majority (80–100%) of individuals with acute pityriasis rosea.[1, 4]

1. Evidences supporting an infectious aetiology

a) Case clustering

Cluster analysis is an epidemiologic approach to investigate a possible infectious cause. Significant temporal clustering independent of seasonal variation occurred in a series of patients with Pityriasis rosea. This may be indicative of an infectious etiology. [23, 29]

b) Concurrent cases

There is no report of any true epidemic for Pityriasis rosea.[4] However, since the first descriptions of this condition, there have been many case reports of two or more patients with Pityriasis rosea in the same family or intimate environment. [30]

c) Associations

Epidemiological studies reported associations of Pityriasis rosea with prodromal illness of respiratory tract infections,[31] unfavourable social and economic backgrounds, [17] and contact with patients with Pityriasis rosea. [32]

An interesting study compared the incidence of Pityriasis rosea of dermatologists and otolaryngologists. It showed higher incidence of Pityriasis rosea in dermatologists when compared to otolaryngologists, claiming that frequent exposure to Pityriasis rosea by dermatologist during their practice led to an increased risk. [4, 32]

d) Immunology

Immunohistologic data shows perivascular aggregates of predominantly active CD4 T lymphocytes in the superficial dermis. An increase in Langerhan cells, which is an antigen presenting cell and the expression of HLA-DR^+ antigen on the surface of keratinocytes located around the area of lymphocytic exocytosis suggesting an infectious aetiology for Pityriasis rosea. [33, 34, 36]

e) Human Herpes virus 6 and human Herpes virus 7

Herpes virus like particles has been found in 71% of Pityriasis rosea lesions.

Human herpes virus 6 and Human herpes virus 7 have been suggested as the cause for the eruption. 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 Pityriasis rosea. Human herpes virus 7 is detected slightly more frequently than human herpes virus 6. However, evidence for the presence and activity of human herpes virus 6 or human herpes virus 7 is also found in a proportion (10–44%) of unaffected individuals. Hence, its role as a causative agent is yet to be proved.[4, 35, 48, 94]

f) Influenza and Para influenza viruses

One study analysed whether Pityriasis rosea is due to Influenza and Parainfluenza viruses as many patients gave a history of antecedent upper respiratory illness, but the investigators concluded that Pityriasis rosea is not related to these viral infections because there was no significant rise of antibody for these viruses. [37]

g) Picornovirus

Earlier work has demonstrated Picornovirus like intra nuclear inclusion bodies in the tissues of African green monkey, inoculated with fluid from Pityriasis rosea lesions; however other attempts failed to demonstrate this virus genome. [38]

i) Enterovirus

Enteroviruses tend to produce a variety of exanthemas. A case has been reported with Pityriasis rosea like skin eruption with a typical Christmas tree pattern and the demonstration of a monoclonal antibody that identified enteroviruses. This suggests that an unusual enterovirus could be the possible cause of the rash. [39]

k) Streptococcus

A recent Indian study concluded that there is no association between streptococcal pharyngitis and pityriasis rosea.[41]

2. Autoimmunity

An autoimmune element in the pathogenesis of Pityriasis rosea has been suspected by some investigators.[42] They proposed that Pityriasis rosea is an auto aggressive 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 Pityriasis rosea have T lymphocytotoxic antibody, an autoantibody present in 82% of patients with systemic lupus erythematosus (SLE).[42] Pityriasis rosea has also been reported to occur in a patient with Behçet's disease. Whether the Pityriasis rosea eruption is related to the disease process, the interferon treatment or is totally coincidental is unknown.[43]

Treatment with systemic corticosteroids for recalcitrant Pityriasis rosea has been advocated.[24] In a study 20 cases with extensive inflamed eczematised Pityriasis rosea were treated with systemic steroids with a short tailing course of oral prednisolone over two to three weeks. Improvement was noted in all 20 patients which may support the hypothesis that Pityriasis rosea may be an autoimmune disease, but it was not known that, whether such improvement was related to the systemic corticosteroids *per se* or due

to spontaneous remission.[44]

Treatment of Pityriasis rosea with erythromycin has been reported to have remarkable effects in modifying the course of the disease.[45] Apart from its effects as an antibiotic, erythromycin also has anti-inflammatory and immunomodulatary effects. In the case of autoimmunity or immune dysfunction being an important component in the pathogenesis of Pityriasis rosea, these effects may also contribute towards the action of erythromycin in Pityriasis rosea.[46]

3. Atopy and genetic predisposition

In a case control study, patients with Pityriasis rosea and their relatives were reported to have a higher incidence of asthma and eczema. Such findings support genetic predisposition being an underlying factor in Pityriasis rosea, and that an infectious agent may be the trigger factor.[3, 4, 42.]

4. Other factors

a) **Psychogenic aetiology:** has also been proposed in highly stressed individuals. Though the psycho somatic theory is considered to be unlikely, it may the depressant effect of stress on the immune system that makes these individuals more susceptible to Pityriasis rosea.[47]

b) **New Clothing:** Because, the distribution of the skin lesion in Pityriasis rosea

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.[3]

c) **Pregnancy:** The slightly increased prevalence of Pityriasis rosea in pregnant women is possibly due to new clothing, but this fact has not been confirmed.[3]

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. [1, 18, 48]

e) **Drugs:** Many drugs have been attributed in causing Pityriasis like rashes for example arsenic. [4]

Probable Pathogenesis:

Human herpes virus 7 and human herpes virus 6 do not infect keratinocytes but instead infect $CD4^+$ T cells within blood and are retained within these cells in a latent form in most individuals. These cells are the likely source of cell-free viral DNA found in plasma or serum samples of patients with Pityriasis rosea. They are also the likely source of the scattered perivascular and perifollicular virus-positive cells observed within some lesions of Pityriasis rosea.[49]

CLINICAL FEATURES AND DIAGNOSIS

Classical pityriasis rosea

The characteristic features of a Classical pityriasis rosea are as follows:

Herald Patch (Synonyms: Mother patch, Primary Plaque or Plaque Primitive, Primary Medallion)

The herald patch is the first lesion to appear in a Pityriasis rosea 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 be hyper pigmented in dark individuals.[2] 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.[24] Rarely it may occur on the face, scalp or the penis[4]. When the plaque is irritated, it may have an eczematous papulovesicular appearance.[3]

The Herald patch is seen in 80% of all Pityriasis rosea patients. [47] In another study, where a series of 127 patients were examined, 76 % were reported to have a herald patch.[50] However, its true incidence is difficult to be ascertained as it is easily missed by the patients.

Herald patch sometimes develops at the site of recent lesion such as minor cutaneous infection, flea bite, wasp sting, and BCG or Hepatitis B vaccination[1, 7]. Prodromal symptoms are absent in most of the patients.[4] In a minority of patients, flu-like symptoms have been reported, including general malaise, headache, nausea, loss of

appetite, fever, and arthralgias.[3]

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 few hours to months, but is predominantly around 5 to 15 days.[1, 4] Two main types of lesions are seen, one is the lesion of similar morphology as the herald patch, but smaller in size, and the other is small, red, non scaling papules that gradually increase in number and spread peripherally. Both the forms can exist concomitantly.[2] Old 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.[1, 2, 24]

Collarette scaling

The word *pityriasis* comes from the Greek meaning *bran.* [51] In Pityriasis rosea, it describes the fine desquamation of the lesions. *Collarette* means *collar-like*. This term denotes the characteristics of the centripetal scaling pattern in Pityriasis rosea, the scaling is circinate or oval and 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. [51] The whole lesion can also be covered with a fine scale initially, then desquamating to leave collarette scaling around each lesion. The amount of scales present is highly variable.[51] When stretched across the long axis the scales tend to fold

across the lines of stretch which is called as the "Hanging Curtain" sign. [52] Collarette scaling is important for the diagnosis for Pityriasis rosea. However, its recognition is sometimes difficult.

