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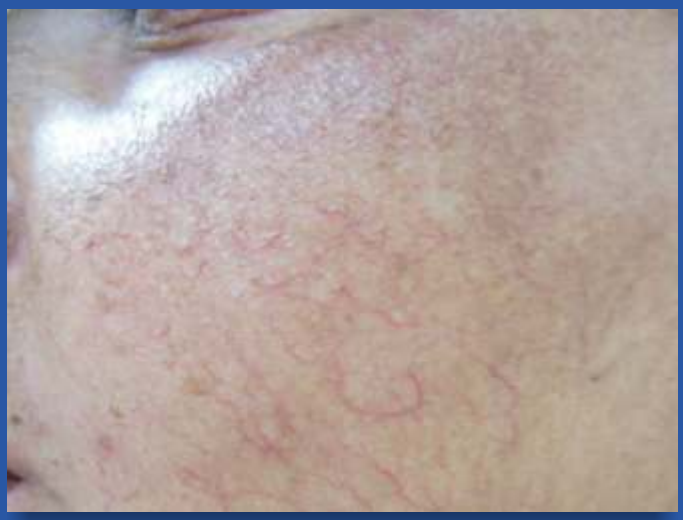

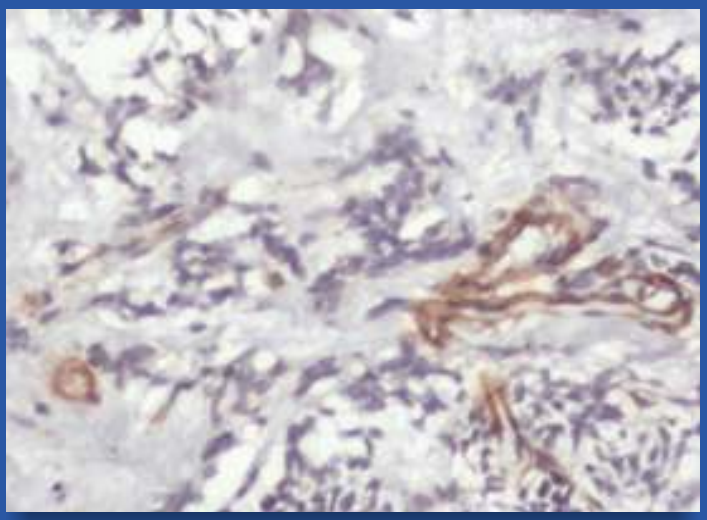

HIGHLIGHTS

Dermoscopic Study of Primary Cutaneous Amyloidosis : New Cartwheel and Horse-shoe Pattern Revealed

A Cross Sectional Descriptive Study on Clinical Type and Etiological Agent of Superficial Dermatophytosis

Misuse of Topical Corticosteroids on Face: A Hospital Based Clinicoepidemiological Study

Chondroid Syringoma – A Rare Mixed Appendageal Tumour at Multiple Sites

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JDA INDIAN JOURNAL OF CLINICAL DERMATOLOGY

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JDA INDIAN JOURNAL OF CLINICAL DERMATOLOGY

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FROM THE DESK OF EDITOR

Dear friends

We are moving forward and I bring you the third issue of **JDA INDIAN JOURNAL OF CLINICAL DERMATOLOGY.** I am pleased to tell you that we have many subscribers now and the support for the journal is increasing. I congratulate and thank our authors, reviewers and my team for that. We are now moving forward to get our journal indexed. As promised we take every effort to publish only quality articles after proper review still your valuable suggestions are always welcome. Please go through our current issue and give your feedback. I thank you for your support.

Happy reading !!!!

Dr. Dinesh Mathur
Editor

ORIGINAL ARTICLE
Year: 2018 I Volume: 1 I Issue: 3 I Page: 68-70

DERMOSCOPIC STUDY OF PRIMARY CUTANEOUS AMYLOIDOSIS : NEW CARTWHEEL AND HORSE-SHOE PATTERN REVEALED

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Abstract

Cutaneous Amyloidosis is the anomalous deposition of amyloid in the skin. It is broadly classified into primary localized cutaneous amyloidosis(papular , macular and nodular amyloidosis) and secondary localized cutaneous amyloidosis. Some time it is difficult to differentiate it from other causes of pigmentation and similar non specific conditions. So we did an extensive one year dermoscopic study to reveal all possible dermoscopic signs of primary cutaneous amyloidosis(PCA) in Indian cases. Aim – To study dermoscopic features of Primary cutaneous amyloidosis in Indian skin type patients. Study period – One year (from December 2016 to December 2017). Study subjects-All the patients who attended the dermatology clinic from December 2016 to December 2017 with the clinical diagnosis of PCA and who fulfilled the inclusion and exclusion criteria. Methodology- All the patients who attended the dermatology clinic from December 2016 to December 2017 with the diagnosis of PCA and who fulfilled the inclusion and exclusion criteria were recruited for the study. Dermoscopy was performed with DL4 dermatoscope. The images were further magnified with the help of smart phone. Results-. Our study showed various dermoscopic signs in cases of PCA. The commonest pattern was Central hub pattern (38.15 %) followed by non specific pigment configurations (17.1 %).Our study discovered two new dermoscopic signs(not mentioned in the earlier literature) which includes the Horseshoe pattern (6.57 %) and Cart-wheel pattern (7.89 %). The other classical patterns revealed in our dermoscopic study were amorphous pigment globules (14.47 %), central scar pattern (7.89 %) and patchy pigmentation with other patterns (7.89%). Discussion-Dermatoscopy is a novel helpful bedside noninvasive technique in clinical dermatology practice which allows us to make a quick and accurate diagnosis of PCA. It also helps to distinguish it from other mimickers of pigmentation. The common patterns were Central hub pattern (38.15 %) and non specific pigment configurations (17.1 %).Our study identified two novel dermoscopic signs which includes the Horseshoe pattern (6.57 %) and Cart-wheel pattern (7.89 %). The dermatoscopic diagnosis of PCA is made by the acquaintance and meticulous search of various signs and should not be dependent on the presence of single observation. Other recent dermoscopic study of PCA supported our findings . There are very few dermoscopic studies in PCA across the world. Therefore our study may be helpful in developing a strong scientific scaffold for further dermoscopic research to reveal its importance in the cases of PCA.

Key Words - Primary cutaneous amyloidosis, Dermoscopy, Dermatoscope, Dermoscopy of cutaneous amyloidosis, PCA, Dermoscopy of dark skin

Introduction

Cutaneous Amyloidosis is the anomalous deposition of amyloid like substance in the skin which can be associated with mild pruritus, abnormal pigmentation and can affect skin texture too.[1,2] As far as skin is considered it is broadly classified into primary localized cutaneous amyloidosis(papular amyloidosis, macular amyloidosis and nodular amyloidosis) and secondary localized cutaneous amyloidosis.[2,3,4] Some time it is difficult to differentiate it from the other causes of pigmentation and similar non specific dermatological conditions.[4] So we did an extensive one year dermoscopic study to reveal possible dermoscopic signs of PCA in Indian cases. There are very few dermoscopic studies in PCA.[5,6] Therefore our study may be helpful in developing a scientific scaffold for further dermoscopic research to reveal its importance in the cases of PCA.

Materials & Methods

Aim – To study dermoscopic features of Primary cutaneous amyloidosis in Indian skin type patients.

Study subjects-All the patients who attended the dermatology clinic from December 2016 to December 2017 with the clinical diagnosis of PCA and who fulfilled the inclusion and exclusion criteria.

Inclusion criteria

All clinical cases of PCA

Exclusion criteria

1) Inability to give consent
2) Patients with non specific lesions
3) Having co-existent other skin diseases near the lesions

Study period – One year (from December 2016 to December 2017).

Methodology- All the patients who attended the dermatology clinic from December 2016 to December 2017 with the diagnosis of PCA and who fulfilled the inclusion and exclusion criteria were recruited for the study. Dermoscopy was performed with DL4 dermatoscope. The images were further magnified

with the help of smart phone.

Results

We recruited 76 patients of PCA on the basis of their clinical diagnosis during the one year study period(Fig. 1,2). Our study showed various dermoscopic signs in cases of PCA. The commonest pattern was Central hub pattern (fig. 3) (38.15 %) followed by non specific pigment configurations (fig. 4) (17.1 %).Our study discovered two new dermoscopic signs(not mentioned in the earlier literature) which includes the Horseshoe pattern (6.57 %) and Cart-wheel pattern (fig. 5) (7.89 %). The other classical patterns revealed in our dermoscopic study were amorphous pigment globules (fig. 6) (14.47 %), central scar pattern (fig. 7) (7.89 %) and patchy pigmentation with other patterns (7.89%). Figure 8 summarizes the findings.

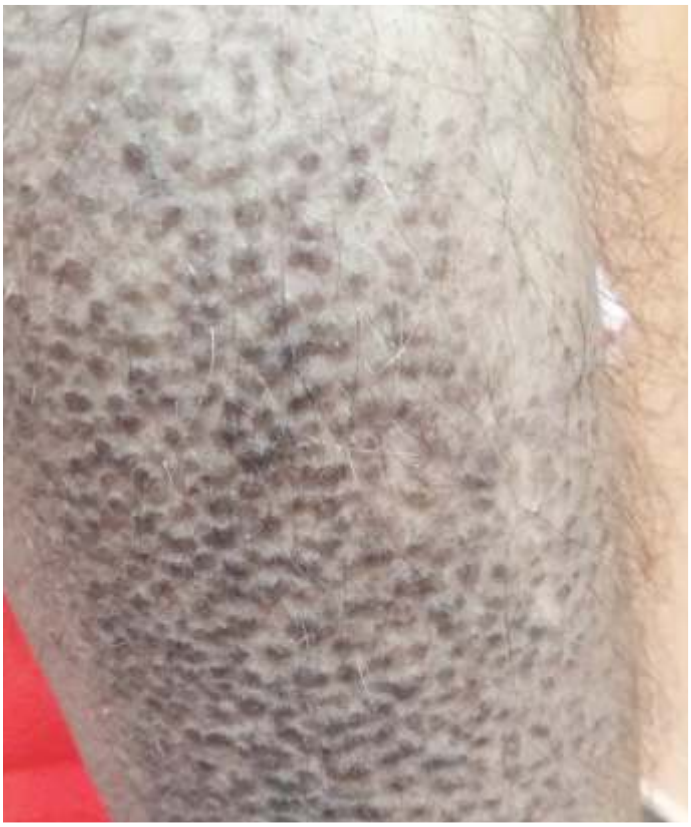

Figure 1: Lichen (papular) amyloidosis clinical image

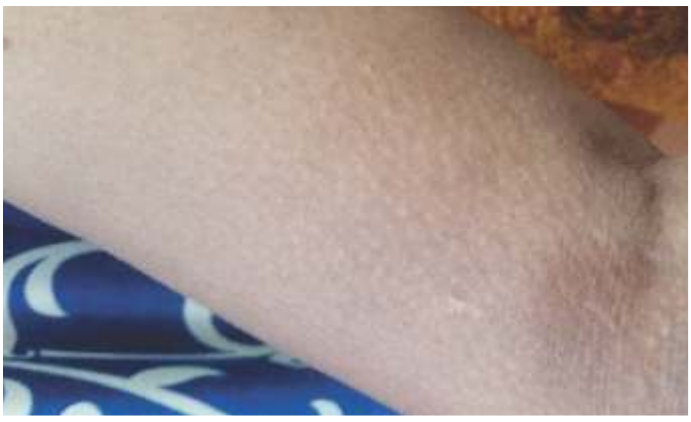

Figure 2: Clinical image of macular amyloidosis with classical rippled pattern of pigmentation

