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Original Research Article

A Study of Psoriasis Histopathological Analysis of Lesional and Perilesional Skin Biopsy

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Abstract: Psoriasis is defined as a common, genetically determined, inflammatory and proliferative disease of skin, the most characteristic lesions consisting of chronic, sharply demarcated, dull-red, scaly plaques, particularly on the extensor prominences and in the scalp. The prevalence of Psoriasis in Western Europe and Scandinavia is between about 1.5 and 3%. The low prevalence of Psoriasis in oriental people is now well-recognized. It is commoner in Indians than in Chinese or Malays. Female tend to develop Psoriasis earlier than males. This study ware undertaken to characterize histopathological features of the lesional and perilesional skin in the psoriatic skin biopsy and to evaluate the epidermal and dermal changes in psoriasis this is a prospective analytical study at Osmania General Hospital, Hyderabad for a period of 24months and total 100 cases formed the study group. Punch / Incisional biopsies were obtained from lesional and perilesional skin, routine processing done and H & E stain is used for staining. Itching and scaly, erythomatous plaque is most common clinical presentation. Age incidence – 8-70yrs and M: F ratio was 1.6: 1. Chronic plaque psoriasis is the most common variant. Lesional epidermal changes were acanthosis with parakeratosis with regular elongations and thickening in lower portion of rete ridges. Micromunro abscess in 39% and Kogoj abscess in 23% of cases. Perilesionally epidermal changes – mild acanthosis, parakeratosis, dilated dermal capillaries, mild dermal edema, and perivascular mononuclear infiltration was noticed most of the cases.
Keywords: Psoriasis, Incisional biopsy, perilesional skin, scalpy plaques, Micromunro abscess

INTRODUCTION:

Psoriasis is a chronic, autoimmune, and complex genetic disorder that affects 2~3% of the European population. Psoriatic skin has symptoms of inflammation and raised and scaly lesions [1]. The prevalence of Psoriasis in western Europe and Scandinavia is between about 1.5 and 3%. The low prevalence of Psoriasis in oriental people is now well-recognized [2]. It is commoner in Indians than in Chinese or Malays [3]. Female tend to develop Psoriasis earlier than males [4] .The prevalence of photosensitivity in psoriasis was estimated at 5.5% [5].

In psoriasis lesion great acceleration of transit time of cells from basal layer to upper most row of squamous cell layer. 53 days in normal epidermis to seven days in active psoriatic lesions. In other study 28 it was shown that germinative cell cycle time in normal 200 hrs and in psoriatic epidermis 100 hrs two fold rather than 8 fold acceleration [6]. Strong support of genetic transmission found by Elder *et al.;* calculated risk ratios a multilocus model of inheritance by reanalyzed data of population studies of Lonholt [7] and hellegren[4].

Brandrup *et al.;* found the heritability of psoriasis in homozygous twins to be greater than 90% [8]. Another study of twins demonstrated a concordance 70% - with monozygotic, 23% dizygotic [9]. Empiric support for a polygenic mode of inheritance comes from a number of observations for eg. Incidence in families and twins associated with various HLA antigen phenotypes [10].

A strong association exist between psoriatic phenotype and HLA CW6 locus (relative risk =11) the prevalence of this gene in patients with psoriasis is 70% compared with normal 10% [7]. The koebner reaction usually occurs 7 to 14 days after injury and reported incidence has varied between 38 and 76% of patients

with psoriasis. If psoriasis occurs at one sight of injury it occurs at all sight of injury [11].

Immunological factors play very important role in pathogenesis of psoriasis. It has been shown that T lymphocytes predominant in inflammatory infiltrate [12]. Psoriasis may be divided into - Psoriasis vulgaris, generalized pustular Psoriasis, Localized pustular Psoriasis, Flexural (inverse) Psoriasis, Uncommon variants and atypical forms

Psoriasis vulgaris is common chronic inflammatory skin disorder that affects approximately 1.5-2% in western countries seen in all age groups. An acute variant, guttate or eruptive Psoriasis characterized by abrupt eruption of small lesions associated with group-A beta hemolytic streptococcal infection [13] .Nail involvement is common, 30% of the cases pitting is the commonest change resulting from focal involvement of nail matrix [14].