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.[50] However, it has been reported that lesions can be distal to the elbow in 4.8% to 12% of cases, and distal to the knees or the elbows in 6% to 15.3% of cases. [4, 21]

The distal involvement is commonly seen in older patients.[21] The face is usually spared, except in children.[2, 4] Palms and soles are spared in most cases.[21] This fact is usually taken as one of the differentiating features from secondary syphilis. However, palmoplantar involvement in Pityriasis rosea showing lesions similar to secondary eruptions elsewhere in the body was reported by Klauder JV as early as 1924.[10]

Symmetry

The secondary rash in Classic Pityriasis Rosea is very symmetrical.[2, 3, 5, 53]

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 [7, 54]

Pruritus

Pruritus in Pityriasis rosea may vary from no pruritus at all to severe pruritus. Pruritus is severe in 25% of patients; slight to moderate in 50% and absent in 25% of patients.[2] When Pityriasis rosea is irritated, itching is usually prominent. [3, 55] The nature of pruritus in Pityriasis rosea has not been specifically reported in the literature.

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 Pityriasis rosea perstans.[56] Post-inflammatory hyper or hypopigmentation may also last for month.[5] Pityriasis rosea has been termed as *Doctor's Delight,* owing to spontaneous remission with not much of complications. [50]

Relapse

Second attacks of pityriasis rosea occur in about 2% of cases after an interval of a

few months or many years.[1] Rarely, partial or complete relapse of a fading eruption may be seen[4]. In a series of 826 patients, the rate of relapse was noted as 2.8%. [47] The eruption is usually less severe in relapse. [47]

Atypical pityriasis rosea

Atypical forms of Pityriasis rosea are fairly common. An epidemiological study estimated that up to 39% of patients with Pityriasis rosea may have some atypical features. Atypical features include atypical rash morphology, rash size, rash distribution and site of the lesions. [17, 57]

Atypical Herald Patch

The Herald Patch may be absent or undetected in about 20% of cases.[4] There may be two or more patches. The primary plaque may be the sole manifestation of the disease or only one of two lesions. [3]

Atypical morphology of secondary lesions

Atypical rash morphology includes papular, vesicular, purpuric or haemorrhagic, urticarial, lichenoid and erythema multiforme - like forms. [3, 4, 58, 95]

Vesicular Pityriasis rosea usually presents as a generalised eruption of 2-6 mm vesicles. Palms and soles can be involved, but like the classic Pityriasis rosea, the face and scalp are usually spared. Vesicular Pityriasis rosea is said to be commoner in children and young adults and is more commonly seen in Africa [59, 60]. Vesicular Pityriasis

rosea may exist alone or may be concomitantly seen with classic oval scaly Pityriasis rosea patches.[58] Vesicular Pityriasis rosea may be severely pruritic and extensive.[61] The lesions may simulate a wide spread eczematous eruption with weeping and crusting. In a series of 138 patients with Pityriasis rosea, four patients were reported to have vesicular Pityriasis rosea.[11] It is believed that the vesicular lesions are due to exaggerated spongiosis and exocytosis with intraepidermal separation. [62]

Purpuric Pityriasis rosea usually presents with tiny purpuric spots affecting the trunk and proximal part of the extremities. Accompanying petechiae may be visible over the palate. The histopathology is usually the same as for classic Pityriasis rosea. Some lesions may reveal mild to moderate extravasation of erythrocytes in the papillary dermis, associated with dilatation of capillaries. Histopathological evidence of accompanying allergic vasculitis is usually absent. The prognosis is the same as for classic Pityriasis rosea. It is likely that *haemorrhagic* and *purpuric* Pityriasis rosea are different terminologies describing the same condition. [62, 63, 64]

Urticarial Pityriasis rosea, also known as *pityriasis rosea urticata*, present with considerably raised lesions resembling urticarial wheals. It is often accompanied by intense pruritus. [10, 68]

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.[65, 68] The secondary lesions were of sizes about 5cm by 6.3cm [65]. The clinical course of pityriasis rosea gigantea of Darier is similar to classic Pityriasis rosea .[10, 68]

Papular Pityriasis rosea is the other extreme in the size of Pityriasis rosea lesions.[66] It is more often seen in children.[68] 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 Pityriasis rosea patches.[10]

Atypical distribution of lesions

Cases with relatively asymmetrical rash distribution are uncommon but not rare. Atypical distribution of lesions is frequently in the form of acrally distributed rash, also known as *Pityriasis rosea inversus.* [67, 68, 95] The face, axilla and groin are predominantly affected. In the *limb-girdle* type, the eruption is restricted to the shoulders or the hips. [22] Even cases with strictly unilateral involvement have been reported. [69]

In localised Pityriasis rosea the lesions are confined to one part of the body[4]. The trunk is usually the predilected site. The rash morphology and time course of localised Pityriasis rosea are the same as for classic Pityriasis rosea. [70]

Pityriasis circinata et marginata is sometimes considered as a special or atypical form of Pityriasis rosea. [10, 11] This is mainly seen in adults, with fewer and larger lesions often localised to the axilla or inguinal region. Pityriasis circinata et marginata usually persists for a longer period than classical Pityriasis rosea, and sometimes precedes a typical generalised Pityriasis rosea rash[4]. In a series of 138 patients with Pityriasis rosea, four patients were reported to have pityriasis circinata et marginata of Vidal. [11]

Atypical site of lesions

It has been reported that involvements of the face, scalp, hands and feet are uncommon in Pityriasis rosea. [10] The scalp, eyelids and penis can also be involved in Pityriasis rosea.[10]Two cases of Pityriasis rosea with palmoplantar plaque lesions have been reported.[71] Atypical lesions on the patients feet and wrist may occur, because of their respective environments which are prone to sweating and irritation (lesion under the watch band; socks and shoes on the feet).This variant is known as *Pityriasis Rosea Irritata,* which may resemble guttate psoriasis. [51, 68]

The oral cavity is another atypical site for Pityriasis rosea lesions.[68, 72] Oral lesions may occur in discrete or confluent patches. They may be white, haemorrhagic, erosive, or bullous. Two major types of oral involvement were described: tiny punctate haemorrhages with pinhead erosions over the buccal and palatal mucosa, and discrete, slightly elevated lesions sometimes with superficial erosions can be seen.[11] Intraoral lesions are usually asymptomatic. They either follow a similar course as cutaneous

Pityriasis rosea lesions, or tend to subside several days before the cutaneous eruption. [11, 68] Rarely, Pityriasis rosea lesions can also occur over vulval region.[1]

Drug induced pityriasis rosea-like rashes

Many drugs including arsenic,[4] tyrosine kinase inhibitor,[73,74] captopril,[75] gold,[68] isotretinoin,[68] lithium,[14] non-steroidal anti-inflammatory agents,[76] omeprazole[77] and terbinafine[78] have been implicated in causing Pityriasis rosea-like rashes.

