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Original Article

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Role of Prolotherapy in Wound Bed Preparation

Imran Pathan[1], Ravi Kumar Chittoria[2], Saurabh Gupta[3], Chirra Likhitha Reddy[4], Padmalakshmi Bharathi Mohan[5], Shijina K[6]

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Abstract

Wound is a common problem following burn, trauma or infection. There are various methods to limit the infection and to cover the raw area. But there is no well-established method that accelerates the wound healing rate. Prolotherapy is a technique that involves injecting some irritant locally in the wound that is claimed to hasten the healing. This article highlights the role of prolotherapy in wound bed preparation.

Keywords: Prolotherapy; Wound; Management.

Introduction

Wound is a common problem encountered by plastic surgeon. Many methods are there with varying success.[1] Large wounds often require graft or flap for wound coverage, but for this wound bed should be prepared first. Various modalities exist that helps in enhancing the wound bed preparation.

Prolotherapy is one of the recent therapeutic strategies for wound healing. Prolotherapy is a procedure in which an irritant is injected into wound that instigating an inflammatory reaction, thought to promote healing of wound.[2] The most common prolotherapy agent used in clinical practice is dextrose, with concentrations ranging from 12.5% to 25%. Dextrose is considered to be an ideal proliferant because it is water soluble, a normal constituent of blood chemistry, and can be injected safely into multiple areas and in large quantity. Hypertonic dextrose solutions act by dehydrating cells at the injection site, leading to local tissue trauma, which in turn attracts granulocytes and macrophages and promotes healing In review of literature we have seen very few Indian studies on prolotherapy in wound management. We share our experience on prolotherapy using Dextrose 25%, in wound management.

Methodology

This is case report of use of prolotherapy in post burn raw area. This study was conducted in a tertiary care hospital in 2019. The patient was 20 year female with post thermal burn raw area on thigh. Patient was thoroughly investigated. Wound tissue culture was sent and appropriate antibiotic therapy was given. Regular cleaning and dressing was done, but wound was not showing any good sign of healing. To hasten the wound bed preparation decision was made to give trial of prolotherapy.

Dextrose 25% solution was used as agent for prolotherapy. It was spread evenly on to the wound followed by gauze dressing. Repeated session of prolotherapy was given every three days. After 4 session of prolotherapy wound bed was prepared and skin grafting was done. There was almost complete uptake of graft and in follow-up after 6 weeks, the scar was well settled and of good quality.

Result

After 4 session of prolotherapy over two weeks period, the wound bed was prepared and grafting was done. Graft uptake was satisfactory. No adverse local or systemic effect was noted with the use of prolotherapy.

Discussion

The spectrum of modalities available to manage a wound is very wide. Conveniently it can be grouped into four categories – conventional therapy, novel therapy, reconstructive therapy and cell based. Conventional therapies include – conventional dressings with or without topical application of anti-microbial agents, growth factors; various biological dressings such as silver and alginate; hyperbaric oxygen etc. Novel therapies include the use of platelet-rich plasma, negative pressure wound therapy (NPWT), and skin substitutes. These are minimally invasive with much better healing efficacy than conventional therapies. Reconstructive therapy, such as skin and flap grafting, are invasive and damage the normal tissue also. Cell based therapy is rapidly emerging as a part of wound management, but is seldom used alone. These cell can be harvested from bone marrow or adipose tissue.

The term prolotherapy was coined by Dr. George Hackett in 1956. This word is derived from the Latin word proles meaning offspring or progeny and the English word–therapy. It involves injecting an irritant substance (such as dextrose) into aligament or tendon to promote the growth of new tissue. Multiple agents are used in prolotherapy, some classified as irritants (such as phenol), some as chemo attractants (commonly sodium morrhuate), and others as osmotic agents (commonly dextrose).

Although the exact mechanism of prolotherapy is not clear, proponents of the technique believe

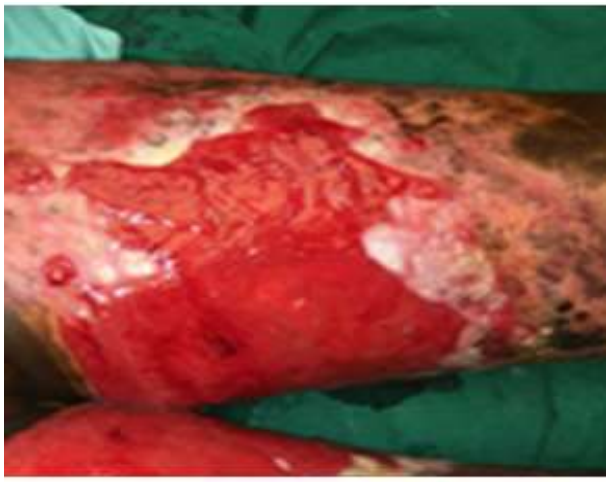

Fig. 1: Raw area on thigh

Fig. 2: Dextrose 25% used for prolotherapy

Fig. 3: Skin grafting after the wound bed preparation

Fig. 4: 6th week Post-operative

that the injection of hypertonic dextrose causes cell dehydration and osmotic rupture at the injection site that leads to local tissue injury that subsequently induces granulocyte and macrophage migration to the site, with release of the growth factors and collagen deposition.[3] In vitro studies have shown that even concentrations as low as 5% dextrose have resulted in production of a number of growth factors critical for tissue repair. Some of these growth factors include PDGF, TGF–b, EGF, b–FGF, IGF–1, and CTGF.[4]

In vitro studies have shown that cultivation of cells in high glucose culture medium can increase PDGF expression. PDGF has multiple pro-reparative effects in skin wounds, including promotion of angiogenesis, fibroblast proliferation, extracellular production. TGF–b expression is also upregulated by high glucose.[5,6] TGF–b is involved in all steps of wound healing including inflammation, angiogenesis, fibroblast proliferation, collagen synthesis, matrix deposition, and remodeling, and wound reepithelialization. Other growth factors upregulated by high glucose include EGF, b–FGF, IGF and CTGF, all having multiple proreparative functions and improves healing in some animal wound models of impaired healing.

Some studies on prolotherapy suggest that there are direct effects on collagen synthesis.[7] A few studies demonstrateup-regulation of matrix in response to dextrose prolotherapy or in vitro cultivation with high concentrations of glucose. Collagen expression is increased after exposure of patellar tendon fibroblasts to the prolotherapyagents dextrose and thus may contribute to tissue regeneration within a cutaneous wound. Collagen type Isynthesis is also increased in high-glucose cultivation of renal fibroblasts, in a TGF–b–mediatedpathway.[8] Changes in the cartilage matrix proteinaggrecan is reported in chondrocytes cultured inhigh glucose, and in patients who have received intra articular injections of 12.5% dextrose.[4-8]

Conclusion

In this study we found that prolotherapy has role in healing of the wound and the wound heals at faster rate. The resultant scar was also of better quality. But since it is a single case study, definite conclusion cannot be made. Large randomized control trials are required to confirm the efficacy of prolotherapy in wound healing.