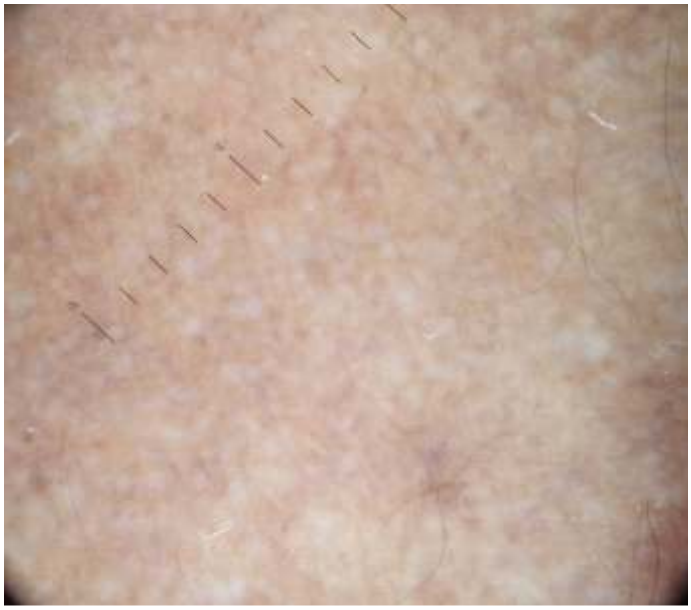

Figure 3: Central hub pattern

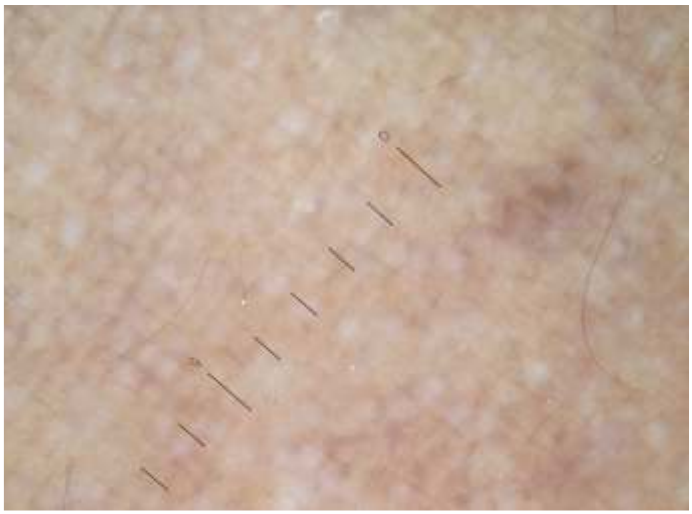

Figure 4: Non specific pigment configurations

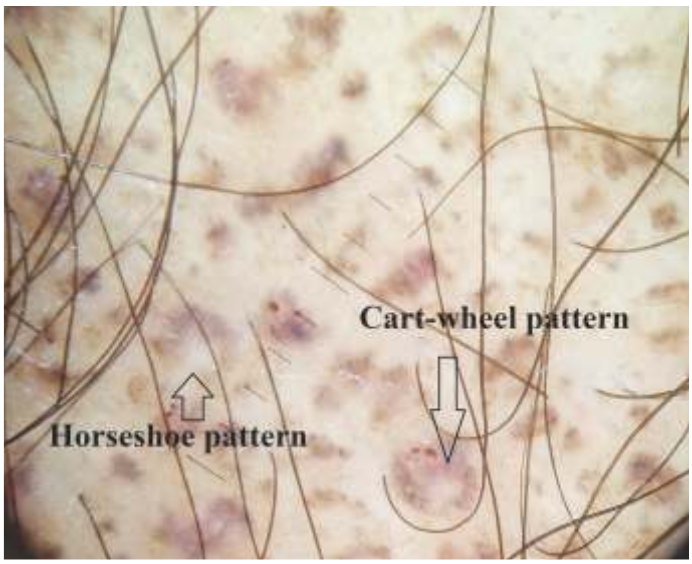

Figure 5: Cart-wheel and horseshoe pattern (new dermoscopic signs in PCA revealed by our study)

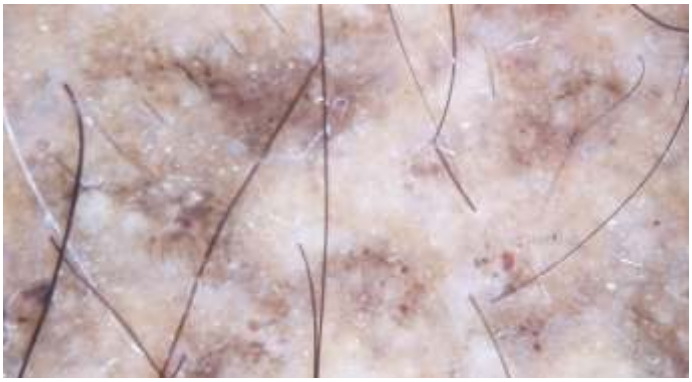

Figure 6: Amorphous pigment globules

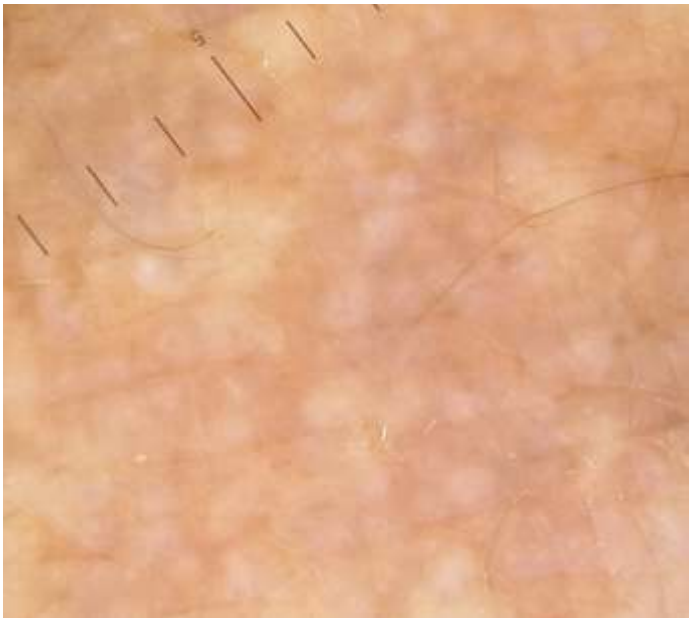

Figure 7: Central scar pattern

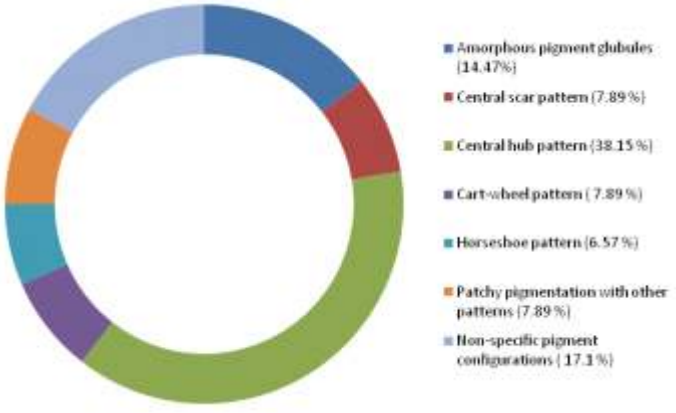

Figure 8: Various dermoscopic signs and their incidence seen in our study

Discussion

Dermatoscopy is a novel helpful bedside non invasive technique in clinical dermatology practice which allows us to make a quick and accurate diagnosis of PCA. It also helps to distinguish it from the other mimickers of pigmentation and similar cutaneous conditions. The common patterns were central hub pattern (38.15 %) and non specific pigment configurations (17.1 %).Our study identified two novel dermoscopic signs which includes the Horseshoe pattern (6.57 %) and Cart-wheel pattern (7.89 %). The dermatoscopic diagnosis of PCA is made by acquaintance and meticulous search of various signs and should not be dependent on the presence of single observation. Other recent dermoscopic study of PCA supported our findings.[6] There is very limited literature available as far as dermoscopy of PCA is considered.[5,6] There are very few dermoscopic studies in PCA in the world. Therefore our study may be helpful in developing a strong scientific scaffold for further dermoscopic research to reveal its importance in the cases of PCA.

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ORIGINAL ARTICLE
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A CROSS SECTIONAL DESCRIPTIVE STUDY ON CLINICAL TYPE AND ETIOLOGICAL AGENT OF SUPERFICIAL DERMATOPHYTOSIS

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Abstract

Introduction: Present study was undertaken to identify the clinical pattern of superficial dermatophytes at a tertiary care center and to etiological correlation fungal pathogen responsible for the dermatophytosis. **Materials & Methods:** A prospective, observational study was conducted on patients attending the dermatology department of a tertiary care hospital with clinical features of dermatophytosis. A total of 115 eligible cases were included in the study after informed consent. Detailed socio-demographic and clinical history was taken from all patients. A skin sample was collected from the lesion site from all patients. The samples were transported to and processed at the Microbiology Department of the hospital laboratory. **Results:** Most common age group affected was 16-30 years with mean age of 28.4 years with male predominance (69.6%) Overall 149 sites were involved in 115 cases. Most common clinical type was T. Cruris (53%) followed by T. Corporis (23.5%). KOH mount was positive in 79 (53%) isolated out of 149 while culture was positive in 101 isolates (67.8%). Most common organisms isolated were T. rubram (54.5%) and T. Mentagrophytes (45.5%). T. rubram is the common organism isolated in Tinea corporis and cruris while T. Mentagrophytes was more commonly isolated in cases of Tinea pedis, capitis and faciei. **Conclusion:** Present clinico mycological study showed tinea cruris as the most common clinical pattern followed by tinea corporis and T. rubrum as the most common causative agent of dermatophytosis in this region with increasing trend of T. mentagrophytes. Involvement of face and scalp in adult population. Both direct microscopy and culture are important tools of diagnosis for the superficial fungal infections.

Key Words- Dermatophytic infections, Tinea Corporis, Tinea Cruris, Trichophyton

Introduction

Dermatophytes are a group of closely related keratinophilic fungi that infect keratinized tissues such as hair, nails and skin. The disease caused by dermatophytes is known as dermatophytosis which constitutes an important public health problem, not only in underdeveloped countries but also in elderly and immuno-compromised patients worldwide [1].

The etiologic agents of the dermatophytosis can be categorized into one of three genera: Epidermophyton, Microsporum and Trichophyton. They possess keratinophilic and keratinolytic properties. The infections due to these pathogens are generally cutaneous and restricted to the non-living, cornified layers of the skin [2].