It was reported that when ampicillin was consumed by 29 patients with Pityriasis rosea, the lesions got worse and urticated.[79] The face was frequently involved and pruritus was also more frequent. This phenomenon was analogous to ampicillin rash in infectious mononucleosis. It is also reported that this intolerance to ampicillin was not seen with other antibiotics such as erythromycin or co-trimoxazole. [79]

Characteristics of Drug induced Pityriasis rosea

Drug induced Pityriasis rosea may be of the classic type, but it often shows atypical features. The lesions are generally less numerous, larger and more scaly than usual. The Herald Patch and the Christmas tree distribution are frequently absent. In addition, associated oral lesions, persistence of lesions, striking resistant to therapy, post inflammatory hyper pigmentation and transformation to lichenoid dermatitis are more common.[3]

Drugs associated with a Pityriasis rosea like eruption	
Arsenic	Isotretinoin
Barbiturate	Interferon α
Bismuth	Ketotifen
Captopril	Lithium
Clonidine	Levamisole
Diptheria Toxoid	Metronidazole
D-Penicillamine	Methopromazine
Gold	Omeprazole
HydroxyChloroquine	Terbinafine
Imatinib mesylate	

Pityriasis rosea 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 ulcers.[2]

Pitting of Nails and onychodystrophy after Pityriasis rosea has also been reported.[80] Lymphadenopathy may occur in patients in Pityriasis rosea, especially early in the course of the disease and in association with flu like symptom.[2] Other associated skin

diseases more commonly found along with Pityriasis rosea are atopy and seborrheic dermatitis and acne vulgaris.[3]

Systemic Involvement

Involvement of internal organs has not been documented in Pityriasis rosea. [81]

Complications

No complications have been reported except for flu like symptom but these are relatively mild if they occur. [2]

Differential diagnoses

1. ***Christmas Tree Eruptions:*** The following conditions also have Christmas tree distribution of lesion and should be differentiated from Pityriasis rosea.

- *Erythema dyschromicum perstans (Ashy Dermatosis)*: In ashy dermatosis slight erythema precedes the characteristic bluish brown patches following the clefts of the skin. The colour also differs from the pigmentation that follows Pityriasis rosea and fades faster in Pityriasis rosea. Histopathology shows hydropic degeneration of the basal cell layer of epidermis which is different from that of Pityriasis rosea.[3]
- *Lichen planus and lichenoid reactions*: These are often caused by drugs and have

a Pityriasis rosea distribution. The lesions have the characteristic of both Pityriasis rosea and Lichen planus on naked eye examination and also on microscopy.[3]

- *Pityriasis lichenoides et varioliformis acuta or chronica*: These lesions also follow lines of cleavage of the skin and may present with a "Christmas Tree" pattern on the trunk, but as a rule, typical lesion will be found on the extremities. Histologically also, it mimics Pityriasis rosea. [3, 82]
- *Kaposi's sarcoma*: May present with a Pityriasis rosea like distribution pattern with oval, violaceous papules and nodules chiefly on the arms, neck and trunk.[3]

2. ***Annular eruptions and Herald patch:*** Annular Pityriasis rosea and Herald patch should be differentiated from the following clinical entities.

- *Pityriasis Alba:* It usually occurs in children and young adults over the face, arm and thorax, and when the lesions are irritated an annular erythema develops similar to Pityriasis rosea.[3]
- *Nummular eczema*: Nummular eczema localised to the trunk can also pose difficulties in diagnosis, but these lesions are usually round, not oval and the papulovesicular elements are more prominent than in Pityriasis rosea. also, the duration of the disease is more when compared to Pityriasis rosea.
- *Seborrheic dermatitis:* can present as annular or figurate lesion on the trunk and arms, but as a rule the scalp and face show the typical picture of seborrheic

dermatitis and the course is protracted.[3]

- *Dermatophyte infection:* may mimic Pityriasis rosea in acute stages. This can be ruled out by mycologic investigations like scraping for KOH examination or by fungal culture.[3]

3. ***For Papular Eruptions:*** When there is no primary plaque, the papular eruptions should be excluded from

- *Drug Eruptions and erythema multiforme*[3]
- *Guttate psoriasis:* is very difficult to differentiate from Pityriasis rosea when only few lesions are present. Histological examination is not always helpful. Psoriasis runs a longer course. In psoriasis the lesions are surmounted by silvery scales.[4]
- *Secondary syphilis*: involvement of palms and soles is characteristic of secondary syphilis, but it is rare in Pityriasis rosea. There is no herald patch and the lesions are roseolar or maculopapular.[4] Serologic test for syphilis will differentiate the two.[3]

4. ***Purpuric Pityriasis rosea:*** should be differentiated from vasculitis and haematological diseases. [5]

5. ***Inverse Pityriasis rosea:*** Papular Acrodermatitis of Childhood (Gianotti-Crosti Syndrome). [3, 83]

6. ***Other Differential diagnosis:*** Hodgkins disease, mycosis fungoides, gastric carcinoma and bronchogenic carcinoma are associated with Pityriasis rosea like eruptions.[24]

Diagnostic criteria

A set of diagnostic criteria has been devised and validated for Pityriasis rosea, but its reliability and applicability in other ethnic groups remains to be ascertained.[22]

Diagnostic criteria of pityriasis rosea
A patient is diagnosed as having pityriasis rosea if: 1. On at least one occasion or clinical encounter, he / she has **all** the **essential clinical features** and **at least one** of the **optional clinical features**, and 2. On all occasions or clinical encounters related to the rash, he / she does **not** have **any** of the **exclusional clinical features**.
Essential clinical features: 1. Discrete circular or oval lesions 2. Scaling on most lesions 3. Peripheral collarette scaling with central clearance on at least two lesions
Optional clinical features (at least one has to be present):

1. Truncal and proximal limb distribution, with less than 10% of lesions distal to mid-upper-arm and mid-thigh 2. Orientation of most lesions along direction of the ribs 3. A herald patch (not necessarily the largest) appearing at least two days before the generalized eruption
Exclusional clinical features: 1. Multiple small vesicles at the centre of two or more lesions 2. Most lesions on palmar or plantar skin surfaces 3. Clinical or serological evidence of secondary syphilis

Investigations

Pityriasis rosea is a clinical diagnosis. Lesional histopathological changes are non-specific. Taking a lesional biopsy thus cannot confirm a diagnosis of Pityriasis rosea. The roles of investigations are to exclude important differential diagnoses and to provide additional support for the diagnosis in atypical cases. Histopathological examination was not performed even in a recent clinical trial on Pityriasis rosea, as *it seldom helps in diagnosis* according to the investigators [45]. Skin scraping for potassium hydroxide smear and fungal culture may be indicated only if tinea corporis is suspected clinically.[3]

It has been suggested that, for adolescents at least, secondary syphilis should be excluded with serology tests for all cases diagnosed with Pityriasis rosea, especially when the palms or soles are affected, when a herald patch is not seen, or when morphologic atypia complicates the clinical picture .[84]

The blood picture is usually normal, but leukocytosis, neutrophilia, basophilia and lymphocytosis have been reported. Slight increase in erythrocyte sedimentation rate, total protein, α1 and α2 globulins and albumin also have been observed. Tests for rheumatoid factor, cold agglutinins and cryoglobulins have been normal but none of these helps in diagnosing Pityriasis rosea. [3, 85]