Conflicts of Interest: None.

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Original Article

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Study of Nail Diseases in South Karnataka Population

Srinivas K[1], Nanda K[2]

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Abstract

Background: 150 patients of both sexes aged between 18 to 65 years were studied for different nail diseases to rule out the cause.

Method: The patients who were visiting to OPD of Skin with complaint of nails were studied. To confirm the onychomycosis nail samples were processed by direct microscopy of the KOH mount followed by two sets of culture on Sabouraud's Dextrose Agar and incubated at 25ºC and 37ºC and were examined once a week for a period of 4-6 weeks to look for fungal causative agent. The routine Blood examination were carried out to rule out Anaemia (Hb%), Diabetic mellitus, Esinophilic count.

Results: 58(38.6%) had onychomycosis, 11(7.3%) pseudomonas nail infection, 13(8.6%) acute paronychia, 16(10.6%) periungal verrucae, 4(2.6%) of psoriasis, 7(4.6%) lichen planus, 2(1.3%) alopecia Areata, 5(3.5%) chronic paronychia, 6(4%) longitudinal melanonychia, 7(4.6%) longitudinal Erythronychia, 9(6%) subungualexostosis, 12(8%) digital nail cyst.

Conclusion: This pragmatic approach towards nail diseases at different ages, in both sexes will certainly help the physician, dermatologist to treat efficiently because most of the nail diseases are neoplastic and life threatening.

Keywords: Nail; Onychomycosis; Paronychia; Subungual tumor, Melanonychia.

Introduction

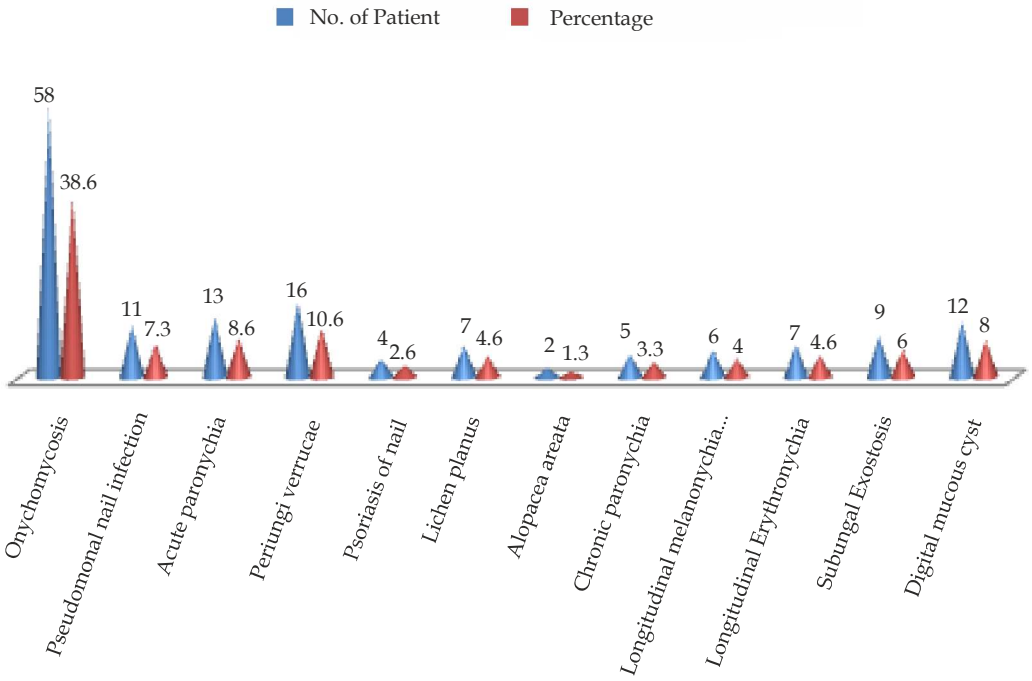
Nail changes are common presenting complaints of patients. Clinical evaluation can be challenging, and the differential diagnosis at times is broad However, familiarity with several common diagnosis and their appropriate evaluation can improve care of the patient with nail complaint.[1] Inflammatory non-infectious diseases of the nail are not uncommon. The nail changes may look different in the same diseases but also very much alike in various different nail disorders depending on which particular structure of the nail apparatus is involved.[2] of all skin diseases onychomycosis (fungal infection) common disorder of nail plate, accounting for half of the reported nail diseases.[3-4] Psoriasis and lichen planus involved frequently nail, adjacent skin and mucous membranes. Most of the auto-immune dermatosis may also affect the nails but changes not clinically specific. Hence attempt was made to evaluate the various nail diseases in different age groups.

Observation and Results

Diseases of the nail: Diseases of the nail 58(38.6%) onychomycosis, 11(7.3%) had pseudomonal nail infection, 13(8.6%) acute paronychia, 16(10.6%) periungual verrucae, 4(2.6%) psoriasis of nail, 7(4.6%) lichen planus, 2(1.3%) alopecia areata, 5(3.3%) chronic paronychia, 6(4%) longitudinal melanonychia (neoplasm), 7(4.6%) longitudinal Erythronychia, 9(6%) subungual Exostosis, 12(8%) digital mucous cyst.

Table 1: Diseases of the Nail.
No of Patients 150

Sl No.	Particulars	No. of Patient	Percentage
1	Onychomycosis	58	38.6
2	Pseudomonal nail infection	11	7.3
3	Acute paronychia	13	8.6
4	Periungual verrucae	16	10.6
5	Psoriasis of nail	4	2.6
6	Lichen planus	7	4.6
7	Alopaceaareata	2	1.3
8	Chronic paronychia	5	3.3
9	Longitudinal melanonychia (meoplasm)	6	4
10	Longitudinal Erythronychia	7	4.6
11	Subungual Exostosis	9	6
12	Digital mucous cyst	12	8



Material and Method

150 patients of both sexes aged between 18 to 65 years. Who were regularly visiting skin and VD OPD of Dr. B. R. Ambedkar Medical College, Hospital Kadugondanahalli, Bangalore-560045 (Karnataka), were selected for study.

Inclusion Method: The patients belonged to middle socio- economic status and majority of them were labors exposed to dust, chemicals etc. Were selected for study.

Method: To confirm the onychomycosis nail samples were processed by direct microscopy of the KOH mount followed by two sets of culture on Sabouraud's Dextrose Agar and incubated at 25°C and 37°C and were examined once a week for a period of 4–6 weeks to look for fungal causative agent. The routine Blood examination was carried out to rule out Aneamia (Hb%), Diabetis mellitus, Eosinophilic count. The duration of study was about two years (2008–2010).