Traditionally, infections caused by dermatophyte (ring-worm) have been named by appending the latin name of the affected body part after the word "tinea" [3]. Tinea capitis (ringworm of the scalp) is the most common fungal infection in children. More than 90% of the infections are caused by Trichophyton tonsurans, and fewer than 5% are caused by Microsporum species [3].

Since these infections are often confused with other skin disorders, it is therefore, necessary to make early laboratory diagnosis for better management of these conditions [4]. Various studies have been conducted in different parts of the country including Chennai[5], Madhya Pradesh[6], Andhra Pradesh, Gujarat[7], Chandigarh[8], Karnataka[9] and few other states[10,11]. The distribution, frequency and the causative agents involved vary from place to place depending upon the climatic, socioeconomic conditions and the population density.

This study of superficial dermatophytes with clinical type was conducted in a service hospital catering to serving as well as retired personnel and their dependent family members. The working condition and environment of serving personnel are different from the general public. They have long working hours, continuous duty hours, wearing tight uniforms and shoes for long time which make them more prone to have dermatophytes infection. The incidence and type of dermatophytes infections in serving personnel may be different from general population. This study is thus undertaken to identify the clinical pattern of this disease in our center and to identify the most common fungal pathogen responsible for the dermatophytosis.

Material and Methods

A Prospective, observational study was conducted on patients attending the Dermatology department of a tertiary care hospital with clinical features of dermatophytosis. Patients with use of antifungal therapy (oral as well as topical) within 2 months, presence of serious underlying systemic conditions, bacterial or

fungal infections in the skin folds and nails and with debilitating conditions like DM, CKD, etc. were excluded. Consecutive type of non-probability sampling was followed for the selection of the study subjects. A total of 115 eligible cases showing clinical features of dermatophytosis and fulfilling the eligibility criteria were included in the study after informed consent.

Study was commenced after approval by the institutional scientific research and ethical committee of hospital. Patients were included after taking written informed consent. Detailed socio-demographic and clinical history was taken from all patients. The information about the applications of antifungal therapy was obtained through inquiry from the patients or the clinician asked them to produce the outpatient chit if any treatment was taken during past 2–3 months and also the other information regarding chronic illness, immunosuppressive/ immunocompromised state including co-infection with HIV and other conditions such as diabetes. Detailed clinical examination done to diagnose the clinical type of tinea and to assess the size, shape, number , inflammation and for any secondary infection. A skin sample was collected from the lesion site from all patients. The samples were transported to and processed at the Microbiology Department of the hospital laboratory.

Sample Collection

The samples was collected in sterile black paper envelop after cleaning the site with 70% ethanol in order to remove the dirt and environmental contaminants. Skin scrapings was collected from advancing margins of the lesions with the help of sterile scalpel blade. In the toe cleft, material was collected by epileation forceps. Hairs for examination were plucked; only those hairs that are broken or lack luster were selected.

Wood's lamp was used whenever required to see the infected hairs as few dermatophytes produce a characteristic fluorescence. In case of black dot type of tinea capitis, material was obtained by scraping the scalp. Material from nail was collected by clipping the proximal part of the involved nails.

Examination of direct KOH mount

The samples of hair follicles, scrapings of skin and nails collected were treated with 10-40% KOH for 10 minutes to overnight (nail), and the samples mounted on a glass slide with Lactophenol blue were examined under microscope low power of magnification (10x and 40x) for fungal hyphae, spores, or yeast cells. The samples were then processed for the isolation of the dermatophytes species on Sabouraud's Dextrose Agar.

Isolation of dermatophytes

The samples were inoculated on Sabouraud dextrose agar (SDA) containing chloramphenicol (0.05 mg/mL) and cycloheximide (0.1–0.4 mg/mL) and incubated at 25 to 30degree temperature. The cultures were examined once a week and were declared negative if no growth was obtained till 4 weeks. The isolates were further identified by studying the culture characteristics, pigment production, and microscopic examination of the lactophenol cotton blue (LPCB) mounts. Those samples that yielded 3 or more growth and were negative in KOH mount were considered contaminants/ mixed growth. Contaminants were defined as mixed growth on SDA without a positive KOH mount. The colonies were examined for their morphology, texture and examination of the reverse of the colony for the presence of characteristic pigmentation. The confirmation was done by microscopic examination of the stained preparations.

Statistical Analysis

All the collected data was entered in Microsoft Excel Sheet 2007.The data was then transferred and analyzed using SPSS ver. 21. Quantitative and qualitative variables were presented as mean +/- SD and as frequency with percentages.

RESULTS

Most common age group affected by dermatophytic infections as observed in present study was 16-30 years (42.6%) with mean age of 28.4 years. Male predominance (69.6%) was observed in the present study with male to female ratio of 2.29:1. Most of the cases in present study were active servicemen (40.9%) with students (29.6%) and housewives (16.5%) being the next common groups. In most of the cases only a single site was involved (71.3%) while multiple sites were involved in 28.7% cases. Overall 149 sites were involved in present study. Most common clinical type was T. Cruris (53%) followed by T. Corporis (23.5%), T. faciei (9.4%), T. capitis (6%), Onychomycosis (4%), T. Pedis (2.7%) and Scutular tenia (1.3%)(Table 1).

Clinical Types	N	%
T. Cruris	79	53.0%
T. Corporis	35	23.5%
T. Faciei	14	9.4%
T. Capitis	9	6.0%
Onychomycosis	6	4.0%
T. Pedis	4	2.7%
Scutular Tinea	2	1.3%
Total	149	100.0%

Table 1: Distribution of subjects based on clinical type of dermatophytic infection

Most common mixed infections were of T. Cruris and T. corporis (24/32; 72.7%) followed by T. cruris and T. faciei (4/32; 12.1%). KOH mount was positive in 79 (53%) isolated out of 149. Culture was positive in 101 isolates (67.8%) while it was negative/ contaminated in 48 isolates (32.2%). Most common organisms isolated were T. Rubram (n-55; 54.5%) and T. Mentagrophytes (n-46; 45.5%) (Table 2).

Species Isolated	N	%
T. Rubram	55	54.5%
T. Mentagrophytes	46	45.5%
Total	101	100.0%

Table 2: Distribution of Subjects based on Species Isolated

T. rubram is the common organism isolated in Tinea corporis and cruris while T. Mentagrophytes was more commonly isolated in cases of Tinea pedis, capitis and faciei. T. rubram was the only organism isolated in cases of scrotal tinea and Onychomycosis (Table 3).

Clinical Types	Species Isolated		Total	Total Organisms	Culture positivity
	T. Rubram	T. mentagrophytes			
T. Corporis	17	11	28	35	80.0%
T. Cruris	28	20	48	79	60.8%
T. Pedis	0	3	3	4	75.0%
T. Capitis	3	4	7	9	77.8%
T. Faciei	2	8	10	14	71.4%
Scrotal Tinea	2	0	2	2	100.0%
Onychomycosis	3	0	3	6	50.0%
Total	55	46	101	149	67.8%

Table 3: Association of etiological agent with Clinical Type

DISCUSSION

Most common age group affected by dermatophytic infections as observed in present study was 16-30 years (42.6%) with mean age of 28.4 years. Male predominance (69.6%) was observed in the present study with male to female ratio of 2.29:1.

These observation are in accordance with the findings of other authors[12-17] who observed maximum number of cases in the second and third decade of life. Surendra et al.[17] observed 44% cases in the age groups of 16-30 years. Mahajan S et al.[14] observed the most commonly affected age group as 20–40 years (52.4%). Although the majority of studies have observed higher incidence in the third decade, the study done at Calicut by Bindu et al.[16] observed higher incidence in the second decade. Male predominance was also observed in majority of the studies[12-17]. The higher incidence in males could be due to greater physical activity and increased sweating. Surendra K et al.[17] in their study observed 62% males as compared to 38% females. Mahajan S et al. observed the male to female ratio as 3:1 in their study[14] while Janardhan et al.[15] observed the ratio as 1.86:1.

Most common clinical type observed in present study was T. Cruris (53%) followed by T. Corporis (23.5%), T. faciei (9.4%), T. capitis (6%), Onychomycosis (4%), T. Pedis (2.7%) and Scutular tenia (1.3%). Tinea Cruris and corporis are the most common clinical types observed across various studies[12-18]. In the studies by Sardari et al.[19] and Verma et al.[20] it has been reported that tinea cruris was the most common clinical type. While in the studies by Surendra et al.[17], Bindu et al. [16] and other studies[13-15], tinea corporis was the most common clinical type of dermatophytic infections. In another clinicomycological study of superficial mycosis in a hospital in north-east India[21], it was observed that tinea pedis (29.2%) as the most common dermatophytosis followed by tinea cruris (26.2%), which differs from other studies.

Prevalence of mixed infection as observed in present study was 28.7% cases. Most common mixed infections were of T. Cruris and T. corporis (24/32; 72.7%) followed by T. cruris and T. faciei (4/32; 12.1%). Prevalence of mixed infection as observed by Surendra et al.[17] was 46% while Mahajan et al.[14] observed the prevalence as 46.8%. Among the mixed clinical types, tinea corporis with tinea cruris combination was the highest in both studies. Similar findings have been reported by Peerapur et al.[22].

KOH mount was positive in 79 (53%) isolated out of 149 while culture was positive in 101 isolates (67.8%). Our results are in accordance with the study by Belukar et al.[23], Malik et al.[24] and Janardhan B et al.[15] which showed culture positivity of 71%, 58.8% and 72% respectively. However, Kumar et al.[12] and Surendra et al.[17] observed overall positivity by culture as 42.4% and 39% respectively. KOH positivity rate as observed by various authors is as follows: Malik A et al. (61.1%)[24], Kumar et al. (55.2%)[12], Santosh K et al. (55.4%)[13] and Mahajan et al. (79.6%)[14]. High positivity rate was observed by Janardhan et al. (90%)[15] and Surendra et al. (96%)[17].

Most common organisms isolated were T. Rubram (54.5%) and T. Mentagrophytes (45.5%). This is in accordance to reports of other workers from different regions of India where T. rubram is the common organism followed by T. mentagrophytes[13,15-18,21]. Mahajan et al. and Peerapur BV et al.[14,22] observed T. mentagrophytes as the commonest organism isolated while in another study by Grover et al.[21] in north-east India, isolated T. tonsurans as the most common dermatophyte followed by T. rubrum, which differs from other studies that reports T. rubrum as the most common fungal pathogen. Overall, the Trichophyton genera dominate the isolates in majority of the studies undertaken[12-27].