PATHOLOGY

Lesional Pathology

Histopathological changes in Pityriasis rosea are non-specific. Lesional biopsy can therefore provide evidence to support the diagnosis and exclude certain important differential diagnoses only, but not to confirm a diagnosis of Pityriasis rosea. Histopathological changes in the generalised eruption typically shows a reaction pattern classified as superficial perivascular dermatitis. [24, 85] 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. A most impressive sign of Pityriasis rosea is the presence of microscopic vesicles, sometimes subcorneal in a clinically dry lesion. In the dermis, a superficial perivascular infiltrate of lymphocytes, histiocytes and occasionally eosinophils may be observed. There is papillary dermal oedema and a variable number of extravasated red cells.[34]

Extravasated red cells may be seen not only in the papillae but also in the epidermis. The papillary dermal red cells are particularly prominent in purpuric Pityriasis rosea. [7, 85]

Histopathological changes in the herald patch are similar to generalised eruption. The Herald patch may have in addition to the histopathological changes described above more pronounced acanthosis, less of spongiosis and deeper and denser perivascular inflammatory infiltrate and papillary dermal oedema. [3, 5]

Other histologic features that have been reported include focal necrosis of epidermal cells; dyskeratotic cells in the upper and middle epidermis with an eosinophilic, homogenous appearance, suggesting primary damage to the basal cells; multinucleated epidermal giant cells can be seen as in other inflammatory states. Focal acantholytic dyskeratosis have also been reported.[86, 87, 88]

Histological changes in Pityriasis rosea may also closely resemble superficial gyrate erythema, erythema annulare centrifugum, guttate psoriasis, and small plaque parapsoriasis. As small vesicles or micro-abscesses similar to those present in psoriasis are sometimes seen, the histological differentiation of Pityriasis rosea from early lesions of guttate psoriasis can be particularly difficult.[28] The distinction of guttate psoriasis

from Pityriasis rosea should therefore still be made on clinical ground.[51]

An electron microscopy study on lesional biopsy specimens of patients with Pityriasis rosea showed cytolytic changes in keratinocytes adjacent to Langerhans cells, which are the antigen presenting cells and these findings suggest a cell mediated immune reaction in the epidermis. [89]

A study of skin biopsy specimens from patients with Pityriasis rosea reported high CD4: CD8 ratio in the dermal T cell infiltrate in active Pityriasis rosea lesions. This ratio significantly decreases in old inactive Pityriasis rosea lesions.[1, 90] This might indicate that Pityriasis rosea is also an inflammatory dermatosis similar to psoriasis and that cell mediated immunity may play an important role in the pathogenesis of the disease. [90]

Alterations in the peripheral blood

In the peripheral blood, elevation of the erythrocyte sedimentary rate, a slight decrease in the number of T-lymphocytes and an increase in B-lymphocytes were reported.[47] These changes are similar to those seen during acute viral infections, offering some support for a viral aetiology in Pityriasis rosea. [47]

The case controlled study reported normal levels of serum IgG and IgA but higher levels of total IgM, decreased C3 and normal C4 in patients with Pityriasis rosea. These findings were compatible with a viral aetiology for Pityriasis rosea.[91]

QUALITY OF LIFE AND Pityriasis rosea

The QUALITY OF LIFE (QOL) of patients with Pityriasis rosea is significantly less affected when compared to patients with atopic dermatitis and acne vulgaris. The effects on the QOL of patients with Pityriasis rosea doesn't correlate with rash severity, but rather correlates significantly with the patient's concerns regarding the aetiology and infectivity of the disease.[92]

TREATMENT

a) Since Pityriasis rosea is self limited, there is no need for active treatment. [1, 2]
b) For all patients: Education about the disease process and reassurance.[2]
c) Water, soap, wool and sweating may cause irritation and should be avoided in acute stages.
d) For patients with mild pruritus zinc oxide or calamine lotion will suffice.
e) 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 (Tab. Erythromycin 250 mg QID for 2 weeks; for children 25-40 mg/kg/day in four divided doses for 2 weeks).[45] along with anti histamines and mid potency topical corticosteroid for symptomatic relief of pruritus.[3]
f) The use of systemic corticosteroids should be restricted to adult patients with exceptionally recalcitrant and symptomatic Pityriasis rosea resistant to other treatments.[44]

g) For patients early in the disease course who demonstrated 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. [2, 76]

h) In certain patients phototherapy has been found to be useful. Ultraviolet radiation in erythmogenic doses (a dose large enough to produce erythema and desquamation) is necessary. But, post inflammatory pigmentation at the site of the Pityriasis rosea lesion following UV radiation therapy has prompted warning against this form of treatment. [2, 3, 93]

i) Dapsone has been used in severe vesicular Pityriasis rosea.[3]

AIM OF THE STUDY

TO ANALYSE

1) The incidence of Pityriasis rosea in Coimbatore Medical College Hospital, Coimbatore during the period May 2008 to April 2009.
2) Age and sex distribution.
3) Probable aetiological factors.
4) Symptoms of Pityriasis rosea.
5) Morphological types and distribution of Pityriasis rosea.
6) Associated cutaneous findings.
7) Course of the disease.

MATERIALS AND METHODS

The study was conducted at the Department of Dermatology, Coimbatore medical college during the period May 2008 to April 2009. All the patients attending the dermatology out patient department at Government general hospital, Coimbatore, were screened and patients with pityriasis rosea were enrolled. The clinical diagnosis of Pityriasis Rosea (Pityriasis rosea) was made in each case based on the morphology and distribution of the skin lesions.

Inclusion criteria:

Patients living in and around Coimbatore, who were willing for follow up and who had not taken any treatment for the present condition.

Exclusion criteria:

Patients who already tested positive for VDRL, ELSIA for HIV and for spores / hyphae in KOH examination of the scales. Patients who were pregnant at the time of diagnosis of Pityriasis Rosea.

During the first visit of the patients, their name, age, sex, address, occupation and income etc. noted. Complaints, probable precipitating factors, prodromal illness, contact and family history were also noted.

A thorough examination of both general and systemic carried out. Dermatological examination including the number, site, size, morphology and distribution of primary and secondary lesions followed by examination of palms and

soles, hair, nail and mucous membrane carried out. Based on this, patients were classified as classic or atypical variety of Pityriasis rosea.

Investigations like haemogram, urine and motion examination, skin scraping for fungus, blood VDRL etc. were done. Skin biopsy was done in 5 cases. Mantoux test was done in all cases, to find if there was any immune suppression, as seen in the other diseases with viral aetiology, since Pityriasis rosea is also thought to be caused by virus.

All the patients were referred to ENT and Dental departments to rule out focal sepsis. All the patients were given symptomatic treatment and followed up for a period of 3 months at weekly intervals. The sequelae and complications if any were recorded.

Details thus obtained were compiled, tabulated and statistically summarised.

OBSERVATIONS AND RESULTS

After analysing 100 cases of Pityriasis Rosea the following observations were made.

Sex incidence

Number of Males	Number of Females
73	27

Out of 100 cases studied 27(27%) were females 73 (73%) were males. This indicates a male preponderance**.** (Fig.1)

Age incidence

An analysis of 100 cases under study revealed the age incidence as noted in Fig. 2

Age in yrs	**Number of cases**
0-10	20
11-20	27
21-30	26
31-40	18
41-50	07
51-60	01
61-70	01

Maximum incidence was noted in the age group between 11-30 years (53%). The youngest patient in the study was 3 years old and the oldest was 68 years old. Age and Sex incidence is given in Fig. 3.