Exclusion Method: The patients having neurological and cardiovascular complication associated with nail diseases were excluded from the study.

Statistical Analysis: The patients having different clinical manifestation of Nail diseases were classified with percentage. The ratio of male and females were 3:1.

Discussion

In the present study of diseases of nail at different ages in South Karnataka population of both sexes-58 (38.6%) onychomycosis, 11 (7.3%) pseudomonal nail infection, 13 (8.6%) acute paronychia, 4 (2.6%) psoriasis of nail, 7 (4.6%) lichen planus, 2 (1.3%) alopecia areata, 5 (3.3%) chronic paronychia, 6(4%) longitudinal melanonychia (neoplasm), 7 (4.6%) longitudinal Erythronychia, 9(6%) subungual Exostosis, 12 (8%) digital mucous cyst (Table 1). These findings were more or less enlargement with previous studies.[5-7]

Nail diseases or deformity of the nail, although the nail is a skin appendage, have distinct classification as they have their own signs and symptoms which may relate to other medical conditions. Hence nail shows signs of infection or inflammation which may require medical assistance. Onychia is an inflammation of the nail folds with formation of pus and shedding of the nail. Which results from the introduction of microscopic pathogens through small wounds, while onychocryptosis is commonly known as ingrowing nails; moreover no known pathognomic nail signs of human immuno deficiency virus infection, Candida is a primary pathogen of nail bed and nail plate. A destructive almost granulomatous like psoriatic involvement, squamous cell carcinoma of the nail bed commonly observe in young adults.[7] In addition to this subungual hematoma or ungual warts may be due to nail tumours caused by human papilloma virus (HPV) associated with squamous cell carcinoma.[8]

Onychomycosis is a fungal infection of nails caused by dermatophytes, yeasts and moulds, accounting for about 50% of onychopathies. A high frequency of onychomysosis caused by candida species reported in India and abroad.[9] The treatment of onychomycosis often require prolonged treatment. Pseudomonal nail infection is the most common organism implicated in bacterial infection of the nail pseudomonas nail infection presents blue-green discoloration beneath the nail typically associated with onycholysis.[10] Acute paronychia is bacterial infection of the proximal and lateral nail fold. Lichen planus can occur in association with either skin or mucosal lichen planus or associated nail unit involvement.

Alopecia areata occurs commonly in young patients, in which nail changes more commonly tend to present simultaneously with hair loss. But rarely can proceed following hair changes by months or years.[11]

Chronic paronychia is considered as form of contact dermatitis related to chronic exposure to irritants or allergens, It is common in patients with ongoing exposure to wet work and other irritants such as food handlers, health care professionals and cleaners.[12] Although candida and bacteria can frequently to be isolated from the affected digits. Longitudinal melanonychia or pigmented band of nail plate, presents as a tan brown, or black stripe that originate as pigmented nail formation. Differential diagnosis includes inherited syndromes, systemic disease (vit B12 deficiency and Addison's disease), drug related, trauma (results intraungual hemorrhage or melanin deposition) fungal infection, melanoma. Longitudinal Erythronychia or red band of the nail associated with splinter hemorrhages in the area of red discoloration. It is nail bed papilloma. Subungual exostosis is a tumor of bone, which presents as a painful hyperkeratotic nodule of the digit frequently associated with onycholysis and deformity of nail plate. Digital mucous cyst (myxoid cyst) (DMC) is a common cystic growth of the nail unit. DMC presents as a firm nodules overlying distal inter pharyngeal joint. DMC are thought to be either reactive or caused by herniation of the synovium. DMC commonly occur overlying joint affected by osteoarthritis.

Summary and Conclusion

The present study of nail diseases in South Karnataka population is quite useful to physician and dermatologist. Nail diseases includes infectious, inflammatory and neoplastic conditions. Onychomycosis is a common nail disease. Inflammatory condition of nail unit can mimic onychomycosis. Subungual tumors often require biopsy. This present study further demands genetic, embryological and nutritional studies because nails are epidermal appendages developed as a modification involving mainly stratum lucidum of epidermis, moreover nails are seen at the end of third month of fetal life. The exact mechanism of formation of nail is still unclear

This research work is approved by the ethical committee of Dr. B. R. Ambedkar Medical College, Kadugondanahalli, Bangalore 560045 (Karnataka).

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Original Article

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Dermatological Manifestations of Dengue Fever in South Karnataka

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Abstract

Background: Dengue fever (DF) is a viral replication which occurs in dandric (langerhens) cells of the skin as early target of infection. Hence 50% DF involes Dermatological Manifestation.

Methods: The Diagnosed DF has petechial or Marbilliform skin lesion was noted. Detailed clinical features, laboratory investigations were also recorded.

Results: Dermatological involvement of DF was 26(52), had only cutaneous involvement, 11(22%) had muco-cutaneous involvement, 13(26%) had no Dermatological involvement. The distribution of rash was 21(42%) had generalised rash 17(34%) had Truncal 12(24%) had extremities. Rash with or without puritis was 30(60%) were with purities, 20(40%) without puritis.

Conclusion: As DF continuous to be one of the commonest causes of acute febrile illness with involvement of skin and mucosa have significant morbidity and mortality. Hence involvement skin could be the early diagnostic value for DF to treat efficiently with all preventive measures.

Keyboard: DF; DHF; Petachae; Elisa Igm; NSI antigen; Muco-cutaneous.

Introduction

It is rightly said that, skin is the window to within the body. Hence skin can provide important clues to systemic diseases; enabling the practioner to make a tremendous contribution to the patients care if cutaneous manifestations are in disorder can be identified. As dengue is an viral fever focuses on various Muco-cutaneous manifestation which becomes challenge to dermatologist apart from physician.

Dengue Fever (DF) is a severe Flue-like illness that affects infant's, children, adolescent and adults. The incubation period of DF after Mosquito bite is between 2 to 8 days. The clinical features vary accoutring to the age of patient. Infants and young children usually have only a non-specific febrile illness with rash that is hard to distinguish from other viral illness.[1] The temperature rapidly >39^{o}C and lasts approximately 5 to 6 days and sometimes biphasic. During the febrile period the patient may experience severe headache, retro-orbital pain, myalgia, arthalgia, nausea and/or vomiting. More than 50% of infected patients report rash during this period that initially macular or macula-popular and becomes diffusely erythmatous.[2] Minor hemorrhagic manifestations such as petechiae, epistaxis and gingival bleeding occur in some patients.