Figurated lesions are frequently seen in sub acute or chronic forms of generalized pustular Psoriasis [15]. Psoriatic Arthritis characteristically involves terminal interphalageal joints and usually associated with nail dystrophy [16].

The histopathological picture of psoriasis vulgaris varies considerably with stage of lesion and usually diagnostic only in early, scanning papules and near the margin of advancing plaque. The earliest pin head sized macules are smooth surfaced papules show subtle histologic picture which preponderance of dermal change [17].

At first, capillary dilation and edema in papillary dermis with lymphotic infiltration surrounding the capillaries. The mounds of parakeratosis with neutrophils represent early manifestation of Munro micro abscess [18].

In fully developed lesions of psoriasis as best seen at the margin of enlarging plaques the histological picture characterized by acanthosis with regular elongation of rete ridges with thickening in their lower portion. Thinning of supra papillary epidermis with diminished to absent granular layer, Confluent parakeratosis, the presence [19].

METERIAL & METHODS:

This is a prospective analytical study of lesional and perilesional analysis of psoriasis at Osmania General Hospital, Hyderabad for a period of 24 months. 100 cases formed the study group. Age, sex, duration of illness, body surface area and routine lab investigations analyzed. Punch / Incisional biopsies were obtained from lesional and perilesional skin, routine processing, H & E stained sections evaluated for epidermal and dermal changes in psoriasis.

RESULTS:

The youngest patient was 8 years old child. The oldest was 70 years. The highest frequency of cases was seen in 31-40 age group 25%, less frequency found in age group 1-10 years 6% and > 60 years only 2% of cases. Of the 100 patients 61 were males, 39 were females. Male: female=1.6:1. Most of the biopsies taken from the lower limb 49% followed by upper limb 31%, Abdomen 8%, back 7% , buttock 2% and from chest, chin, prepuce each 1 biopsy.

Majority of patients came with history of duration between 6 to 9 months 31% and one year above duration are 8%. The common symptom was scaly plaque 86% followed by itching 72%, erythematous plaque 64%, auspitz sign in 61%, koebner phenomenon in 52%, nail, joint involvement and oozing also seen.(Fig-1)

Routine investigations done-ESR raised in 52%, Hemoglobin less than 10grams in 27%. 4 cases were diabetic with raised sugar levels. BT,CT and platelet count is within normal range.HIV test is suspected in 18 cases and found all are negative. High serum uric acid levels were seen in 24% of cases, ASO titer done in 22 cases and found positive in two cases.

Clinically of all chronic plaque psoriasis were the commonest of 73% followed by guttate, palmoplantar, and erythodermic and psoriasis form dermatitis. In most of the biopsies marked acanthosis in 59% followed by moderate and mild acanthosis. Continuous parakeratosis in 54%, with intermittent and mild to none parakeratosis. The granular layer is absent in majority of cases 58% followed by diminished 31% and no loss of granular layer in 11%. Micromunro abscesses were present in 39%, Kogoj abscesses seen in layers of spinosum in 23%. Out of 100 cases moderate dilatation of capillaries was seen in 48%, perivascular mononuclear inflammatory infiltrate found markedly increased in 51%.(Fig-2)

In perilesional skin only in 8 cases found mild acanthotic change. Intermittent parakeratosis seen in 12.5%, diminished granular layer seen in 10%, irregular elongation of rete ridges seen in 13 cases. In perilesional skin most are showing dilated dermal capillaries in papillary dermis 57.5%, mild edema 52.5%, and perivascular mononuclear infiltration in 13 cases out of 40 cases.