FAMILIAL OCCURRENCE

There was no history of familial occurrence in the 100 cases studied

PROVOKING FACTORS

1. New clothes – 4 patients (4%) gave history of wearing new clothes, prior to the appearance of the rash.
2. Drugs – No patient gave history of drug intake prior to the onset of rash.
3. Stress- Stress in the form of unemployment reported by 2 patients (2%).

PRODROMAL ILLNESS

Prodromal illness prior to the onset of rash was reported by 8 patients (8%), of whom 1 had fever alone, 1 had fever with history of jaundice and 6 had upper respiratory tract infection.

ITCHING

Grade	No of patients
Absent	51
Mild	46
Moderate	01
Severe	02

Itching was graded subjectively. Itching was absent in 51 patients. 49 patients (49%) had itching of whom most patients (46) had only mild itch. Among the two patients with severe itching, one gave history of swimming in river water, the other had lichenoid Pityriasis Rosea.

HERALD PATCH

Herald patch was present in 82 cases and absent in 18 cases.

Herald Patch was seen to occur more frequently over the trunk (45) followed by Upper limb and Thigh. The size of the herald patch varied from 1 to 7.5 cm in diameter. The largest sized herald patch was 7.5cm in diameter. The shape was Oval or round with peripheral collerette of scales in all the cases. The central area of the patch was light brown coloured and appeared wrinkled in majority of the cases.

In 2 patients only Herald patch was seen. One patient had 2 Herald patches. In one patient only one secondary lesion was seen near the Herald patch.

The following are the location of the Herald Patch:

Site of patch	**Number of patients**
Chest	09
Abdomen	07
Umbilicus	02
Loin	08
Back	16
Shoulder	02
Scapula	01
Neck	04
Face	07
Upper limb	13
Thigh	09
Lower limb (Knee & below knee)	04

Secondary lesions

After a varying period, ranging from 1- 30 days following the appearance of the Herald Patch, the generalised eruptions followed. They appeared in crops, intermittently at various intervals of 3-10 days. In most of the cases, it appeared on the trunk, upper thigh and arms, in a swimming suit distribution. The secondary eruption followed the skin cleavage lines/langer's lines in all the cases.

TYPES OF CLINICAL PRESENTATION

Clinical presentation	**Number of Patients**
Classical	45
Atypical	55

Both morphology and distribution (typical bathing suit) were Classical in 45% patients. In the remaining 55% atypical morphology and/or atypical distribution were observed.

Morphology	**Number of Patients**
Classical	71
Atypical	29

Distriution	**Number of Patients**
Classical	79
Atypical	21

ATYPICAL LESIONS

Atypical Morphology:

Papular Pityriasis rosea	19
EMF like Pityriasis rosea	06
Lichenoid Pityriasis rosea	02
Only Herald patch	02

Atypical Distribution:

Inverse Pityriasis rosea	11
Unilateral Pityriasis rosea	01
Flexural	03

Atypical Sites:

Face	07
Palm & soles	03
Distal limb	07
Penis	01
Thighs	02
Abdomen	01

Morphologically classical secondary lesions were seen in 71 cases. Morphologically atypical lesions seen in 27 cases of which papular lesion seen in 19 cases, erythema multiforme like lesion seen in 6 cases, and Lichenoid lesion in 2 cases. No secondary lesion seen in 2 cases. In one patient only single secondary lesion near the

Herald patch seen.

Classical distribution of secondary lesion seen in 79 patients of whom distal limb involvement seen in 6 patients, face involvement seen in another 6 patients and face and palms & sole involvement in 2 patients.

Atypical distribution seen in 18 patients, of whom 11 cases had inverse distribution of whom one with few lesions over the abdomen, 3 had flexural involvement (including 1 with both flexural and distal limb involvement), 2 patients with predominantly involvement of thighs of whom one with lesions over the penis, 1 case with involvement of abdomen only and 1 case of unilateral Pityriasis Rosea. 2 patients had only Herald patch one patient had a single secondary lesion. Vesicular, purpuric, urticarial, and pustular forms were not seen in our patients.

MUCOUS MEMBRANE LESIONS

In 100 patients analysed no one had lesions over the mucous membrane.

NAIL CHANGES

No nail changes associated with Pityriasis Rosea observed in all cases.

RECURRENCES

Only 2 patients gave history of recurrence (past history of similar illness 1 year back) in our study.

COMPLICATIONS/ SEQUELAE

Post inflammatory hypopigmentation was seen in 3 patients (3%). No other complication noted in our study.

TOTAL DURATION OF THE DISEASE

Total duration	Number of patients
6 weeks	27
7 weeks	23
8 weeks	31
9 weeks	10
10-14 weeks	09

In 81% of patients the lesions relolved within a period of 6-8 weeks.

ASSOCIATED SKIN DISEASES:

Associated skin disease	Number of patients
Acne vulgaris	06
Canites	03
Seborrhoeic Dermatitis	04
Nevus	03
Moluscum contagiosum	01
Lichen nitidus	01
Accessory Nipple	01
Eczema	01
Fibrous polyp	01
P.alba	01
Pediculosis Capitis	01
Polymorphic light eruption	01
Striae distentiae	02
Skin tag	02
Lipoma	01

Tinea versicolor	02
Tinea cruris	01
Traumatic fissure	01
Androgenic alopecia	01

Active acne vulgaris seen in 5 patients, of whom 1 had eczema also and 1 had seborrhoeic dermatitis in addition. Post Acne scar seen in 1 patient. Nevus achromicus seen in 1 patient. Epidermal nevus in 1 patient and combined hypo and hyperpigmented moles in 1 patient.

SYSTEMIC INVOLVEMENT

None of the patients had any systemic abnormality.

BIOPSY

Skin biopsy done in 27 cases. The findings were consistent with Pityriasis Rosea in all cases.

OTHER INVESTIGATIONS

1. Complete Hemogram – 3 patients had elevated ESR. 1had leucopenia.
2. Urine analysis – one patient had jaundice; 1had Hematuria.
3. Motion analysis – No ova or cyst seen in any case.
4. VDRL Test – Negative in all patients. Not done in children and adolescents less than 19 years of age.
5. Mantoux Test – Positive in 1 child (18mm).
6. Fungal scrapings – Negative in all cases.

Otolaryngologist opinion

1. Pharyngitis – 5 patients
2. Tonsillitis – 2 patients
3. Upper respiratory tract infection – 3 patients
4. Allergic rhinitis – 1 patient
5. Deviated nasal septum – 1 patient
6. Ear wax – 1 patient

Dental opinion

1. Chronic gingivitis – 6 patients
2. Chronic periodonditis – 1 patient
3. Dental caries – 7 patients

TREATMENT

Reassurance was given to all patients by explaining to them the benign self limiting non contagious nature of the disease. All are treated symptomatically. Patients with mild itching and minimal scaling were treated with emollients and anti histamines at night. Patients with moderate to severe itching were given emollients and anti histaminic at night along with a course of tablet erythromycin 250mg 4 times daily for 2 weeks.

Recalcitrant cases were treated with topical 0.1% betamethasone cream twice daily till symptomatic relief. All the patients were advised to use mild soap, soft loose clothing and to avoid irritants on the skin.