As there are four sereo types of DF (DEN 1.4). Dengue virus is a single stranded RNA virus transmitted mainly through mosquito Aedes-aegypti, It is reported that, viral replication, which occurs primarily in microphages, although dendritic cells (Langerhans cells) in skin may be the early targeted of infection.[3] Dengue virus may directly infect the skin.[4] It was also concluded that, absence of direct viral involvement or immune complexes in the skin lesions could be due to viral

host interaction inducing release of un-identified chemical mediators in the skin and the rash has nothing to do with the direct viral invasion or with the presence of immune complexes[5] presence of Dengue virus can cause DHF (Dengue Haemorrogic Fever), Infections of DSS.[6] Hence various manifestation were evaluated.

Material and Method

50 patients admitted at emergency ward at Dr B. R. Ambedkar Medical college hospital Kondgondan halli, Bangalore 560045, (Karnataka) were studied.

Inclusive Criteria: The patients have positive dengue fever test, above 16 year to 60 years, having skin rash were included in the study.

Exclusive Criteria: The patient had negative dengue fever, urticaria with drugs reactions, food allergy or Immune compromised patients. The patients below 16 year above 70 years were excluded from the study.

Methods: The clinical presentations of acute febrile illness, progressive thrombocytopenia, elevated hepatic transminase and presence of detectable dengue Igm and detectable Virus-expressed soluble man-structural protien1 (NS1) by means of enzyme linked immune sorbent assay (Elisa) (panbio, Dengue duo cassette) with other sereologics being negative and blood cultures sterile or sero conversion of cavelesent sera, were diagnosed to have DF.

The dengue fever patients having petechial and/or morbilliform skin lesions. Detailed clinical features, laboratory investigations, major organ involvement treatment and outcome were also recorded. The duration of study was April 2008–October 2010.

Statistical Analysis: Various dermatological involvements and distribution of rash with or without pruritis were classified with percentage. The ratio of male and female was 2:1.

Observation and Results

Study of dermatological involvement in dengue fever patients– 26(52%) patient only cutaneous involvement, 11(22%) had muco-cutaneous involvement 13(26%) patients had no dermatological involvement.

Table 1: Dermatological Involvement in Dengue Fever Patients.
No of Patients: 50

Sl No.	Particular	No of Patients	Percentage
1	only cutaneous involvement	26	52
2	Muco- cutaneous involvement	11	22
3	No dermatological involvement	13	26

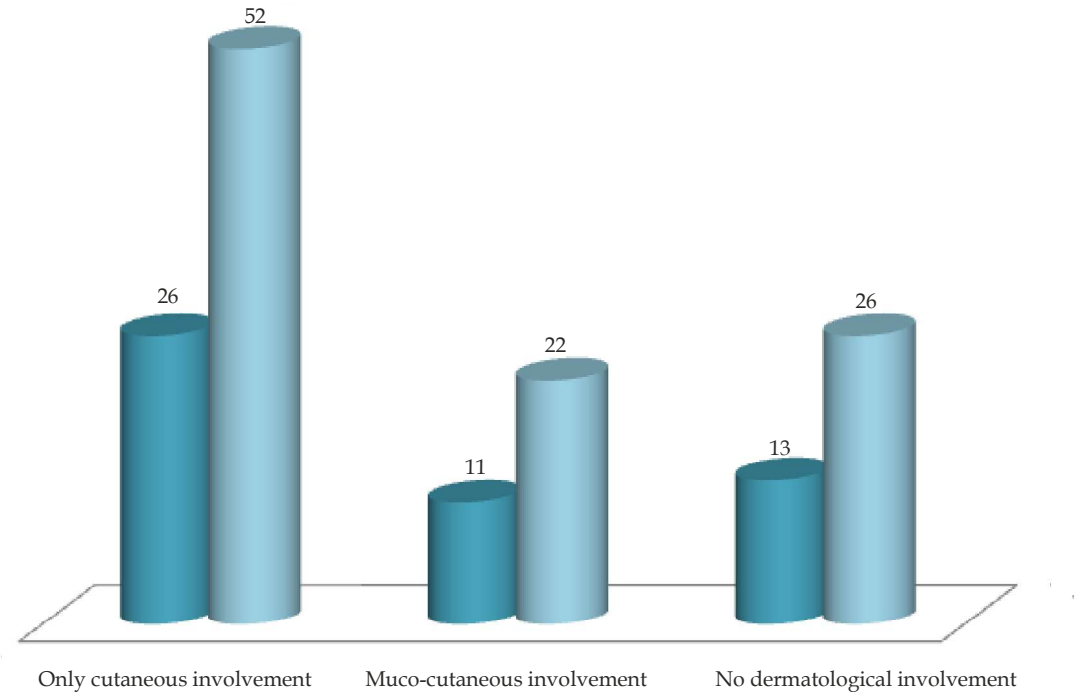


Table 2: Distribution of Rash in Patients with Dengue Fever.
No of Patients: 50

Sl No.	Particular	No of Patients	Percentage %
1	Generalised	21	42
2	Truncal	17	34
3	Extremities	12	24