Correlating clinical and mycological data, we found that T. rubram is the common organism isolated in majority of the cases while T. Mentagrophytes was more commonly isolated in cases of Tinea pedis and faciei. T. rubram was the only organism isolated in cases of scutular tinea and Onychomycosis. Surendra et al.[17] found that in all clinical patterns, T. rubrum was the chief organism isolated followed by T. mentagrophytes. Kumar et al.[12] observed T. rubrum as common isolate from all clinical types. In T. corporis 34 isolates (61.82%), in T. cruris 26 isolates (74.28%). in T. unguium 3 isolates (60%) were Trichophyton rubrum. In T.capitis and T. manuum T. faciei, only T. rubrum was isolated. Siddappa et al.[28] reported T.rubrum as the major isolate (81.82%) from all clinical types except tinea capitis. Patwardhan et al.[25] observed as T.rubrum as the commonest isolate in all clinical cases. It was prevalent in T. corporis and T.cruris. In study done by Seema Bhaduria et al.[29] T.rubrum was the main isolate in all clinical types 17/50 (34%). In the study done by G. Venkatesan et al.[30], T.rubrum was the main causative agent in T. corporis (45.1%), T. cruris (22.6%). T. pedis (2.8%) onycomycosis 2(2.8%). Various other studies too observed T. rubrum as the commonest species isolated from most clinical types[13-16,18,24].

CONCLUSION

Dermatophytic infections are of concern because of their character of chronicity of the disease, relapses and poor quality of life due to itching and appearance of skin lesions. The study highlighted the various types of Dermatophytic infections in and around the places of Mumbai. Present clinicomycological study showed tinea cruris as the most common clinical pattern followed by tinea corporis and T. rubrum as the most common causative agent of dermatophytosis in this region but increasing trend of T. mentagrophytes which was not seen in old studies. Also increase trend of T. capitis and T. faciei in adult population which was not seen in previous studies. Both direct microscopy and culture are important tools of diagnosis for the superficial fungal infections. Chronicity and frequent relapses may be due to changing pattern of species or can be due to antifungal resistance. Further studies require to know the exact cause of chronicty, relapses.

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CLINICOPATHOLOGICAL CORRELATION IN ERYTHRODERMA

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Abstract

Background: Erythroderma, or generalized exfoliative dermatitis, is a disease characterized by erythema and scaling involving more than 90% of the body's surface. Diagnosing erythroderma is easy but finding its cause is difficult. There is a paucity of Indian studies over the etiology, clinical profile and its histopathological correlation.

Aims and objectives: To assess the demographic profile, clinical features and histopathological correlation in erythroderma patients.

Material and Methods: We registered all patients of erythroderma consecutively from January 2013 to December 2014. After a thorough history and clinical examination, a provisional clinical diagnosis was made. We performed biopsy from two representative sites of patient and it was sent for histopathological examination. The slides were examined by three independent observers without any relevant clinical information. The clinical diagnosis was matched with the blinded microscopical diagnosis.

Results: A total of 66 patients were enrolled in this study. The mean age of the study group was 53.7±16.56 years (Range: 14 to 86 years) with male outnumbering female in a ratio of 3.4:1. Most common cause of erythroderma noted in the study was eczema of various types (53.03%), followed by psoriasis (30.30%), drug induced (12.12%), lymphoma (1.515%), mycosis fungoides (1.515%) and idiopathic (1.515%). Clinico-pathological correlation occured in about 67% (range: 63.6% to 68.2%) of patients (k value 0.495 to 0.572).

Conclusion: Most of the clinical features of erythroderma are overlapping. Specific and diagnostic features of diseases are seen only in a few patients. Clinico-pathological correlation should be done for better diagnosis of patient. Repeated evaluations, close follow-up and multiple skin biopsies are recommended for a better clinical diagnosis and patient care.

Key words: erythroderma, clinicopathological correlation, histopathology

Introduction

Erythroderma or exfoliative dermatitis is an inflammatory disorder in which erythema and scaling occur in a generalized distribution involving more than 90% of the body surface.[1] Because most patients are elderly and skin involvement is widespread, the disease implies an important risk to the life of the patient [2]. Hasan and Jansen estimated the annual incidence of erythroderma to be 1 to 2 per 100,000 patients [3]. This disorder may represent a variety of cutaneous and systemic diseases, and therefore a thorough workup is essential which include detailed history of triggering factors like drugs, occupation, sunlight exposure, pre-existing dermatoses, infections, malignancies etc. It should be followed by a meticulous clinical examination for specific diagnostic clues to rule out its etiology. Histopathology can help in identifying the cause of erythroderma in up to 50% of cases, particularly by multiple skin biopsies.[4]

Indian studies showed a higher prevalence of erythroderma than other studies. Sehgal and Srivastava recorded the incidence of erythroderma from the Indian subcontinent as 35 per 100,000 dermatologic outpatients. But there are conflicting views over role of histopathology as some studies were unrewarding.[5]

The study was performed to find out the causes of erythroderma in north-west part of India, to find out the epidemiological, clinical profile of these patients and histopathological correlation.

Material and Methods

The study was conducted from January 2013 to December 2014. All cases of erythroderma attending skin outpatient department were included in the study. A thorough history followed by a meticulous general, physical and dermatological examination was to form a clinical diagnosis. Laboratory investigations including complete hemogram, blood glucose, blood urea, serum creatinine, liver function test, serum electrolytes and chest radiograph were done in all cases. Other relevant investigations including abdominal ultrasound, peripheral blood smear, fine needle aspiration cytology (FNAC) of lymph nodes and CT scan were done wherever needed. A four millimeter skin punch biopsy was performed in all patients from two representative sites. The slides were independently analysed by three different observers without relevant clinical information. Histopathological diagnosis was correlated with clinical diagnosis to make final diagnosis.

Clinical Features	Psoriasis	Eczema	Drug Induced	Lymphoma	Mycosis Fungoides	Idiopathic	Total	P Value
Itching	20	35	7	1	1	1	65	0.214
Fever	10	6	4	1	0	0	21	0.059
Shivering	12	10	4	1	0	0	27	0.151
Arthralgia	7	2	0	0	0	0	9	0.047
Edema	9	20	3	1	0	1	34	0.575
Lymphadenopathy	14	22	3	1	1	0	41	0.409
Palmoplantar Keratoderma	10	5	0	0	0	0	15	0.023
Nail Changes	17	20	1	0	0	0	38	0.005
Pallor	7	10	2	1	0	0	20	0.72

TABLE 1: Clinical Profile of Patients

Results:

A total of 66 patients were enrolled in this study. The mean age of the study group was 53.7±16.56 in years (range : 14 to 86 Years). Males outnumbered females in a ratio of 3.4:1. The total duration of disease ranged from 10 days to 20 years with an average duration of 3.9 years. The exacerbation of disease was from 7 days to 1 year with a mean of 1.9 months. Majority of male patients were involved in outdoor activities and were farmers (39.4%) and laborers (16.67%). Majority of female patients were housewives (80%).

Most common aggravating factor was seasonal variation. Seasonal exacerbation was present in about 51.51% of patients. Winter exacerbation was present in 40% of psoriasis patients and 2.8% of eczema patients. Summer exacerbation was present in 54.2% of eczema patients and 25% of psoriasis patients. History of atopy was present in 19 patients. Drugs were responsible in 8 patients.

History of preexisting skin disease was present in 30 patients (62.1 %). Other co-morbidities like hypertension were present in 26 patients (39.3%), diabetes in 4 patients (6.06%), and tuberculosis in 4 patients (6.06%). The site of onset of erythroderma was scalp and face in 28 patients (42.4%), extremities in 27 patients (40.9%), and trunk & abdomen in 11 patients (16.67%).

Most common clinical features were itching (98.48%), fever (31.8%), shivering (40.9%), arthralgia (13.63%), lymphadenopathy (62.1%), edema (51.5%), palmoplantar keratoderma (22.7%) and nail changes (57.8%) [Table1]. The clinical finding in psoriasis, dermatitis and drug induced ertythroderma have been described in detail in Table 2. Most common nail change was beau's line followed by shiny nails, yellowish discoloration of nails, subungual hyperkeratosis, pitting, and onycholysis. In 3 patients, twenty nail dystrophy was present. Investigations revealed anemia in 33.3%, increased ESR in 37.9%, abnormal TLC in 18.1%, abnormal LFT in 15.2%, hypoalbuminaemia in 34.8% and abnormal RFT in 9.09% of cases.

Clinico-pathological correlation occurred in about 67% (range: 63.6% to 68.2%) of patients with a kappa score ranging from 0.495 to 0.572. It was of moderate agreement. In psoriatic erythroderma patients, we were able to elicit munromicroabscess, dilated blood vessel and suprapapillary thinning in 60%, 80% and 65% cases respectively. Presence of mitotic cells was also specific for psoriasis but it was present in only 20% of cases. The biopsies of drug induced erythroderma patients had necrotic keratinocyte, basal cell vacuolization and eosinophils in infiltrate in 62.5%, 75% and 87.5% of patients respectively. Spongiosis was present in 62.8% of patients of eczema. But it was also present in 50 % of drug induced erythroderma patients and 10% of psoriasis patients. [Table 3] In five patients, clinical findings mismatched histopathological findings. In these patients, clinical findings suggested the diagnosis of eczema but it came out psoriasis histopathologically. [Table 4]

Most common cause of erythroderma in this study was eczema of various types (53.03%), followed by psoriasis (30.30%), drug induced (12.12%), lymphoma (1.515%), mycosis fungoides (1.515%) and idiopathic (1.515%). [Figure 1]

Discussion

The approach to patients with erythroderma depends on their previous dermatologic background. Patient with a preexisting dermatoses are easy to diagnose. Otherwise, erythroderma remains a diagnostic challenge, especially in those patients without history of dermatologic diseases and who deny having recently taken any medications.[6]

In this study, the mean age of diagnosis was 53.7±16.56 years (range: 14 to 86 Years) with men outnumbering women in a ratio of 3.4:1. This is in accordance with various previous studies.[3, 6, 7] However, in a recent study by Hulmani et al, male to female ratio of 14:1 was noted.[8]

In our study, most common cause of erythroderma was air borne contact dermatitis compared to Hulmani et al where most common etiology was psoriasis.[8] The different etiologies of erythroderma found in various studies has been summarized in Table 5.