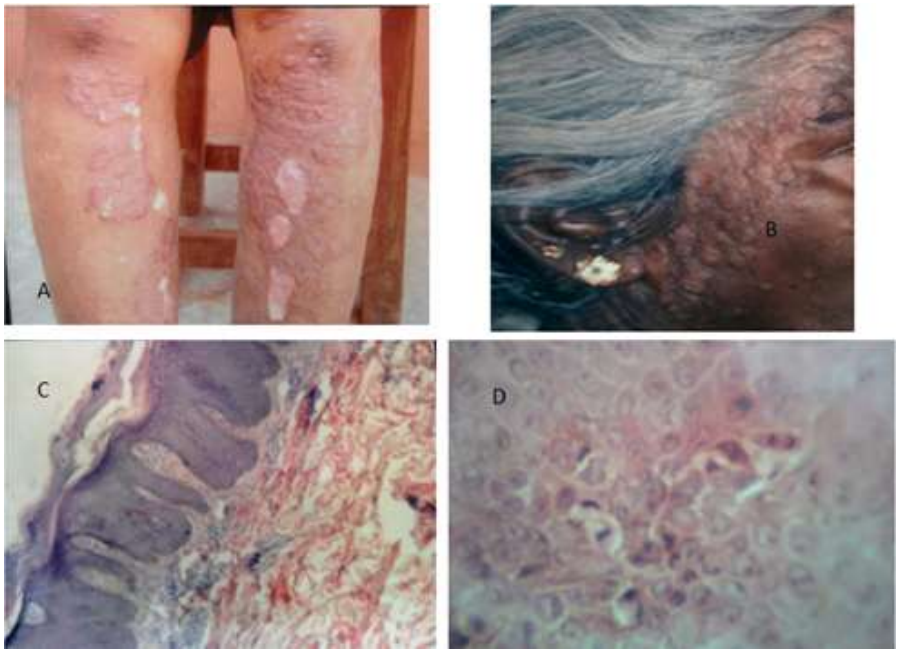

Fig:1-A.Chronic plaque psoriasis-leg, B.face and scalp, C.10X regular elongated rete ridges, munro micro abscesses, dermal inflammatory infiltrate, D.40Xincreased mitotic figures in supra basal layer.