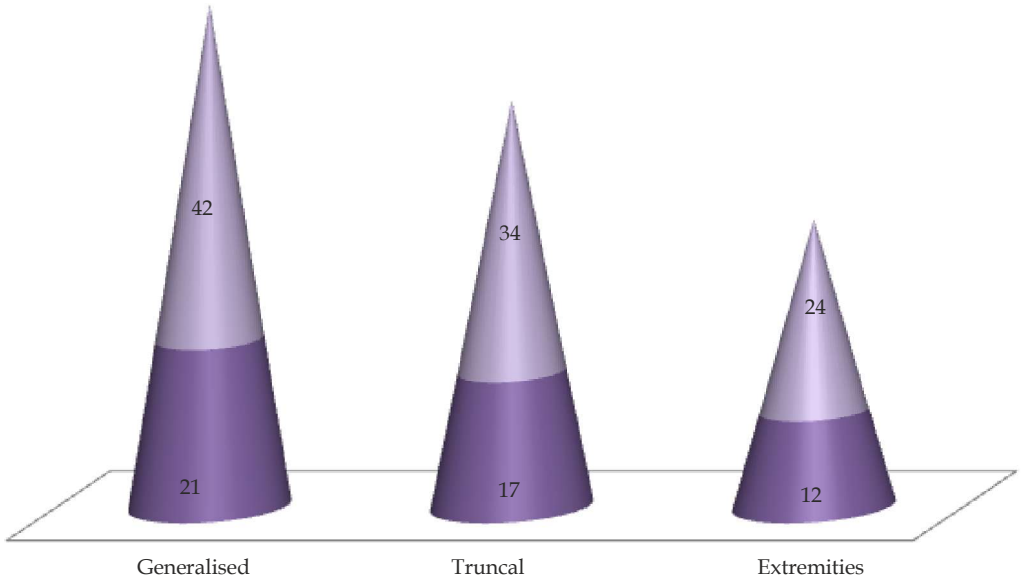
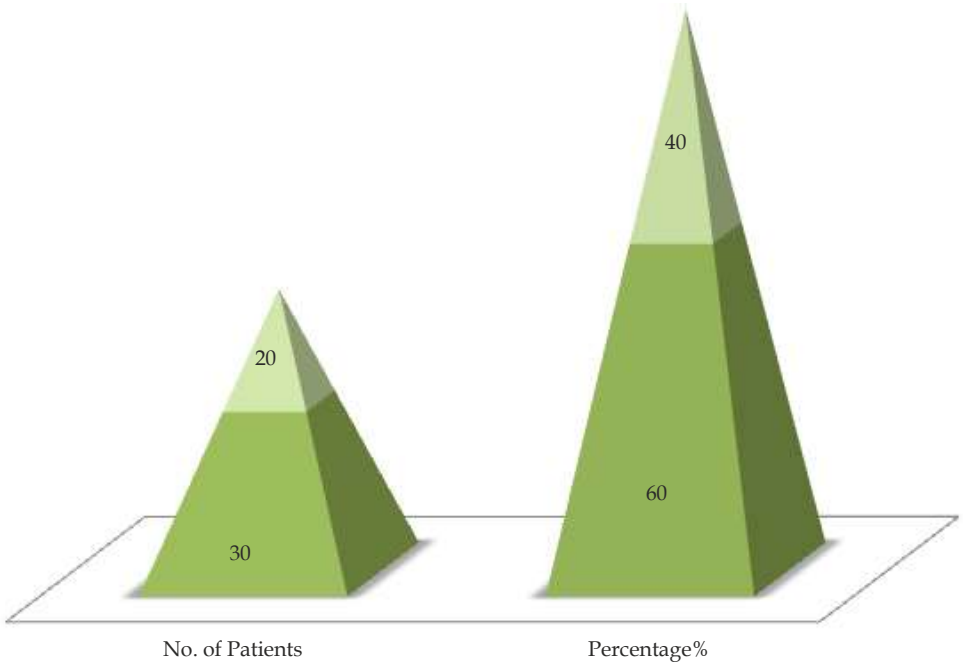


Table 3: Rash with or without Pruritis in Patients with Dengue Fever.
No of Patients: 50

Sl No.	Particular	No of Patients	Percentage %
1	with pruritis	30	60
2	without pruritis	20	40



Study of distribution of Rash in patients with dengue fever 21 (42%) had generalised rash 17(34%) had truncal 12 (24%) had prurities on extremities.

Study of rash with/without pruritis in dengue fever patients 30 (60%) had rash with pruritis 20 (40%) had rash without pruritis.

Discussion

In the present study of Dermatological manifestation in DF 26(52%) patient had cutaneous involvement 11(22%) had muco-cutaneous involvement 13(26%) had no dermatological involvement (Table 1). The distribution of rash in DF was 21(42%) had generated rash 17(34%) had truncal 12(24%) had pruritis on extremities (Table 2). Study of ash with or without pruritis was 30(60%) had rash with prurities and 20(40%) had rash without pruritis (Table 3). These findings were more or less in agreement with previous studies[7-9].

Moreover Tourniquet test is performed by inflating a blood pressure cuff on the upper aspect of arm to a point midway between systolic and diastolic Blood pressure for five minutes. The test is considered positive when >20 petechae.2.5 cm2 are observed. This test certainly indicates involvement has dermatological manifestation has significant role in DF[10]. As DF itself is a viral infection commonly affects conjuctival and sclera mucosa small vesicles on soft palate erythema and crusting of lips and tongue, such involvement on mucosal membrane may be up to 50 % was also observed[11]. It was interesting to note that, in the local skin epithelium was not involved rather small blood versus and endothelium were involved with oedema. In addition to the skin test for dengue virus test by immuno fluroscene was negative. The skin was prominently involved due to intra dermal haemorrhage or petechae. Otherwise involvement skin and skin appendages have no etiological signs rather than diagnostic value, due to involvement of sub-mucosal or sub-dermal capillaries.

Summary and Conclusion

The present study of involvement of dermatological manifestation in DF and related complication will be certainly helpful to Dermatologist and physician to diagnose DF and treat efficiently but these study further demands histo-pathological, virological, nutritional, genetic, bio-chemical studies because exact pathogenesis of DF and its involvement in dermatomes is still unclear.

The Research paper was approved by ethical committee of Ambedkar Medical College, Kondgondan halli Bangalore 45 (Karnataka).

Conflict of Interest: No

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Lichen Planopilaris Presenting with Different Morphologies: 3 Case Reports

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Abstract

Lichen planopilaris is a primary cicatricial alopecia involving hair follicles with a lymphocytic inflammatory process destroying the follicles. Clinically it presents with grouped keratotic follicular papules surrounded by erythema. It is classified as classic LPP, frontal fibrosing alopecia (FFA), and Lassueur Graham-Little Piccardi syndrome. Pseudopelade of Brocq (PPB), can be considered as end-stage LPP which leaves clinical appearance of "footprints in the snow" appearance. Scarring alopecia represents a challenge to patients as well as treating dermatologist where early diagnosis is essential as treatment is difficult in late stage disease. Dermatoscopy accompanied by biopsy is indicated in such cases to start the treatment at the earliest. Three cases of lichen planopilaris with different morphologies is presented here.

Keywords: Lichen Planopilaris; Scarring Alopecia; Pseudopelade of Brocq.

Introduction

Lichen planopilaris (LPP) is a chronic cutaneous disorder selectively involving hair follicles with a lymphocytic inflammatory process that gradually destroys the follicle; thus, called as a primary cicatricial alopecia. It is a disease of unknown etiology, whose pathogenesis is poorly understood, despite a suspected autoimmune origin.[1]

LPP usually affects young adult females, although the age range is wide and can also affects males.[2] It commonly develops in association with lichen planus affecting the skin, mucosa and nails. Clinically it presents with grouped keratotic follicular papules surrounded by erythema in the initial stage which can be confirmed by histological examination, however a late disease has no specific signs, thus making it difficult to separate from other scarring scalp conditions even with the histopathological examination.[3] Early diagnosis is required for early initiation of therapy and halt the progress of the disease.

Case reports

Case 1

A female aged 36 years presented with hair loss and itching over scalp for last 1 year. Of alopecia with atrophy and shiny skin involving parietal scalp was present with typical foot print in snow appearance [Fig. 1a]. No other parts of body, nails or oral mucosa wear affected. Dermatoscopic findings showed lack of follicular orifices and perifollicular scales [Fig. 1b]. Histopathology showed hyperkeratosis, acanthosis and orthokeratosis in epidermis. Dermis showed perifollicular lymphocytic infilterate, periappendegeal and perivascular inflammatory infilterate [Fig. 1c]. Changes were suggestive of lichen planopilaris.