Discussion

In our study maximum incidence was noted in the age group between 11-30 years (53%). The youngest patient in the study was 3 years old and the oldest was 68 years old, which is similar to that reported in literature.[1, 2] Both sexes are equally susceptible according to some authors, while others observed a female preponderance .[1, 9] In our study, a male predominance was observed.

Pityriasis rosea has been labelled as seasonal dermatoses with a higher incidence in winter. [1,2,22] In our study, higher incidence has been similarly recorded in the winter and rainy seasons, from October to December (55%) and from June to July (22%). Case clustering also observed. But sporadic cases were seen throughout the year.

According to literature wearing of new garments may precipitate or aggravate the disease .[3] In the present analysis only 4 patients gave a history of wearing new garments prior to the onset of the disease. Other provoking factors like and psychogenic stress (2%) were also recorded in the present study. Literature reports also indicate similar findings. [47]

Prodromal illness like upper respiratory tract infection and malaise were seen in 8 patients (8%), similar to that seen in literature. [2, 3]

Bjornberg et al reported that incidence of herald patch in Pityriasis rosea was 50% - 90%. [2,47] The incidence in our patients was 82% .The location of the Herald patch has been seen to occur more frequently on the trunk (42%) followed by upper limb and the thigh, which is similar to that reported by Parsons et al and other literatures. [1, 2, 3,

The herald patch which probably represented the primary inoculation site of the virus, according to various authors was seen to occur over the covered areas of the body. [2, 50] In our study also the Herald patch was seen to occur over the covered areas of the body in majority (66%) of the patients.

The Herald patch is absent or undetected in about 20% of cases [1]. In our study also Herald patch was absent in 18% patients. Rarely the Herald patch may be double or multiple often close together [1, 2.] In our study also one patient had double Herald patches close together.

According to literature secondary lesions starts appearing after an interval of 5-15 days (few hours to 2months), in crops at 2-3 days intervals over 1 week to several weeks.[1 ,2] In the 100 patients analysed, after a varying period, ranging from 1- 30 days following the appearance of the Herald Patch, the generalised eruptions followed. They appeared in crops, intermittently at various intervals of 3-10 days.

The generalised eruptions have been found to manifest in various morphological forms like papular, vesicular, lichenoid, erythema multiforme – like, purpuric, urticarial and pustular forms .[64] In our patients only papular, lichenoid, erythema multiforme – like lesions have been observed.

According to Klauder JV Distribution of the secondary eruption over scalp, face and penis was uncommon .[2, 10] In our study, no scalp involvement was seen, but 1

patient had penile lesions, 7 patients (7%) had lesions over the face. Involvement of palms and soles is also uncommon and was seen in 3 patient (3%) in our study.

Unilateral Pityriasis Rosea has been reported in literature. [1] In our study we had a case of unilateral Pityriasis Rosea.

Champion et al observed that mucous membrane involvement is rare in Pityriasis rosea, but may involve the oral and genital mucosa. [1, 4] In our study, mucous membrane involvement was not seen.

According to literature, pruritus is mild in 25% of cases, mild to moderate in 50% cases and severe in 25% cases.[51,3] In our study 46% of patients had mild itching, 1% moderate and 2% had severe itching.

According to Nelson et al, the majority of the cases had a self limiting course and the disease lasted for 6 weeks [2, 56]. In the present analysis, 91% of patients had a self limiting course of about 6-9 weeks. The longest duration of 14 weeks was observed in 2 patients one had erythema multiforme - like Pityriasis rosea, the other one had Classical Pityriasis Rosea with palmoplantar involvement.

In most of the patients (97%) the lesions vanished without trace, which was similar to that in literature.[2] Post inflammatory hypopigmentation was seen in 3 patients (3%). However hyperpigmentation reported in literature was not seen in any of the 100 cases.[2, 3]

Nail dystrophy had been reported by Silvers and Glickman, 1974, following Pityriasis Rosea.[2, 80] In our study none of the patients showed any nail changes.

Literature reports that, Pityriasis rosea is associated with skin diseases like atopy, seborheic dermatitis and acne vulgaris.[3] In our study also association with seborheic dermatitis and acne vulgaris have been found.

Bjornberg A et al reported the incidence recurrence of Pityriasis Rosea to be 2% .There was history of recurrence in 2 cases in our study. [47]

Mantoux test was negative in 99% of the cases. This also supports the present day hypothesis of viral aetiology for Pityriasis rosea, as mantoux test is depressed in viral diseases.

SUMMARY AND CONCLUSION

In this clinical analysis, 100 cases of Pityriasis Rosea were analysed in Department of Dermatology, Coimbatore Medical College Hospital, Coimbatore 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.
- The age incidence was found to be high in adolescents and young adults.
- No familial incidence was observed.
- The diseases were more frequent in the winter and rainy months with sporadic cases occurring throughout the year.
- Factors like wearing of new garments, stress and upper respiratory tract infections were found to precipitate the disease in some cases.
- 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. Unilateral Pityriasis Rosea was also observed in this study.
- Most of the patients had complete resolution of the lesions in 6 to 9 weeks but,

erythema multiforme like Pityriasis Rosea and Pityriasis Rosea with palmoplantar involvement tends to persist for a longer duration.

- In most of the patients the lesions vanished without trace and post inflammatory hypopigmentation was noted in few patients, but none had hyperpigmentation.
- There was no complication in any of the patients.
- History of recurrence of the disease was also present in 2 cases.
- Histopathological study showed the features of non specific chronic dermatitis.

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

BIBLIOGRAPHY

1. Rooks Text Book of Dermatalogy, *7th edition ,* Volume 2, Chapter 25; 25.79 - 25. 83.
2. Fitzpatrick's Dermatology in General Medicine*, 7th edition,* Volume 1, Chapter 41.
3. Fitzpatrick's Dermatology in General Medicine*, 6th edition, Volume 1, Chapter 46.*
4. Champion RH, Burton JL, Burns DA, Breathnach SM. Viral rashes. In: *Rook's Textbook of Dermatology*. 6th ed. Oxford: Blackwell Sciences, 1998: 1092-5.
5. Truhan AP*, Pityriasis rosea. Am Fam Physician 1984; 29: 193-6.*
6. Percival GH. Pityriasis rosea. *Br J Dermatol* 1932; 44: 241-53.
7. Weiss L. Pityriasis rosea – an erythematous eruption of internal origin. JAMA 1903; 41: 20-8.
8. Parsons JM. Pityriasis rosea of Gibert. *J Am Acad Dermatol* 1987; 16:1260-1.
9. Chuang TY, Ilstrup DM, Perry HO, Kurland LT. Pityriasis rosea in Rochester,Minnesota,1969-78, *J Am Acad Dermatol* 1982;7:80-9.
10. Klauder JV. Pityriasis rosea with particular reference to its unusual manifestations. *JAMA* 1924; 82: 178-83.
11. Jacyk WK. Pityriasis rosea in Nigerians. *Int J Dermatol* 1980; 19: 397-9.
12. Jackson R. The lines of Blaschko: a review and reconsideration: Observations of

thecause of certain unusual linear conditions of the skin. *Br J Dermatol* 1976; 95: 349- 60.