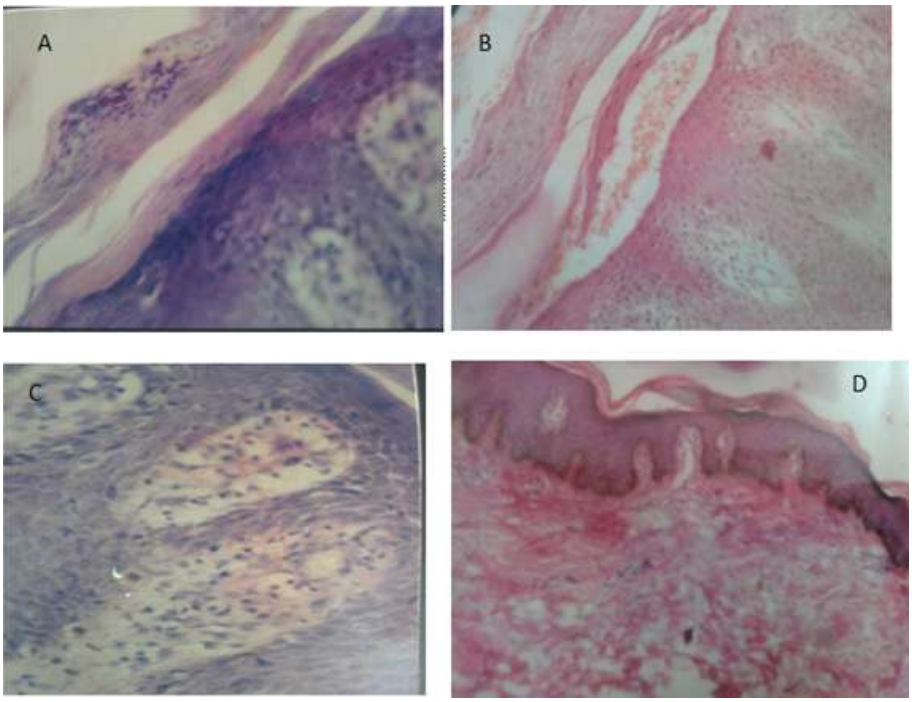

Fig 2: A. 40X showing mounds of parakeratosis with Munro micro abscesses, B. 10X showing hemorrhage in the stratum corneum (Auspitz sign), C. 40X dilated tortuous capillaries in dermal papillae. D. shows lesional perilesional skin-epidermal and dermal changes

DISCUSSION:

A critical analysis histopathological feature of psoriasis in lesional, perilesional areas is done. Clinical parameters of psoriasis were analyzed. According to Rook 1998, early psoriatic changes present the initial pathology in dermis of the skin [20]. The changes noted are Vasodilation, papillary oedema and leukocytic infiltrates appear to precede epidermal changes.

The microscopic examination of Psoriasis is a direct means of confirming or making the diagnosis. Spongiform pustules of Kogoj and Munro micro abscesses are truly diagnostic of Psoriasis and in their absence the diagnosis can be made with certainly on a histologic basis. The rete ridges show regular elongation, increase number of mitosis in lesional areas

than uninvolved skin. Rete ridges show slender in upper portion but clubbing in their lower portion, suprapapillary epidermis thinning. Dermal papillae show edema, dilated and tortous capillaries. In the dermis there was increased vascularity perivascular mononuclear inflammatory infiltrate [19].

Incidence of Psoriasis in medical college hospital Rohtak, Haryana is 0.8% of all dermatological patients. Lal et al from Amritsar found an incidence of 1.25%, Mehta et al from Bombay found an incidence of 1.5% and Ambasy et al from Trivendrum an incidence of 5.6% Yui Yips, the prevalence of Psoriasis in western Europe and scandinaria in between about 1.5 and 3%[2].

In present study showed an incidence of 1.7% of all dermatological patients in Department of Dermatology OGH, Hyderabad. The present study showed youngest was 8 years girl and below 10 years age group. Male and female sex ratio showed equal ratio. 11-20 years age group showed mild female predominance.

According to Henseler T *et al.;* evaluation of the age of onset revealed two peaks an early one at 16-22 years and a later one at 57-60 years [21]. In this present study the peak age incidence was 31-40 years (25%) followed by 21-30 year age group (22%) then 41-50 years (18%) and in above 60 years of the age occurrence of cases was 2%.

Hellgren L. showed the incidence of psoriasis in adult men and women is usually reported to be about equal [4]. Lal *et al.;* from Amritsar Punjab showed M: F ratio was 2:1. In present study showed mild make predominance M: F was 1.6:1.

According to Levers site the scalp, sacral region and extensor of extremities are commonly involved. In present study majority of patients presented the lesions over lower and upper extremities [19]. Farber EM, Nall ML, Walson W: 1974- reported incidence of koebner phenomenon has varied between 38 and 76% of patients with psoriasis [14].

In the present study koebner phenomenon was positive in 52% cases it is in the same range as Farber *et al.;* According to Fitz Patrick's 1999 common laboratory abnormalities were elevated uric acid, mild anemia, increased sedimentation rate [22] . In present study laboratory investigations showed ESR raised in 52%. Hemoglobin was less than 10 gms in 27% and CBP showed normocytic hypochromic in 23%.

Teffer *et al.* showed the role of streptococcal infection in the initiation of guttate psoriasis. The present study showed ASO titre positive in 2 cases out of 22 cases [13].