Case 2

A 38 year old male presented with single lesion over scalp for last 5 months. Lesion was itchy and progressive in nature. Patient was tchronic tobacco chewer having tobacco for last 15 years. On

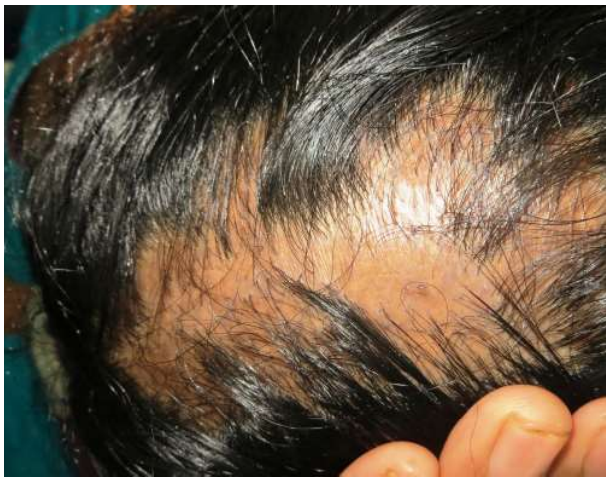

Fig. 1a: Atrophic patches of alopecia involving parietal scalp with typical foot print in snow appearance.

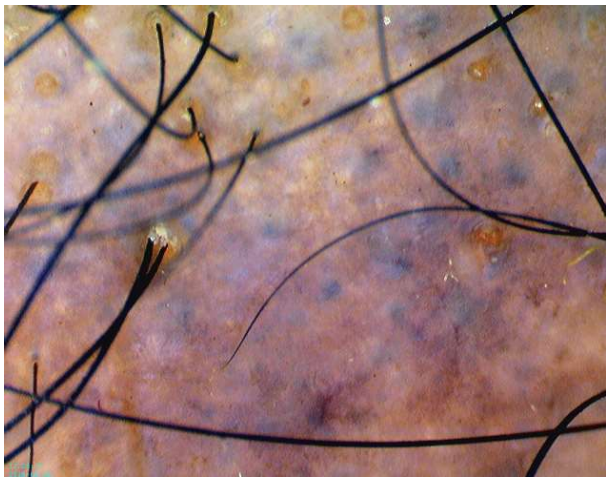

Fig. 1b: Dermatoscopic showing lack of follicular orifices and perifollicular scales.

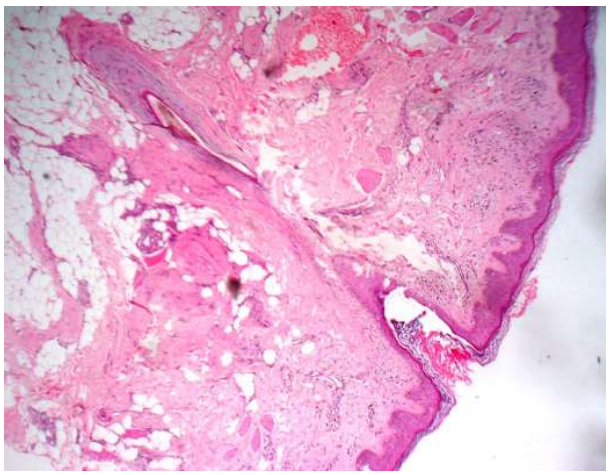

Fig. 1c: Hyperkeratosis, acanthosis, and orthokeratosis in epidermis with periappendegeal and perivascular inflammatory infilterate in dermis. [H&E stain 4X].

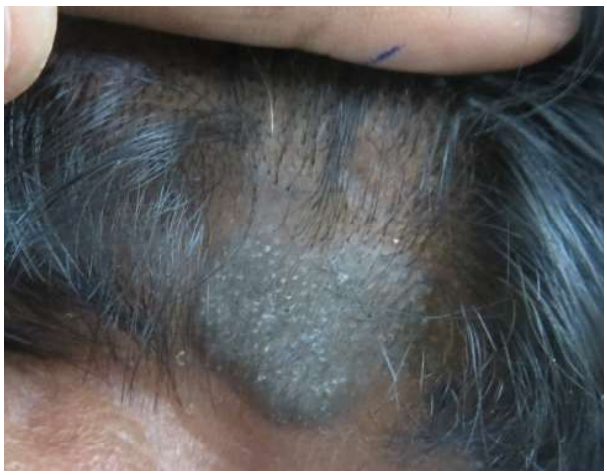

Fig. 2a: Single well defined hyperpigmented plaque with follicular plugging over left side of occiput.

Fig. 2b: Dermatoscopy showing pigmentation, and perifollicular scales, follicular plugging and reduced follicular ostia.

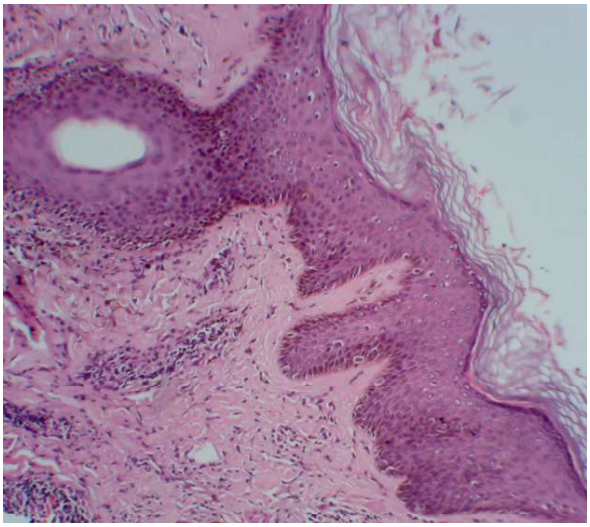

Fig. 2c: Follicular plugging, hyperkeratosis, focal wedge shaped hypergranulosis, elongated rete ridges and focally dense band like perifollicular lymphocytic infilteration at the base of rete ridges and infundibulum. [H&E stain 4X]

Fig. 3a: Single well defined hyperpigmented plaque with follicular plugging over vertex.