S.No.	Points	PSORIASIS	DERMATITIS	DRUG
1	Age group	Any	Predominantly Elderly age group(5- 7 decade)	Any
2	Seasonal Exacerbation	winter	Summer and spring	No
3	Preexisting disease	May	May	No
4	History of drug	No	No	Yes
5	Disease onset	Insidious	Mostly chronic	Acute
6	Site of onset	Predominantly extensors and scalp	Exposed surfaces Involvement of eyelids, other folds(ABCD)	Sometimes cephalo-caudal
7	Scaling	Thick, large, silvery	Fine	Fine
8	Erythema	Red, fiery red	Fading red	Red/ fading red
9	Nose sign	Absent	Seen	Absent
10	Oozing, cracking, fissuring, excoriation	Absent	Present	Absent
11	Vesiculation	Absent	May be	May be
12	Pustulation	May be	Generally Absent except (Secondary infection)	May be
13	Skin	Dry erythematous	Glossy skin with lichenification	Dry erythema
14	Itching	Mild	Severe	Mild
15	Palmoplantar keratoderma	May be (in some studies it is common)	Common	Absent
16	Fever	May be	Gen. Absent	May be
17	Shivering	++	++	+
18	Edema	+	+	++
19	Arthralgia	++	Gen. absent	++
20	Lymphadenopathy	++	+	++
21	Nails	++	-	-
	Pitting	++	-	-
	Beau's lines	-	++	-
	Onycholysis	++	May be	Absent
	Shinning in nails	-	++	-
	Subungual hyperkeratosis	++	+	-

TABLE 2 : Differences in clinical profile of erythroderma patients

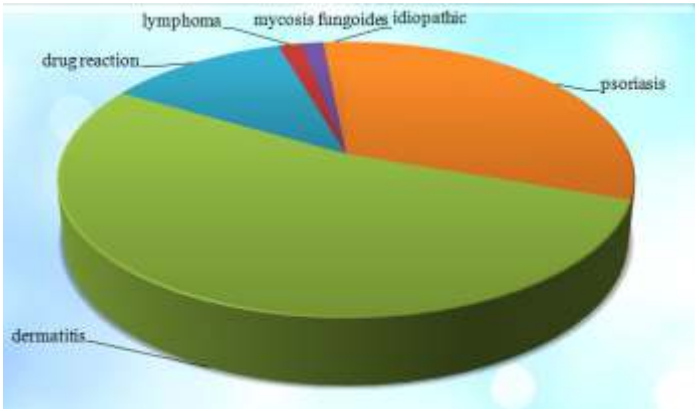

Figure 1: Etiology of erythroderma

Lymphadenopathy was seen in 62.1% of our cases. Previous studies have reported its prevalence varying from 19% to 55%.[6, 8–10] Nail changes were seen in 57.8% of patients. Nail changes were beau's lines, shinning in the nails, subungual hyperkeratosis, pitting, yellowish discoloration and onychodystrophy. Similar findings were present in other studies.[8, 10]

Clinico-pathological correlation occured in about 67% (range: 63.6% to 68.2%) of patients. In a similar study by Zip et al, [4] each set of pathological diagnoses was compared with the final discharge diagnoses, a positive correlation of 86% was observed in the nonblinded (original) diagnostic group as opposed to 66% in the blinded group. The results of blinded group were in accordance to our study which is also a blinded study. In another study by Vasconcellos et al,[1] one or more skin biopsies along with clinical findings were diagnostic or suggestive of the underlying disease in 63.6% of the cases. Khaled et al [14] reported positive clinico–histological correlation in 77%, Jun Li et al [6] in 55.56% and Rym et al[11] in 74% of patients.

The histopathology of eythroderma differs depending on the underlying diagnosis. In our study, in psoriasis patients the findings observed were munromicroabscess (60%), dilated blood vessel (80%), suprapapillary thinning (65%) and mitotic cells (20%). In similar study by Zip et al,[4] biopsies of psoriatic erythroderma patients revealed suprapapillary thinning, dilated blood vessel and munromicroabscess in 69%, 81% and 69% of patients respectively.

The biopsies of drug induced erythroderma patients had necrotic keratinocyte, basal cell vacuolization and eosinophils in

Histopathological Findings	Drug Induced	Psoriasis	Eczema
	%	%	%
Hyperkeratosis	87.5	100	94.2
Parakeratosis	87.5	100	74.2
Munro's microabscess	0.0	60	0.0
Granular layer			
Normal	87.5	15.0	82.8
Hypergranulosis	12.5	5.0	11.4
Hypogranulosis/Absent	0.0	80.0	5.8
Acanthosis	62.5	100.0	94.2
Regular	0	65.0	8.5
Irregular	62.5	35.0	85.7
Mitotic cell	0.0	20.0	0.0
Spongiosis	50.0	10.0	62.8
Suprapapillary thinning	0.0	65.0	2.9
Necrotic keratinocyte	62.5	0.0	0.0
Basal cell vacoulisation	75	0.0	11.4
Exocytosis	0.0	10.0	22.8
Epidermotropism	0.0	0.05	2.9
Dilated blood vessels	0.0	80.0	0.0
Infiltrate			
Lymphohistiocytic	37.5	95.0	62.9
Lichenoid	37.5	0.0	0.0
Mixed	12.5	5.0	28.5
Mononuclear	12.5	0.0	8.6
Eosinophilic infiltrate	87.5	5.0	11.4
Melanin incontinence	75.0	10.0	8.6

TABLE 3: Histopathological findings

infiltrate in 62.5%, 75% and 87.5% of patients respectively. In study by Zip et al [4], necrotic keratinocyte and eosinophils in infiltrate were present in 50% of cases each. Microscopically, eosinophils in infiltrate, necrotic keratinocyte and basal cell vacuolization were the most specific findings to diagnose a case of drug induced erythroderma. In previous studies, spongiosis was one of characteristic finding to diagnose a case of erythroderma due to eczema, present in 62.8% patients. But it was also present in 50 % of drug induced erythroderma patients and 10% of psoriasis patients. Thus as spongiosis was not one of the specific findings to diagnose a case of erythroderma due to eczema we had to collaborate it with other findings for diagnosis. It is generally thought that oozing, cracking, fissuring, presence of beau's lines, nail discoloration are useful for diagnosing eczema in erythroderma patients, but in our study we found that these changes were also present in some patients of psoriasis. Thus these changes might help in diagnosis of eczema but they are not diagnostic and a biopsy should be done in all patients to confirm diagnosis even when sure clinically.

Comparison of our etiologic diagnosis with the previous studies is compiled in table 4. In our case series, most common final diagnosis was eczema. It is quite different from other studies where it constituted a minority group.

Conclusion

Although clinical diagnosis is possible in most cases, histopathology is required to corroborate with clinical diagnosis and to avoid any misdiagnosis as clinical features might overlap, for example psoriasis Versus eczema. Microscopical clues that might help in diagnosis are munromicroabscess, dilated blood vessel and suprapapillary thinning for psoriasis and necrotic keratinocyte, basal cell vacuolization and eosinophils for drug induced erythroderma patients. Erythroderma still remains a challenge and requires skills of the dermatologist.

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S. No.	Itching	Oozing	Lichenification	Seasonal exacerbation	H/O Atopy	Previous H/O	Palmoplantar involvement	Nail changes	Scaling	Histopathological Diagnosis	Clinical diagnosis
1.	Mild	+	-	-	-	-	-	Beau's line	Fine	Psoriasis	Dermatitis
2.	Severe	+	+	Summer	+	Eczema	-	Yello discoloration	Fine	Psoriasis	Dermatitis
3.	Severe	+	-	Summer	-	-	-	-	Fine	Psoriasis	Dermatitis
4.	Mild	+	+	Summer	+	+	+	Beau's line	Fine	Psoriasis	Dermatitis
5.	Severe	+	+	-	-	Eczema	-	Beau's line	Fine	Psoriasis	Dermatitis

TABLE 4: clinical findings of patients with mismatched histopathological findings

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Study causes	Pal et al[10]	Rym et al[11]	Bandyopadhyay et al[9]	Sudha et al [12]	Chaudhary et al[13]	Hulmani et al[8]	Our study
Psoriasis	37.8	51.25	33.33	32	40	33.33	53.03
Eczema of various types	12.2	7.5	4	12	20	20	30.3
Ichthyosis	7.8	0	1.33	0	0	0	0
Pityriasis rubra pilaris	2.2	5.25	1.33	0	0	3.33	0
Scabies	2.2	1.25	3.33	0	0	0	0
Pemphigus foliaceus	5.6	6.25	5.33	4	0	0	0
Lichen planus	0	1.25	0	0	0	0	0
Atopic Dermatitis	0	0	13.33	8	6.66	6.6	0
Other Dermatoses	6.6	3.75	0	8	0	0	0
Drug Reaction	5.5	11.25	12	24	10	16.6	12.1
Malignancy	5.5	8.75	2.67	4	6.66	3.3	1.5
Mycosis fungoides	0	0	0	0	0	0	1.5
Idiopathic	14.6	7.5	21.33	08	16.6	16.6	1.5

TABLE 5: Comparison of different etiology of erythroderma in various studies

LETTER TO EDITOR
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MISUSE OF TOPICAL CORTICOSTEROIDS ON FACE: A HOSPITAL BASED CLINICOEPIDEMIOLOGICAL STUDY

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Sir,

Misuse of topical corticosteroids (TCS) is common in India. Unrestricted sale and lack of adequate knowledge about TCS are largely responsible for it. Inappropriate use of TCS results in several cutaneous and sometimes, even systemic adverse effects.[1] The facial skin, on account of its lesser thickness, is extremely vulnerable to these adverse effects.[1,2] Therefore, this study was carried out to determine various adverse effects of TCS on face and various factors associated with it.

All patients presenting to the dermatology OPD, who were found to be using TCS containing products on face inappropriately, were included in the study. The relevant details were recorded in a proforma for analysis and interpretation of data. The data were analyzed using the chi-square test and p value < 0.05 was considered to be significant. The study duration was three months.

A total of 163 patients were included in the study, including 84 males and 79 females (Table 1). Amongst these patients, 140 (85.89%) had applied TCS on face alone, while the remaining had applied on other body parts as well. The most common primary dermatosis was acne (71; 43.56%), followed by melasma and other pigmentary disorders (43; 26.38%), while 20 (12.27%) patients had been using these products without any disease (Table 2). The duration of use varied from 1 week to several years (Table 3). The most common TCS compound in the study was betamethasone valerate (69; 42.33%) followed by clobetasol propionate (57; 34.97%) [Table 4]. The composition of various products used by patients is shown in table 5. Various cutaneous adverse effects on facial skin are depicted in table 6 and figures 1 – 5.

Age Group (in years)	M	F	Total	Percentage
0 - 10	1	0	1	0.61
11 - 20	40	26	66	40.49
21 - 30	34	39	73	44.79
31 - 40	4	12	16	9.81
41 - 50	2	2	4	2.45
51 - 60	3	0	3	1.84
Above 60 yrs	0	0	0	0.00
Total	84	79	163	100.0

Table 1: Age and sex distribution of patients with TCS abuse

Primary dermatosis	M	F	TOTAL	Percentage
Acne vulgaris	41	30	71	43.56
Melasma and other pigmentary disorders	17	26	43	26.38
Dermatophytosis	20	7	27	16.56
Nil	6	14	20	12.26
Herpes simplex	0	1	1	0.61
Varicella scars	0	1	1	0.61
Total	84	79	163	100.0

Table 2: Primary dermatoses for which TCS preparations were being used.

Duration of use	M	F	TOTAL	PERCENTAGE
< 1 WEEK	2	2	4	2.45
1 WEEK TO 1 MONTH	33	24	57	34.97
1 – 3 MONTH	23	13	36	22.09
3 – 6 MONTH	19	16	35	21.47
6 – 12 MONTH	4	11	15	9.20
>1 YEAR	3	13	16	9.81
TOTAL	84	79	163	100.00

Table 3: Duration of TCS abuse.

Compound	M	F	T	PERCENTAGE
CLOBETASOL PROPIONATE	37	20	57	34.97
BETAMETHASONE VALERATE	26	43	69	42.33
MOMETASONE FUROATE	16	14	30	18.40
BECLOMETHASONE DIPROPIONATE	5	0	5	3.06
HYDROCORTISONE ACETATE	0	2	2	1.22
TOTAL	84	79	163	100.00

Table 4: Topical corticosteroid compound in the products.