According to Levers, the thickness of granular layer in normal skin is proportional to thickness of the Horney layer. Granular layer is only 1-3 cell layers thick. In psoriasis there is often an inverse relationship between the presence and thickness of granular layer and parakeratois [19]. In this study showed granular layer is absent in majority of cases 58%, diminished granular layer in 31% and no loss in 11% of cases.

Van Scott EJ, Ekle TW, Arch dermatologists 1963 showed considerable lonthening of the basal layer due to elongation of rete ridges in increase in number of mitosis calculated to be 27% times to number of mitosis in uninvolved skin [23]. Showed mitotic figures 1 per 1 rete ridges in 41% and also increased numbers in supra basal layer in 17% markedly, moderate increase in 12%, mild increase in 9% of cases, the remaining not showed any mitotic figures. mounds of parakeratosis with neutrophils represent the earliest manifestation of munromicorabscesses [18]. A relatively mild inflammatory infiltrate present in the upper dermis and papillae it consist of lymphocytes, except in early lesions, in which neutrophils are also present [24].

Gerald G. Kruiger, assessment by skin transplant to congenitally athymic (nude) mice, Nude mice were grafted with one of three types of skin. Normal human skin or skin form psoriatic subjects involved (or) involved with disease [25]. Most of common histologic observations in the epidermis involved with psoriasis are - regular acanthosis, parakeratpsis and a decrease in granular layer.

Skin from normals none had parakerotosis, similar results were noted in the uninvolved skin. This contacts with parakerotosis in 64% in involved in skin. The perilsional skin biopsies in the present study showed 20% miles acanthotic change. Where as in lesional skin showed acathosis in 84% of cases it is similar to Gerald *et al.;* observations.

In present study perilesional area showed 12.5% intermittent parakerotosis whereas lesional area showed 78% intermittent to continuous parakerotosis, diminished granular layer seen in 10% of perilesional biopsies. Irregularly elongated rete ridges are seen in 13 cases. In perilesional skin most showed dilated dermal capillaries in papillary dermis in 57.5%, followed by mild dermal edema in dermis in 52.5% of cases. Perivascular mononuclear infiltration is seen in 4 cases.

According to Gerald G. Kruiger 1981: showed in normal skin aconthosis in 7% uninvolved specimens had minimal acathosis in 41% for involved lesions 88% showed acathosis [25]. The common change seen in perilesional skin as dermal edema, dilated dermal capillaries and perivascular mononuclear inflammatory

infiltration. The perilesional skin showed dermal changes were prominent then epidermal changes, whereas in psoriatic lesional skin epidermal changes were dominant than dermal changes.

SUMMARY

Prospective analytical study of lesional and perilesional skin of 100 cases of psoriasis undertaken at OGH for a period of 24 months. Evaluated with parameters like age, sex, site, BSA. Itching and scaly, erythromatous plaque is most common clinical presentation. Lesional epidermal changes were Acanthosis with parokeratosis with regular elongations and thickening in lower portion of rete ridges. Micromunro abscess in 39% and Kogoj abscess in 23% of cases. Perilesionally epidermal changes – mild acanthosis, parakeratosis, and Regular elongation rete ridges were not seen. Dilated dermal capillaries, mild dermal edema, perivascular mononuclear infiltration was noticed most of the cases. The perilesional skin showed dermal changes were prominent then epidermal changes, whereas in psoriatic lesional skin epidermal changes were dominant than dermal changes.

CONCLUSION:

The perilesional changes observed in our study may shift our focus "Psorasis as Keratinocyte disorder" to a inflammatory disorder with immuno dysregulation as underlying cause.