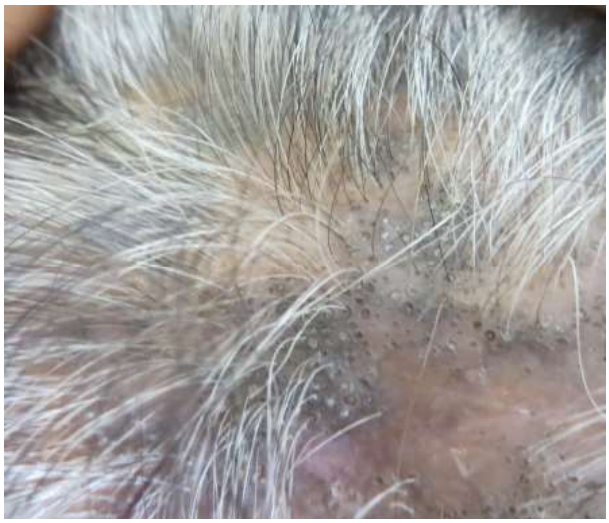

Fig. 3b: Multiple patches of scarring alopecia over parietal and occipital regions.

Fig. 3c: Dermatoscopy showed reduced follicular orifices, white dots as well as perifollicular scale.

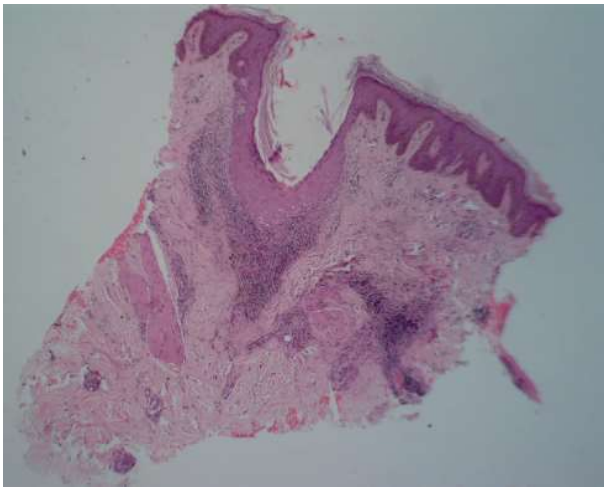

Fig. 3d: Follicular plugging and intense lymphocytic inflammatory infiltrate in perifollicular region. [H&E stain 4X].

examination, single well defined hyperpigmented plaque with follicular plugging was present over left left side of occiput [Fig. 2a]. Violaceous pigmentation was present over bilateral buccal mucosa. Nails showed clubbing and longitudinal melanonychia. No other areas of body were affected. Dermatoscopy showed pigmentation, perifollicular scale, follicular plugging and reduced follicular ostia [Fig. 2b]. Biopsy showed follicular plugging, hyperkeratosis, focal wedge shaped hypergranulosis, elongated rete ridges and focally dense band like perifollicular lymphocytic infilteration at the base of rete ridges and at the level of infundibulum [Fig. 2c]. Changes were suggestive of LPP.

Case 3

A 65 yr old male presented with multiple patches of hair loss over scalp for last 1 month. Lesions were progressive in nature but non itchy. On examination, single well defined hyperpigmented plaque with follicular plugging over vertex and multiple patches of scarring alopecia over parietal and occipital regions were present [Fig. 3a&b]. Dermatoscopy showed reduced follicular orifices, white dots and perifollicular scales [Fig. 3c]. Histopathological examination showed follicular plugging and intense lymphocytic inflammatory infiltrate in perifollicular region [Fig. 3d]. No evidence of any other body parts ie skin, nails or mucosal involvement was seen. Dermatoscopic and Histopathology finding showed changes of lichen planopilaris.

Patients were given topical steroids and tacrolimus 0.1% cream to applied.

Discussion

LPP is also known as lichen follicularis or follicular lichen planus, and was initially described by Pringle in 1895.[4] It is an uncommon inflammatory scalp disorder that is clinically characterized by perifollicular erythema, follicular hyperkeratosis, and permanent hair loss. It is considered a follicular variant of lichen planus. According to the NAHRS (North American Hair Research Society) classification, LPP has been subdivided into 3 variants: classic LPP, frontal fibrosing alopecia (FFA), and Lassueur Graham-Little Piccardi syndrome.[5]

LPP is more common in females (60 to 90%) than in males[6,7] and the age of onset of LPP is frequently between 40 and 60 years.[8] Two of the patients were males while one was female with 2 in their thirties and one with 65 years of age. Endogenous or exogenous agents such as drugs, viruses or contact sensitizers have been proposed as potential triggers for the autoimmune response observed in LPP[6].

Symptomatically, it presents with itching, pain or burning when inflammation is present, aggravated by heat and also associated with seborrheic dermatitis but not pathognomic for the disease. The early lesions are characterised by a follicular violaceous erythema and keratotic plugs, commonly located at the periphery of expanding areas of alopecia. Some hairs affected by the inflammation process can persist in the centre of the bald area with some tufted hairs. A positive pull test of anagen hairs is commonly present at the margin of alopecia, which indicates disease activity. Atrophic scarring without follicular units is seen in late stage of LPP without any typical papules of lichen planus on the scalp, as was seen in 1st case.

Parietal and fronto-vertical regions are the most frequently involved part of the scalp.[9]

Scarring alopecia of the scalp can be associated with other regional involvements in 17–28% patients in one study,[6] while 50%[9] in another study. Lesions of typical lichen planus over nonfollicular skin, mucous membranes, and nails are present in only 50% of cases and usually follow the onset of cicatricial alopecia.[7] Two of the patients didn't showed involvement of any other site while case 2 had oral involvement in the form of purplish pigmentation over bilateral buccal mucosa and nail changes showing clubbing and longitudinal melanonychia. This can be attributed to the habit of tobacco chewing in him since last 15 years.

Patients may present with prominent and characteristic multifocal irregular areas of patchy scarring hair loss resembling moth eaten alopecia which is similar to secondary syphilis and alopecia areata.[10] The presence of small asymptomatic truncal follicular papules is not noticed by the patients unless closely looked for by the dermatologist, so careful examination of all body regions are mandatory in evaluation of scarring scalp conditions.

The main diagnostic challenge for LPP is DLE, thus making it difficult to separate from other scarring scalp conditions even with the histopathological examination. However there are many clinical and histopathological points which can help the dermatologists to differentiate between these two primary lymphocytic cicatricial conditions.[11]

Pseudopelade of Brocq (PPB), can be considered as end-stage LPP, may leave the clinical appearance of "footprints in the snow" as presented by Case 1. If most of the lesions of PPB are old and the LPP is not expanding with perifollicular inflammation and hyperkeratosis, it is impossible to distinguish PPB from LPP. Dermoscopy shows lack of follicular orifices in the centre of bald areas and on the margin, the pink/red translucent inflammation is clearly perifollicular, with keratin surrounding and extending along the proximal part of the hair shafts.