COMPOSITION	M	F	TOTAL	PERCENTAGE
Only steroid	5	9	14	8.59
Steroid + antibiotic	22	30	52	31.90
Steroid + antifungal	2	0	2	1.22
Modified Kligman formula	16	15	31	19.01
Steroid + antifungal + antibiotic (3 drug)	8	6	14	8.59
4 drug combination	31	15	46	28.22
Miscellaneous	0	4	4	2.45
Total	84	79	163	100.00

Table 5: Composition of products used by patients.

Adverse effect	M	F	Total
Acneiform eruption/comedones/flare of acne	53	38	91
TSDF	15	41	56
Modified dermatophytosis	20	7	27
Hypertrichosis	0	8	8
Telangiectasia	2	5	7
Pigmentary change	6	6	12
Others	4	4	8

Table 6: Cutaneous adverse effects noted in the study.

Easy availability of TCS preparations, even without a prescription, has resulted in their widespread misuse in India. Various studies focusing on cutaneous adverse effects of TCS compounds have been conducted in India.[3–6] Since facial skin is relatively thinner, it is more vulnerable to these adverse effects.

In previous Indian studies,[4-6] proportion of females has been reported to be relatively higher, but in the present study, it was found to be almost equal (M: F:: 1.063). This can be attributed to overall low attendance of females in OPD, as well as, it might reflect increased cosmetic concern amongst males too, thus making them vulnerable to misuse of various products available in the market. This fact is further substantiated by the fact that the most common affected age-group was 21-30 years; an age-group which is commonly concerned about one's appearance.

The most common primary dermatosis in the present study was acne followed by melasma. This is in accordance with a previous study [6] conducted in Punjab. Although some patients (12.27%) had been using TCS as a part of routine skin care, without any skin disease, the proportion of such patients was relatively small in the current study.

The most commonly used TCS compound noted in the study was betamethasone valerate followed by clobetasol propionate. Similar results were reported by Jha et al.[4] Further analysis of product composition revealed that steroid with antibiotic combination was the most commonly (31.9%) used followed by an irrational 4 drug combination containing clobetasol, ofloxacin, ornidazole and terbinafine (28.22%). Modified Kligman formula was being used by 19.01% cases.

The most common adverse effect noted in the study was aggravation of acne, comedone formation or acneiform eruption followed by topical steroid – dependent facies (TSDF) and steroid modified dermatophytosis.

TSDF is a relatively newer entity in context of TCS induced local adverse effects, described in 2008.[2] It is defined as the semi-permanent or permanent damage to the skin of the face precipitated by the irrational, indiscriminate, unsupervised, or prolonged use of TCS resulting in a plethora of cutaneous signs and symptoms and psychological dependence on the drug.[2] Various terms were initially coined for these cutaneous signs and symptoms such as dermatitis rosaceiformis steroidica, red skin syndrome and steroid-induced rosacea-like dermatitis.[7–9] In the present study, TSDF was noted in approximately one-third cases. These patients presented with complaints of facial erythema, which was either persistent or aggravated with sun exposure, burning, itching, stretching sensation, scaling and thinning of skin. These symptoms were promptly relieved on self application of TCS and recurred on stopping, which forced the patients to use them again and again, thus creating a vicious cycle of TCS abuse and dependence. Here the classical history of prompt improvement on reapplication of TCS points towards steroid dependence.

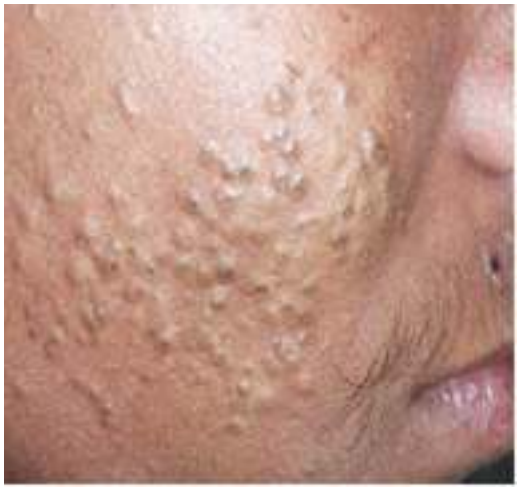

Figure 1: Aggravation of acne and comedone formation

Previous studies indicate that self-medication, trust on chemist and advice of family and friends, along with ignorance of non-dermatologist prescribers contribute to misuse of TCS.[5, 10-12] These prescribers usually try to cover up all possible etiologies as the diagnosis is not known, thus prescribe irrational combinations. Also, we need to note that the cost of rational products for the treatment of acne, melasma and dermatophytosis is relatively very high.[12] Thus, prompt relief in symptoms at a lower cost allures the patients to use these products again and again. Lack of adequate dermatologists across the country and lack of awareness about adverse effects of topical medications has

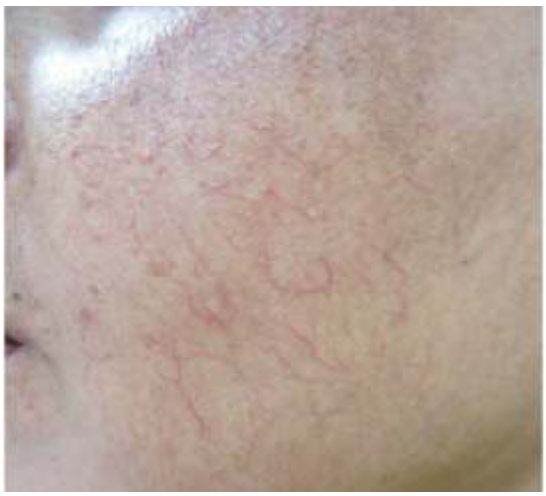

Figure 2: Telangiectasia on face.

resulted in massive misuse of TCS preparations.

Conclusion

Misuse of TCS is rampant across India. Adequate regulation to stop the sale of TCS preparation as OTC drug is the need of the hour. At the same time, prescribers need to be made aware of the possible consequences of using TCS inappropriately. Further, a stronger referral system to the dermatologist needs to be developed.

Limitations

Several patients in whom the composition of the used products could not be identified, were excluded from the study, thereby decreasing the actual number of patients with TCS abuse.

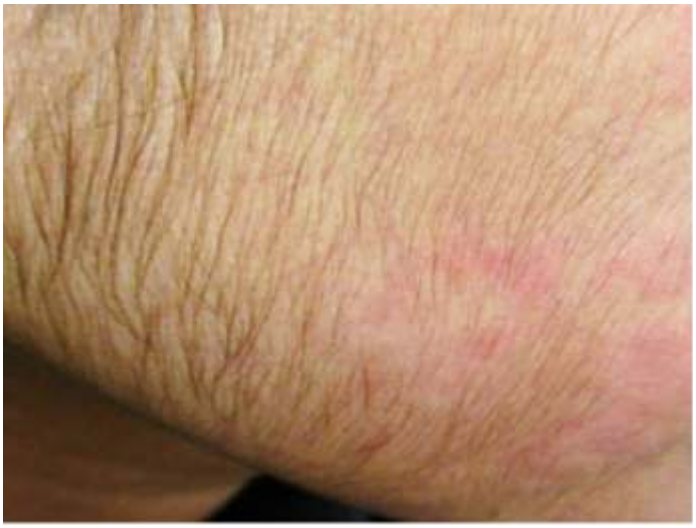

Figure 3: Hypertrichosis with modified Tinea faciei.

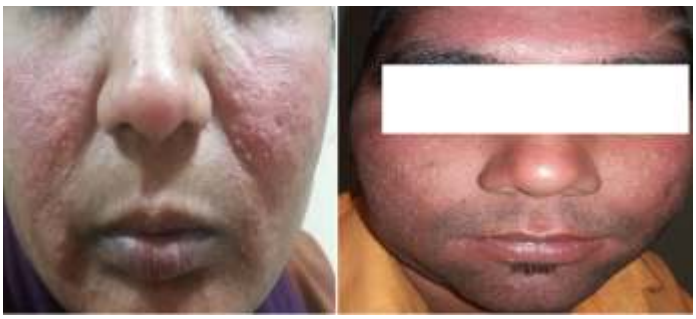

Figure 4: Topical steroid – dependent facies.

Figure 5: Steroid modified Tinea faciei.

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LETTER TO EDITOR
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COMPARATIVE STUDY OF EFFICACY AND SAFETY OF LACTIC ACID VERSUS GLYCOLIC ACID CHEMICAL PEELS IN THE TREATMENT OF MELASMA

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Sir,

Melasma is a common, acquired, symmetric hyperpigmentation commonly involving the cheeks, forehead, upper lip, nose, and chin.[1] It predominantly affects women (90%) and is common in individuals with Fitzpatrick skin type IV-VI. Different modalities like keratolytics (tretinoin, resorcin, glycolic, and trichloroacetic acids, etc.) and depigmenting agents (hydroquinone, kojic and azelaic acids) are being used but chemical peeling provides more rapid response than topical therapy.[2] Chemical peels create injury at a specific skin depth and causes exfoliation that stimulates new epidermal growth and collagen with more even distribution of melanin.[3] Most commonly used peels include phenol, trichloroacetic acid (TCA), alpha hydroxyacids (AHAs), and beta hydroxyacids.

Sixty patients with moderate to severe melasma of epidermal variety only (differentiated by Wood's lamp examination, according to darkness (D) of pigmentation) were included in the study after getting due ethical clearance from our institute ethics committee. Treatment groups for lactic acid peel and glycolic acid peel were selected randomly and divided into two groups of 30 each. Written informed consent was taken from all the patients included in the study. Cases of dermal melasma were not included in this study because both are superficial to medium depth peels and are not effective for dermal melasma. Patients with a history of herpes, taking oral contraceptive pills or isotretinoin, pregnancy, lactation, history of keloids or hypertrophic scars, concomitant systemic or skin disease and those with unrealistic expectations were excluded from the study.

Melasma Area and Severity Index (MASI) score of the right and left cheeks were calculated for each patient at baseline, at the beginning of each peeling session, and at the end of follow up, along with photography.

The response in each patient was graded as: no response (no change in MASI score at the end of three peels); mild response (less than 25% change); moderate response (25 to <50% decrease in MASI); good response (50 to <75% decrease); very good response (more than 75% decrease). In pre-peel session patients were advised to apply kojic acid 2% or tretinoin 0.025% at night and topical sunscreen daily [SPF-15].

The first group was treated with 35% Glycolic acid after a test peel and second group with 92% Lactic acid. In Glycolic acid group after 3-5 min of application of peeling agent, washing with neutralizer (sodium bicarbonate) was done. In Lactic acid group an erythematous response was awaited within 2 to 3 minutes; if not, then a second layer of application was applied to obtain the desired response and left for 10 minutes after that, it was washed off with water. Peels were performed every 2 weeks for six sessions and participants were instructed to apply sun block cream and emollients. No topical Hypo pigmenting agent was applied.

The primary objective of this study was to assess the degree of improvement in pigmentation objectively using MASI at baseline, 2, 4, 6, 8, 10 and 12 weeks. Color photographs were taken of all patients at baseline and 1 month after the last peel.