13. www. icd9cm.chrisendres.com. Free online searchable **2009** ICD-9-CM.
14. www.who.int/classifications/ICD-10 online
15. Vollum DI. Pityriasis rosea in the African. *Trans St Johns Hosp Dermatol Soc* 1 973; 59: 269-71.
16. Ahmed MA. Pityriasis rosea in the Sudan. *Int J Dermatol* 1986; 25:184-5.
17. Traore A, Korsaga-Some N, Niamba P *et al.* Pityriasis rosea in secondary schools in Ouagadougou, Burkina Faso. *Ann Dermatol Venereol* 2001; 128: 605-9.
18. Spelman LJ et al,Pityriasis Rosea like eruption after bone marrow transplant. *J Am Acad Dermatol* 1994*;* 31*:348*
19. Hendricks AA, Lohr JA, Pityriasis Rosea in infancy. Arch Dermatol 1979; 115:896-7.
20. Hyatt H. Pityriasis rosea in a three month old. *Arch Pediatr* 1960; 77: 364.
21. Cohen EL. Pityriasis rosea. *Br J Dermatol* 1967; 79: 533-7.
22. Chuh AA, Albert Lee, Vijay Zawar.Pityriasis Rosea – an update. *IJDVL*, Sep-Oct 2005, Vol-71, Issue 5 ;311-314.
23. Antonio AT Chuh, Albert Lee, Nicolas Molinari. Case Clustering in Pityriasis rosea. *Arch Dermatol* 2003; 139: 489-493.
24. Parsons JM. Pityriasis rosea update: 1986. *J Am Acad Dermatol* 1986; 15: 159-

67.

25. Allen RA, Janniger CK, Schwartz RA. Pityriasis rosea. *Cutis* 1995; 56: 198-202.
26. Wile UJ. Experimental transmission of pityriasis rosea. A preliminary report. *Arch Dermatol Syph* 1927; 16: 185-8.
27. Aoshima T, Komura J, Ofuji S. Virus-like particles in the herald patch of pityriasis rosea. *Dermatologica* 1981; 162: 64-5.
28. El-Shiemy S, Nassar A, Mokhtar M, Mabrouk D. Light and electron microscopic studies of pityriasis rosea. *Int J Dermatol* 1987; 26: 237-9.
29. A G messenger et all : Case clustering in pityriasis rosea: support for role of an infective agent. *British Medical Journal* Volume 284 6 February 1982:371-372.
30. Miller TH. Pityriasis rosea: report of three cases in one family with clinical variations in two of them. *Arch Dermatol Syph* 1941; 44: 66-8.
31. Chuang TY, Perry HO, Ilstrup DM, Kurland LT. Recent upper respiratory tract infection and pityriasis rosea: a case-control study of 249 matched pairs. *Br J Dermatol* 1983; 108: 587-91.
32. McPherson A, McPherson K, Ryan T. Is pityriasis rosea an infectious disease? *Lancet* 1980; 2: 1077.
33. Sugiura H, Miyauchi H, Uehara M. Evolutionary changes of immunohistological characteristics of secondary lesions in pityriasis rosea. *Arch Dermatol Res* 1988; 280: 405-10.

34. Lever's Histopathology of the Skin, 9th edition: chapter7; 192.

35. Werner Kempf, Volker Adams. Pityriasis Rosea is not associated with HHV 7. *Arch Dermatol* Vol 135. Sep1999:1070-72.

36. Antonio AT Chuh. The association of Pityriasis Rosea with CMV, EBV, Parvovirus B19 Infections. *Eur J of Dermatology* Vol.13, Jan- Feb 2003: No.1, 25-8.

37. Hudson LD, Adelman S, Lewis CW. Pityriasis rosea. Viral. complement fixation studies. *J Am Acad Dermatol* 1981; 4: 544-6.

38. Raskin J. Possible dermatrophic virus associated with Pityriasis Rosea. *Acta Derm Venerol (Stockl*) 1968; 48:474-81.

39. John K. S Chia et al, Enterovirus infection as a possible cause of Pityriasis Rosea: demonstration by immunochemical staining. *Arch Dermatol* Vol 142, Jul 2006:942-3.

40. Antonio AT Chuh, Henry HL Chan. Prospective case control study of chlamydin, legionella, Mycoplasma Infection in patients with Pityriasis rosea. *Eur J of Dermatology* Vol.12, No.2, Mar- Apr 2002;170-3.

41. Parija M, Thappa DM. Study of role of streptococcal throat infection in pityriasis rosea. *Indian J Dermatol* 2008; 53:171-3.

42. Hosokawa H, Horio S, Takiuchi Y *et al.* Naturally occurring T lymphocytotoxic antibody in viral and related skin diseases. *Acta Derm Venereol* 1984; 64: 275-80.

43. Durusoy C, Alpsoy E, Yilmaz E. Pityriasis rosea in a patient with Behcet's disease

treated with interferon alpha 2A. *J Dermatol* 1999; 26: 225-8.

44. Tay YK, Goh CL. One-year review of pityriasis rosea at the National Skin Centre, Singapore. *Ann Acad Med Singapore* 1999; 28: 829-31.
45. Sharma PK, Yadav TP, Gautam RK *et al.* Erythromycin in pityriasis rosea: A doubleblind, placebo-controlled clinical trial. *J Am Acad Dermatol* 2000; 42: 241-4.
46. Labro MT. Anti-inflammatory activity of macrolides: a new therapeutic potential? *J Antimicrob Chemother* 1998; 41 Suppl B: 37-46.
47. Björnberg A, Hellgren L. Pityriasis rosea. A statistical, clinical and laboratory investigation of 826 patients and matched healthy controls. *Acta Derm Venereol* 1962; 42: 50.
48. De Kayser F et al, Immune mediated pathology following Hepatitis B vaccination.:Two cases of polyarteritis Nodosa and one case of Pityriasis rosea like eruption. *Clin Exp Rheumatol* 2000;18:8,.
49. Broccolo F et al: Additional evidence that pityriasis rosea is associated with reactivation of human herpesvirus-6 and -7. *J Invest Dermatol* 2005; 124:1234.
50. Abercrombie GF. Pityriasis rosea. *Proc R Soc Med* 1962; 55: 556-7.
51. Eslick GD. Atypical pityriasis rosea or psoriasis guttata? Early examination is the key to a correct diagnosis. *Int J Dermatol* 2002; 41: 788-91.
52. Andrew's Diseases of the Skin. Clinical Dermatology, *10th edition*; 2008.
53. Cavanaugh RM Jr. Pityriasis rosea in children. A review. *Clin Pediatr (Phila)*

1983; 22: 200-3.

54. Antonio AT Chuh. Rash Orientation in Pityriasis rosea-a Qualitative study. *Eur J of Dermatology* Vol.12, No.3, May- Jun 2002; 253-6.

55. Gonzalez, Robert Alan. Pityriasis Rosea : An important papulosquamous disorder. *Int J Dermatol* 2005, 44, 757-764.

56. Nelson JSN and Stone MS. Update on Selected viral exanthema. *Curr opin Pediatr* 2000; 12:359-64.

57. Imamura S, Ozaki M, Oguchi M *et al.* Atypical pityriasis rosea. *Dermatologica* 1985; 171: 474-7.

58. Friedman SJ. Pityriasis rosea with erythema multiforme-like lesions. *J Am Acad Dermatol* 1987; 17: 135-6.

59. Garcia RL. Letter: Vesicular pityriasis rosea. *Arch Dermatol* 1976; 112: 410.

60. Griffiths A. Vesicular pityriasis rosea. *Arch Dermatol* 1977; 113: 1733-4.

61. Anderson CR. Dapsone treatment in a case of vesicular pityriasis rosea. *Lancet* 1971; vol 298: 493.

62. Bari M, Cohen BA. Purpuric vesicular eruption in a 7-year-old girl. Vesicular pityriasis rosea. *Arch Dermatol* 1990; 126: 1497, 1500-1.