REFERENCES:

1. Bhalerao J, Bowcock AM; The genetics of psoriasis: a complex disorder of the skin and immune system.Hum Mol Genet **7**: 1537–1545, 1998
2. Yui Yips S; The prevalence of psoriasis in the mongoloid race. J Am. Acad. Dermatol 1984: 10: 965 - 5.
3. Rajan VS, Thiru Moorthy T, Giam YC *et al.;* psoriasis: the Singapore experience. In: Farber EM, Cox AJ, Nall L, Jacobs PH, Eds. psoriasis: proceedings of the 3rd international symposium. New York: Grune and Stratton, 1982: 407-8.
4. Hellen L; psoriasis. The prevalence in sex, age and occupational groups in total populations in Sweden. Morphology, Inheritence and association with other skin and Rheumatic diseases. Skockholm; Almqvist and wiksell, 1967; 19 -53.
5. Ros AM, Eklund G; Photo sensitive psoriasis.J.Am.Acad Dermatol 1987;17:752-8
6. Goeckerman WH, O'Leary PA; Erythroderma psoriaticum: A review of twenty two cases. JAMA 1932; 99: 2102-2105.
7. Elder JT, Nair RP.Guo SW, Henseler T, Christophers E, Voorhees J.J; The genetics of psoriasis.Arch Dermatol. 1994; 130(2):216-224.
8. I-Iansen H.E; psoriasis in monozygotic twin's .Variations in expression in individuals with identical genetic constitution.Acta Derm Venereol (Stockh) 1982; 62:229-236.
9. Faber EM. Nall ML, Watson W; Natural history of psoriasis in 61 twin pairs.Arch Dermatol, 1974; 109:207.
10. Russell TJ, Schultes LM, Kuban DJ; Histocompatability (HL-A) antigens associated with psoriasis N Engl.N.Med. 1972; 287:738-740.
11. Eyre RW, Kruger GG; The Koebner response in psoriasis .In: Roenigk HH, Maibach HI, eds. Psoriasis. New York: Marcel Dekker, 1984; 15-16.
12. Cooper KD; Psoriasis: Leucocytes and cytokines .Dermatol Clin 1990; 8:737.
13. Teffer NR, Chalmers RJG, Whale K, Colman G; The role of streptococcal infection in the initiation of guttate psoriasis. Arch Dermatol 1992; 128: 39.
14. Faber EM. Nall ML, Watson W; Natural history of psoriasis in 5600 patients. Dermatologica, 1974; 148(1): 1-18.
15. Baker H. Pustular psoriasis. Dermatol Clin 1984; 2: 455.
16. BakerII, Ryan TJ, Generalized pustular psoriasis. Br. J. Dermatol 1968; 80: 771.
17. Braum- Falco O, Christophers E. Structural aspects of initial psoriatic lesions .Arch Dermatol Forsch 1974; 252:95.
18. Ragaz A., Ackerman AB: Evolution, maturation and regression of lesions of psoriasis. AM J. Dermatopathol. 1973; 108 : 687.
19. Sonia Tousant, HidakoKamino, Non infectious erythematous papular and squamous disease of the skin. in Lever's Histopathology of the skin 8th ed.Elder Devid,Elenistsas Rosalie,Johnson Bernett .Lippincott-Raven publishers Phyladelphhia 1997;151-184
20. Rook, Wilkinson, Ebling: TExtbook of Dermatology 6th Ed. 1998.
21. Henseler T, Christophers E. psoriasis of early and late onset. Characterization of two types of psoriasis vulgaris J. Am. Acad. Dermatol. 1985.
22. Fitz patricks - Dermatology in general medicine 5th Ed. 1999.
23. Van Scott EJ, Ekel TW, and Kinetics of hyperplasia in psoriasis. Arch Dermatol 1963; 88: 373.
24. Pinkus H,Mehregan AH. The primary histologic lesion of seborrheic dermatitis and psoriasis. J Invest Dermatol 1966; 46 : 109.
25. Gerald g. Krueger, donald a. Chambers, and jane shelby, Involved and Uninvolved Skin from Psoriatic Subjects: Are They Equally Diseased? Assessment by skin transplanted to congenitally athymic (nude) mice. J. Clin. Invest. Volume 68 December 1981; 1548-1557