Paired t-test was used to statistically analyze the change in the mean MASI scoring resulting from treatment in the two groups and to analyze comparative decrease in MASI scoring between the two groups. All the patients in the two groups were examined for any side effects like allergic reactions, hypo or hyperpigmentation, burning, persistent erythema, acneiform eruptions and scarring.

60 patients were included in the study with 54 females and only 6 males, male: female ratio, 1:9. Maximum number of patients was in age group 31-40 year (45%). Mean age of patients in GA group was 32.77±6.88 and in LA group was 30.73±6.03 with p-value of 0.24702 making the two groups statistically comparable. The duration of the disease, in both the groups, in our study ranged from 2 months to 12 years with the mean duration of disease being 3.04±1.927 years in group 1 (Glycolic acid group) and 2.79±2.69years in group 2 (Lactic acid group). The difference between the mean duration of disease in the two groups was statistically not significant (p=0.6687), thus both the groups were comparable in terms of duration of disease. Most common pattern observed was Centro facial (58.3%) followed by malar 35% [Figure 1]. Only 15% patients gave family history of melasma. Pregnancy had no significant association with melasma and only 11(18.3%) patients gave a history of occurrence of melasma during pregnancy.

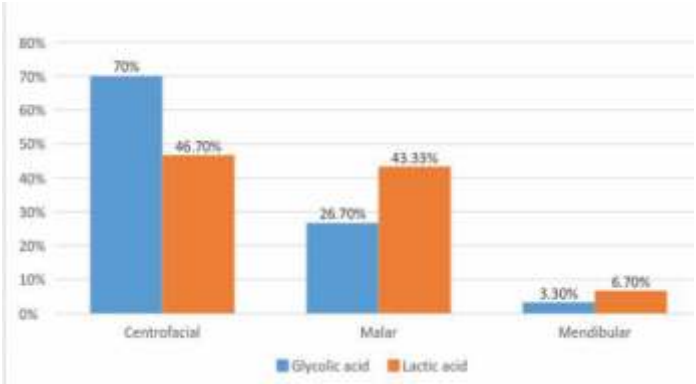

Figure 1: Clinical pattern of melasma in both Glycolic acid and Lactic acid group.

Objective response to treatment was measured by a decrease in MASI scoring after each peel session. Up to 2 week (1 peel) there was no significant response in both the groups (p>.05). After 12 weeks reduction in MASI was 54% (from 22.29 to 10.12) in GA group (Figure 2a, 2b and 3a, 3b) and 68% (from 22.15 to 6.91) in LA group (Figure 4a, 4band 5a, 5b) which was highly significant (p<.001). The MASI scores at baseline, 2 4, 6 8, 10 12 weeks were as shown in [Figure 6].

Thus lactic acid 92% showed better efficacy compared to Glycolic acid 35%. When we assessed the adverse effects, the frequency of serious adverse effects was very low in both the groups [Figure7]. Common adverse effects were mild burning (36.67% of patients in LA group and 50% in Glycolic acid group) only at the time of application of peel but resolved after ice cooling, calamine application and use of sunscreens. Erythema (6.67% in LA and 20% in GA) which resolved after applying 1% hydrocortisone cream for 3-4 days and hyperpigmentation was seen in 3.33% in GA but none of the patient in LA group.

Recurrence at 3 months follow up was seen in 25 patients in GA group in form of increase in MASI score which was significant (p<.05). No relapse was seen in LA group.

Melasma is a symmetric progressive hyperpigmentation of the facial skin that occurs in all races but observed more frequently in darker skin phenotypes. There are three clinical patterns -

Figure 2a, 2b: Melasma in 35 year old female before and after treatment with GA peel.

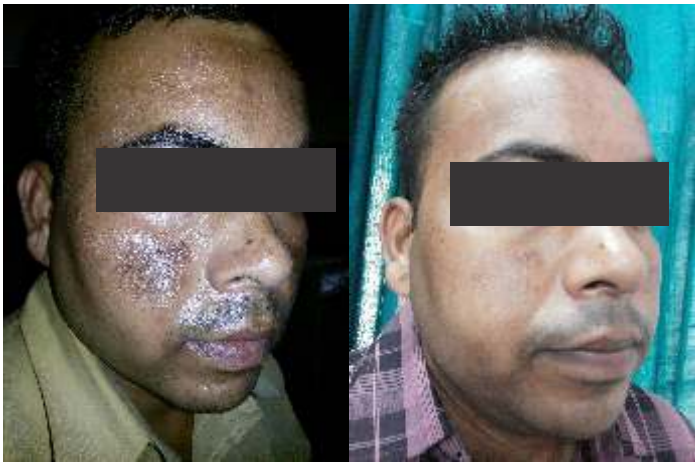

Figure 3a, 3b: Melasma in 30 year old male before and after treatment with GA peel.

centrofacial, malar, and mandibular - depending upon the area of localization.[1] By Wood's light examination, melasma can be classified into epidermal, dermal or mixed type. Different modalities as depigmenting agents, laser, and chemical peeling have been used alone and in combination for the treatment of melasma.[4] Chemical peels are often used as an adjunct to medical treatment because they produce complementary rapid therapeutic effects and improves skin appearance and texture.[5] Peels allow topical agents to penetrate more efficiently into the skin and may improve post inflammatory hyperpigmentation.[6]

Chemical peels create controlled chemical burn of the skin and produces partial thickness wound that heals by secondary intention. The end results are thinning of stratum corneum, epidermolysis, dispersion of basal layer melanin, regulation of epidermal thickness and laying down of new collagen and ground substance in dermis.[7] Depending upon the depth of peeling achieved, the chemical peeling agents are classified into very superficial, superficial, medium and deep peels.[8] Chemical peels useful in treating melasma are trichloroacetic acid, Jessner's

Figure 4a, 4b: Melasma in 28 year old female before and after treatment with LA peel.

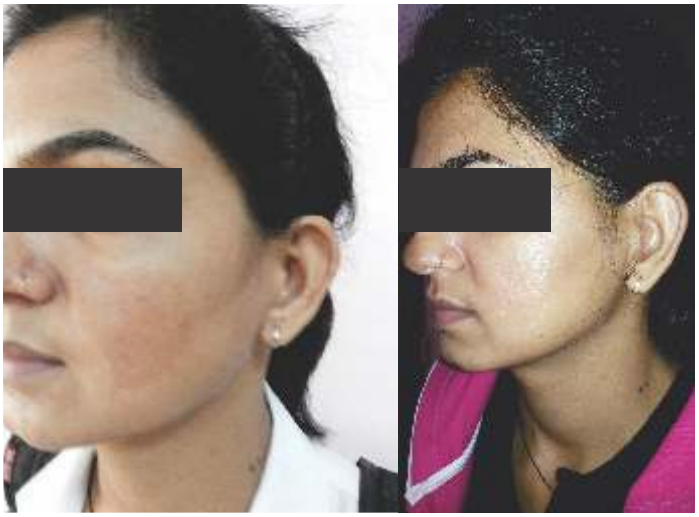

Figure 5a,5b: Melasma in 25 year old female before and after treatment with LA peel.

solution, alpha-hydroxy acid preparations, and salicylic acid, alone or in various combinations. Alpha-hydroxy-acids (AHAs) have been the most commonly used agents for superficial peelings. Both the agents used in this study, LA 92% and GA 35%, are superficial peels.

Glycolic acid (GA) is obtained from sugarcane and is the simplest and most-used alpha-hydroxy acid peel.[9] GA is a popular peel agent because it has the smallest molecular weight amongst all the alpha-hydroxy acids and penetrates skin easily.[6] Fabbrocini, in 2009, classified glycolic peels as: very superficial (30%–50% GA, applied for 1–2 minutes); superficial (50%–70% GA, applied for 2–5 minutes); and medium depth (70% GA, applied for 3–15 minutes).[10] GA peels have anti-inflammatory, keratolytic, and antioxidant effects. GA targets the corneosome by enhancing breakdown and decreasing cohesiveness, causing desquamation.[11] GA peels need to be properly neutralized to stop acidification of the skin. In a study by Sarkar et al, modified Kligman's formula (2% hydroquinone, 0.025% tretinoin, and 1% mometasone), was compared with GA peels (30% GA for the first three sittings; 40% GA for the next three sittings), combined with the modified Kligman's formula and GA peel group showed more rapid and greater improvement ($P < 0.001$).[2]

Alpha hydroxy acids have been used as effective peeling agents in a variety of conditions including melasma, but the clinical

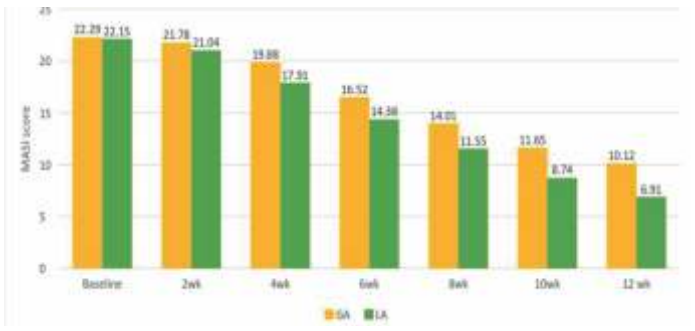

Figure 6: Comparison between change in Mean MASI score of Glycolic acid and Lactic acid with treatment duration.

experience is limited to glycolic acid only,[12] which is expensive and may not be available in every center.[13] Lactic acid, also an alphahydroxy acid having activities similar to GA, it is a non-costly and readily available agent but has not been used extensively as a peeling agent in the treatment of melasma. Further, because of its large molecular size, there is less penetration with an additional advantage of being more hydrating and less irritating.[14] The first study in melasma was done by Sharquie et al[15] and found it to be a safe and effective peeling agent for melasma in dark skin. In their study of 20 patients, 92% pure lactic acid was applied for a maximum of six sessions, and a significant fall in MASI (56%) was observed in all the 12 patients who completed the study. Further, LA was compared with Jessner's solution in melasma, and and it was as effective as Jessner's solution.[16]

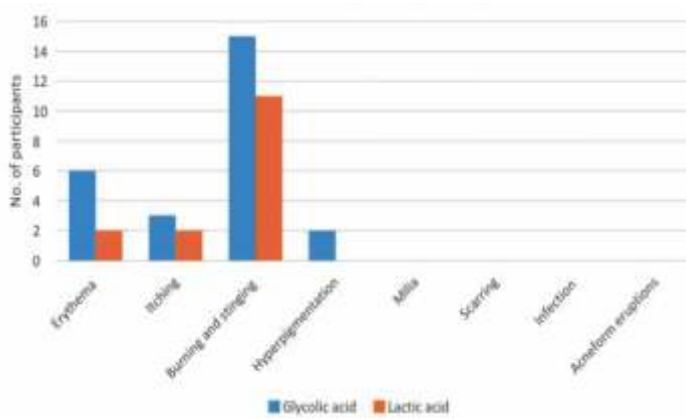

Figure 6: Comparison of adverse effects of peel in both the groups.

However, the clinical experience of LA peeling in melasma is very limited and to the best of our knowledge, there is no study available in the literature comparing the efficacy and safety of Lactic acid peeling with Glycolic acid peeling in melasma. This prompted us to undertake a clinical trial comparing the efficacy and safety of Lactic acid peeling with Glycolic acid peeling in the treatment of melasma.