Treatment is difficult and response with most of the drugs is poor. Topical corticosteroids and intralesional corticosteroid injection are commonly utilized as first-line therapy for LPP.[12]

Conclusion

Scarring alopecia represents a challenge to patients as well as treating dermatologist where early diagnosis is essential for initiation of an early and effective therapy to save the hair follicles from the irreversible damage.This can be done by dermatoscopy supplemented by histopathology if required.

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Henoch-Schoenlein Purpura with IgA Nephropathy Treated with Rituximab: A Case Report

Priya J Kariya[1], Krina B Patel[2], Palak Makwana[3]

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Abstract

Henoch-Schoenlein purpura (HSP) is a distinct subtype of hypersensitivity vasculitis with IgA nephropathy as major complication. We report a case of 8-year old male who presented with recurrent HSP and IgA nephtopathy successfully treated with rituximab leading to complete and sustained remission in skin lesions and renal function.

Keywords: Henoch-Schoenlein purpura; IgA nephropathy; Rituximab.

Introduction

Henoch-Schoenlein purpura (HSP) is an IgA mediated systemic small vessel vasculitis with IgA deposition in vessel walls leading to symptoms involving skin, joints and kidneys. It typically affects children following a respiratory tract infection.[1] HSP is more frequent in two to eight years of age with male predominance (M:F=1.5:1).[2] Although HSP is generally a self-limiting disease, the long-term prognosis depends on the severity of renal involvement. Small subsets of patients are unable to wean off corticosteroids and novel corticosteroid sparing and safe immuno modulators are needed.

Case Report

An eight-year old male child presented with history of recurrent skin lesions on legs associated with fever and joint pains for 4 months (Fig. 1). There was no history of drug intake prior to first episode. Patient was treated with antibiotics and NSAIDs during each episode with recurrence within 15–20 days each time. Patient presented to us during 4th episode with bilateral symmetrical, tender, purpuric lesions on lower extremities associated with edema feet and scrotum.

Baseline laboratory investigations revealed total white blood cell (WBC) count 17,400/cumm with normal differential count. Urine routine microscopy analysis showed; Red blood cells (RBC) 28 cells/ hpf and albumin ++. 24–hour urinary protein was 330mg (N <140mg/day). Platelet count, renal and liver function tests were within normal range. Stool examination for occult blood was negative. The anti-Streptolysin O titter, C-reactive protein, and complement levels were in the normal range. S.ANA, DsDNA and ANCA were negative. HIV, HSV, HbSAg, HCV serology were non-reactive. X-ray chest and abdominal sonography did not

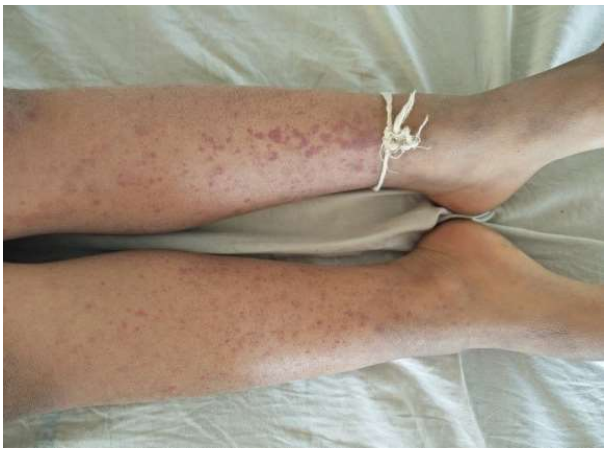

Fig. 1: Palpable Purpura with Oedema of Distal Extremity.

show any abnormality. Skin biopsy on routine histopathology showed an inflammation of small vessels in the upper dermis with a dense infiltrate of neutrophils and fibrin deposits into vascular wall. Direct immuno fluorescence did not show any deposits. USG guided renal biopsy showed localized mesangial proliferation in glomeruli with interstitial mononuclear infiltrate confirming mesangial proliferative glomerulonephritis. Immuno fluorescence showed strong positive IgA and weak positive C3 deposits in the mesangium. Based on clinical, histopathological and renal biopsy findings diagnosis of Henoch-Schoenlein purpura associated with IgA nephropathy (mesangial proliferative glomerulonephritis) was concluded.

Patient was prescribed oral prednisolone 2mg/kg which was tapered to 1.5mg/kg over 20 days. Repeat urine examination at day 20 showed persistent hematuria with RBC 70 cells/ hpf. Clinically fever and edema feet persisted. Nephrologist advised to continue high dose steroid for at least 6 weeks without tapering due to renal hypertension but; as patient was developing side effects of systemic steroids in terms of weight gain; alternative treatment options were searched for and as per references in literature of use of rituximab in pediatric HSP with renal involvement[3,4] and consent from parents, intravenous infusions of Rituximab 400mg (375mg/m2 approximately) in 200ml normal saline twice at 2 weeks interval were given along with tapering dose of corticosteroids. After first rituximab infusion itself patient improved symptomatically with normal temperature and reduction in joint pain. RBC count in urine reduced significantly by day seven (55 cells/hpf). After second infusion rituximab urine examination returned to normal range, total WBC count reduced to 10,400/cumm; scrotal edema, leg edema as well as fever subsided completely. 24-hour urinary protein also returned to normal level. As there was improvement in all parameters, tapering of steroid was started 10 days after 1stinfusion of rituximab and the same was rapidly tapered to 10 mg/day by week two post 2nd infusion. Within 3 weeks of last rituximab infusion; patient was able to stop steroid completely with complete recovery of renal function and no recurrence in one year follow-up.

Discussion

HSP is a systemic vasculitis characterised by the clinical tetrad of palpable purpura, arthritis, haematuria and abdominal pain. The most serious complication is renal involvement which requires treatment with long term corticosteroids. The pathogenesis of the nephropathy involves the deposition of aberrant glycosylated IgA1 and/ or of IgA1 immune complexes in the glomerular mesangium.[5] A meta-analysis has shown that there is no evidence of benefit of prednisone in preventing serious long-term kidney disease in HSP.[6] Thorne et al report three paediatric patients successfully treated with rituximab for severe refractory HSP.[3] Crayne et al also reported steroid sparing effect of rituximab in chronic steroid dependent and immuno modulator refractory HSP in eight paediatric cases.[4] The probable mechanism of action of rituximab in IgA nephropathy is loss of IgA producing B-cells and effect on cytokine production and antigen presentation.[3, 4]

We report here a case of pediatric patient with refractory HSP successfully treated by two rituximab infusions without any adverse events. Our patient had a rapid improvement in skin and renal symptoms after rituximab leading to a dose reduction of corticosteroids without relapse.

Candidates for rituximab treatment could be patients with poor risk factors with progression to end stage renal disease or patients with severe HSP experiencing relapse, as a steroid sparing agent. We recommend more studies of rituximab in patients of HSP with renal involvement.

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