63. Pierson JC, Dijkstra JW, Elston DM. Purpuric pityriasis rosea. *J Am Acad Dermatol* 1993; 28: 1021.

64. Engin Sezer ,Zeynep Nurhan Saracoglu. Purpuric Pityriasis Rosea. *Int J Dermatol* 2003, 42,138-140.

65. Pringle JJ. Case presentation, section on dermatology, Royal Society of Medicine. *Br J Dermatol* 1915; 27: 309.

66. Bernardin RM, Ritter SE, Murchland MR. Papular pityriasis rosea. *Cutis* 2002; 70: 51-5.

67. Gibney MD, Leonardi CL. Acute papulosquamous eruption of the extremities demonstrating an isomorphic response.Inverse Pityriasis rosea. *Arch Dermatol* 1997;133:651-54.

68. A Chuh, V Zawar, A Lee. Atypical presentations of pityriasis rosea: case presentations *JEADV* , volume 19, issue 1, Pages 120-126.

69. Brar BK,Pall A, Gupta RR. Pityriasis Rosea Unilateralis. – a case report, IJDVL 2003 ; 69: 42-3.

70. Ahmed I, Charles-Holmes R. Localized pityriasis rosea. *Clin Exp Dermatol* 2000; 25: 624-6.

71. I Bukari ,Dermatology Online Journal, 2005 ; 11 (1); 27.

72. Kay MH, Rapini RP, Fritz KA. Oral lesions in pityriasis rosea. *Arch Dermatol* 1985; 121: 1449-51.

73. Konstantopoulos K, Papadogianni A, Dimopoulou M *et al.* Pityriasis rosea associated with imatinib (STI571, Gleevec). *Dermatology* 2002; 205: 172-3.

74. Brazzelli MD,Prestinari MD. Pityriasis rosea- like eruption during treatment with Imatinib mesylate: *J Am* Acad *Dermatol*, Nov 2005, Vol 53, Number 5; S240-3.

75. Wilkin JK, Kirkendall WM. Pityriasis rosea-like rash from captopril. *Arch*

Dermatol 1982; 118: 186-7.

76. Drago et al.Use of high dose acyclovir in Pityriasis Rosea.*J Am Acad Dermatol2006;* 54:82.
77. Buckley C. Pityriasis rosea-like eruption in a patient receiving omeprazole. *Br J Dermatol* 1996; 135: 660-1.
78. Gupta AK, Lynde CW, Lauzon GJ *et al.* Cutaneous adverse effects associated with terbinafine therapy: 10 case reports and a review of the literature. *Br J Dermatol* 1998; 138: 529-32.
79. Olumide Y. Pityriasis rosea in Lagos. *Int J Dermatol* 1987; 26: 234-6.
80. Silvers SH,Glickman FS:Pityriasis rosea followed by Nail Dystrophy; *Arch Dermatol* 1964,Jul ;90:31.
81. Hurly HJ: Papulosquamous eruption and exfoliative dermatitis .*Dermatogy* Vol-1, 1975; 427-431.
82. Fox BJ, Odom RB. Papulosquamous diseases: a review. *J Am Acad Dermatol* 1985; 12:597-624.
83. Stefanato, Catherine M. M.D et al: Gianotti-Crosti Syndrome Presenting as Lichenoid Dermatitis *The American Journal of Dermatopathology* Volume 22(2), April 2000, pp 162-165.
84. Beers MH, Berkow R. The Merck manual of diagnosis and therapy. 17th edition. 1999; Chapter 117. Available from: URL: http://www.merck.com/pubs/mmanual/.
85. Bunch LW, Tilley JC .Pityriasis rosea; histologic and serologic study .*Arch*

Dermatol 1961; 84: 79-86.

86. Okamoto H, Imamura S, Aoshima T *et al.* Dyskeratotic degeneration of epidermal cells in pityriasis rosea: light and electron microscopic studies. *Br J Dermatol* 1982; 107: 189-94.
87. Tagami H, Uehara M. Multinucleated Epidermal Giant cells in inflammatory skin disease. *Arch Dermatol* 1981; 117:23-25.
88. Stern JK, Wolf JE Jr, Rosen T . Focal acantholytic diskeratosis in Pityriasis Rosea .*Arch Dermatol* 1979; 115: 497.
89. Takaki Y, Miyazaki H. Cytolytic degeneration of keratinocytes adjacent to Langerhans cells in pityriasis rosea (Gibert). *Acta Derm Venerol* 1976; 56: 99-103.
90. Morel P, Revillard JP, Nicolas JF, Wijdenes J. CD4 antibody therapy in chronic inflammatory dermatological diseases. *Immunol Ser* 1993; 59: 271-6.
91. Abdel-Hafez K, Deyab Z. Pityriasis rosea. An immunologic study. *Int J Dermatol* 1987; 26: 231-3.
92. Antonio AT Chuh, Henry HL Chan. Effects on quality of life in patients with Pityriasis rosea: Is it associated with Rash severity. *Int J of Dermatol* 2005; 44: 372-377.
93. Arndt KA, Paul BS, Stern RS, Parrish JA. Treatment of pityriasis rosea with UV radiation. *Arch Dermatol* 1983; 119: 381-2.
94. Antonio AT Chuh, Henry HL Chan, Vijay Zawar. Is HHV-7 the causative agent of Pityriasis rosea- a critical review. *IJD* 2004; 43: 870-875.
95. *Moschella and Hurley Dermatology*, 3rd edn, Volume 1, chapter 26; 624.

PROFORMA

O.P. No: DATE

Sl.No:

Name

Sex

Age

Address

Occupation

Income

Complaints Duration

1.

2.

3.

Present history

Precipitating factors

Prodromal symptoms Duration

Past history

Family history

General examination

Dermatological examination

Herald patch No Size Site

Secondary eruptions Morphology Distribution

Palms/Soles

Scalp

Hair/Nail/Mucous membrane

Associated cutaneous findings

Lymphadenopathy Group Distribution

Systemic examination

Investigations

Blood- TC

DC

ESR

Urine examination

Motion examination

Blood VDRL test

Mantoux test

Scraping for fungus

Biopsy

ENT examination

Dental examination

Treatment given

Total duration

Follow up

1st week

2nd week

3rd week

4th week

5th week

6th week

7th week

8th week

9th week

10th week

11th week

12th week

Sequelae

Complications

Clinical photograph

Comments

Key to Master chart

- 1˚ to 2˚- Time interval between primary and secondary lesions.
- URI - Upper respiratory tract infection.
- J- Jaundice.
- UL – Upper limb.
- LL – Lower limb.
- DL- Distal limb.
- EMF - Erythema multiforme .
- And.alo – Androgenic alopecia.
- Seb.dermatitis – Seborheic dermatitis.
- Hypo &hyper pig.mole – Hypo & hyper pigmented mole.
- P.alba – Pityriasis alba.
- Epi.nevus –Epidermal nevus.
- T.cruris – Tinea cruris.
- ESR- Erythrocyte sedimentation rate.
- P.R – Pityriasi rosea.
- Ch.pharyngitis – Chronic pharyngitis.
- D.C – Dental caries.
- Ch.periodonditis – Chronic periodonditis.
    - Hypo pigm – Hypo pigmentation.