Limitation of our study was short follow up period of 3 months only so we were not able to detect late recurrences that may also occur in LA peel.

Both the Glycolic acid 35% and Lactic acid 92% are effective peeling agents in epidermal melasma. Both significantly reduces MASI scores ($p<.001$). At the end of treatment LA peel showed better efficacy with rapid rate of clinical improvement. Side effects were seen with both peeling agents but less commonly with LA. Side effects were mild and not significant. More studies need to be conducted with different concentrations of GA and Lactic acid on larger samples and in other pigmentary disorders.

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LETTER TO EDITOR
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LAMIVUDIN INDUCED DRUG RASH- A RARE ENTITY

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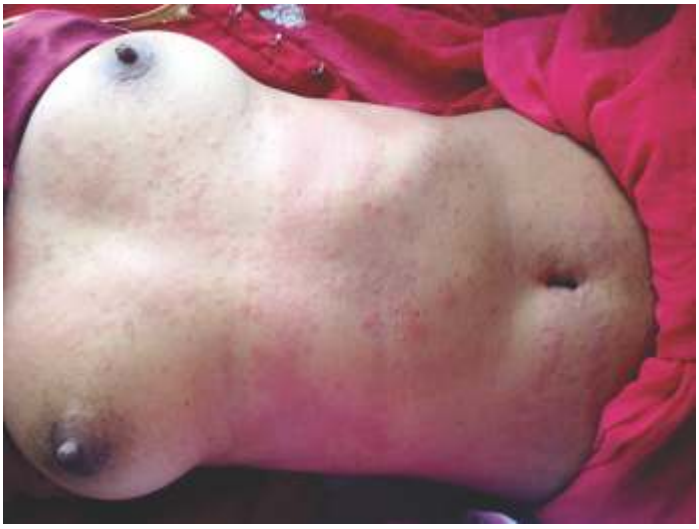
Sir,

The use of highly active antiretroviral therapy (HAART) has had an important impact on the course and treatment of disease and disease-related morbidity of HIV-infected patients, increasing their lifespan and quality of life.[1] However, these advantages have been accompanied with a marked increase in the number of adverse drug reactions, both minor and serious cutaneous adverse drug reactions[2]. Of all the drugs nevirapine is commonly responsible for cutaneous reactions and Lamivudine is considered relatively safer[3]. We are here present a case with cutaneous adverse drug reaction in a female patient with lamivudin.

A 40 year old immunocompromised female, receiving 1st line anti retroviral therapy i.e zidovudine, lamivudine & nevirapine (ZLN),within 10 days of initiation of therapy, she developed diffuse erythematous maculopapular pruritic rash which was first noticed by the patient on the abdomen then progressed to involve the chest, extremities and back (Fig. 1).

Figure 1: diffuse erythematous maculopapular pruritic rash



Mucosa was however spared and there were no constitutional symptoms. Systemic examination was also unremarkable. Laboratory investigations including complete hemogram, liver and renal function tests, urine examination were normal. Because of significant itching, all medications were stopped and she was given antihistamines like cetirizine and topical mid potent steroid with emolients. Within 1 week, patient became comfortable and rash disappeared. Two weeks later, zidovudine, lamivudine & efaverinz (ZLE) was initiated at the ART centre assuming nevirapine to be the offending agent, but within 48 hrs, patient redeveloped similar type of rash. Again ART was stopped and antihistamines were given. Once the rash completely subsided (within 2 weeks) we decided to hospitalize the patient for oral drug provocation test to find the offending agent. We suspected zidovudine and lamivudine to be the culprit drugs as they were common in both the regimen. Using the method described by Ramam et al[4] the patient was provocated first with half dose of Zidovudine followed by full dose on the next day. There was no reaction. On giving half dose of Lamivudine, she developed itching and faint rash over trunk within 12 hours and on giving full dose on the next day she developed diffuse maculopapular pruritic rash over trunk and extremities. Biopsy from the representative lesion showed sparse to moderately dense perivascular lymphocytic infiltrate in upper dermis, no eosinophils and interface changes. Spongiosis and parakeratosis were seen. The histopathology was suggestive of superficial perivascular dermatitis. Routine laboratory investigations were normal. There were no mucosal and systemic symptoms. But due to severe itching lamivudine was withdrawal and antihistamines were introduced leading to subsidence of rash with in 2days. Because the grade of the reaction was mild and controlled with antihistamines, she was continued on the same regimen i.e ZLE along with antihistamines. Patient is doing well since then.

HAART is effective and had lead to significant reduction in mortality and morbidity from HIV[5]. However each of these drugs has a potential to cause adverse cutaneous reactions including both minor and serious side effects. Skin reactions are the most common manifestation of drug hypersensitivity. These may present with exanthem without systemic symptoms or drug hypersensitivity syndromes typically manifesting as an erythematous, maculopapular confluent rash with constitutional features[6]. It is routine to consider the NNRTI component as the culprit agent when the patients are initiated simultaneously with zidovudine/stavudine, lamivudine, and nevirapine/efavirenz and cutaneous reaction appears. Nevirapine can cause skin rash in 17% to 32% of patients,13% of these are mild rashes.[7] Efavirenz can cause mild skin rash, with severe eruptions such as SJS, TEN and erythema multiforme being reported in 0.1% of patients[8]. However, the next common drug group suspected are the NRTIs. Zidovudine and lamivudine have been reported to be associated with skin rash very rarely. An extensive literature search revealed only a few case reports of drug allergy to lamivudine alone. Reaction of lamivudine was first reported in a 49 year old man, who developed an anaphylactoid reaction within 30 minutes of the first dose of lamivudine (150 mg). Withdrawal of the drug was followed by full recovery within the next 24 hours.[9] A case of contact dermatitis has been reported in a

healthcare worker taking lamivudine for post exposure prophylaxis.[10] There is another reported incident of severe skin eruption caused by lamivudine that needed discontinuation in a patient with chronic hepatitis B.[11] A case series of four patients has recently been published in which two patients developed SJS/TEN and other two developed maculopapular purpuric rash with Lamivudine.[3]

Since lamivudine is an effective, safe, and widely used antiretroviral drug, clinicians must be aware of the possibility of such severe adverse reactions to lamivudine, which requires drug cessation and administration of supportive treatment. Also, because lamivudine constitutes a part of all the first line ART regimens in India, a reaction to lamivudine warrants for higher centre referral and 2nd line medications which are associated with severe systemic side effects.

How to cite this article:
Agarwal C. Lamivudin induced drug rash- a rare entity. JDA Indian Journal of Clinical Dermatology 2018;1:87-88.

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LETTER TO EDITOR
Year: 2018 I Volume: 1 I Issue: 3 I Page: 89-90

CAPECITABINE INDUCED HAND-FOOT SYNDROME

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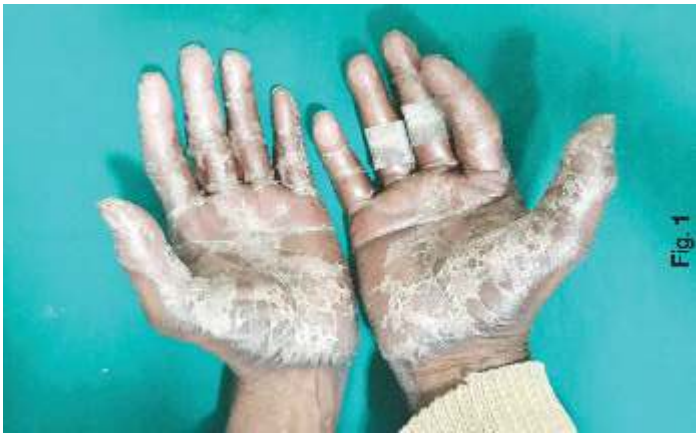
Sir,

The hand-foot syndrome, also known as palmar-plantar erythrodysaesthesia is a relatively common adverse effect of some chemotherapeutic drugs. 5-Fluorouracil, capecitabine, doxorubicin, docetaxel, idarubicin, and cytarabine are the most frequently involved agents.[1] This syndrome is clinically characterized by the gradual onset of bilaterally symmetric erythema, tenderness, tingling, numbness and desquamation over the palms and soles. Here we report a case of HFS along with keratoderma on capecitabine therapy for colorectal cancer. As clinical dermatologists, we should be aware of the cutaneous adverse effects of common chemotherapeutic agents which would help in timely diagnosis and management.

A 52 year old male presented to our outpatient department with 6 months history of darkening and thickening of skin of palms and soles associated with swelling and pain [Fig 1,2]. On detailed history, patient revealed taking chemotherapy for stage II colorectal adenocarcinoma (T2N0M0), which was operated upon 6 months back.



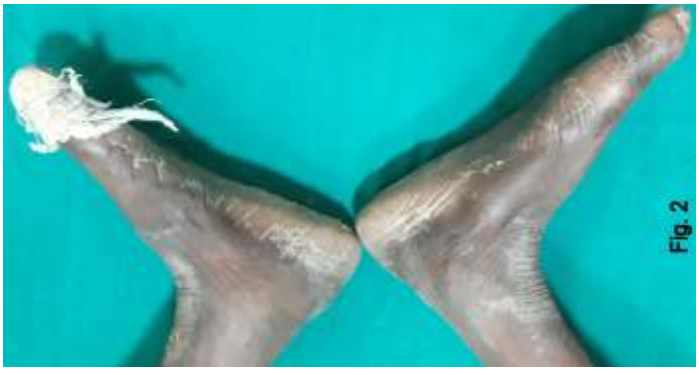


Figure1,2: darkening and thickening of skin of palms and soles

After the surgery patient was started on oral capecitabine in cycles of 14 days with 1 week gap in between cycles. The dose of capecitabine was slowly increased from 1500 mg to 2500 mg daily during this period. After 2 cycles of chemotherapy patient started to developed painful tingling sensation over palms and soles followed by desquamation, erythema, hyperpigmentation and stiffness of skin, affecting his daily routine activities.

On clinical examination, patient had erythema, keratoderma and desquamation of palms and soles, and dorsa of fingers, associated with dysaesthesia. Paitent refused biopsy of the skin of hand or feet. He was clinically diagnosed as a case of grade 2 HFS induced by capecitabine. Patient was treated with topical steroids, emollients and reduction of dose of capecitabine, and showed significant improvement within 3 weeks.

Hand foot syndrome (HFS), also called palmar-plantar erythrodysesthesia, is a cutaneous toxic reaction to several chemotherapeutic drugs. It was first reported by Zuehlke in 1974.2 Most common drugs implicated are 5-FU, Capecitabine, cytarabine, doxorubiocin, epirubicin, high dose interleukin–2, fluorodeoxiuridine, hydroxyurea, mercaptopurine, cyclophosphamide and Docetaxel.[1] The orally administered prodrug capecitabine is converted to 5-fluorouracil by the body. It is FDA approved for adjuvant treatment of colon cancer, metastatic colorectal cancer,and metastatic breast cancer. The hand-foot syndrome is a well-defined adverse effect of capecitabine; others being diarrhoea, nausea, and suppression of the bone marrow.

Histopathological changes include vacuolar degeneration of basal keratinocytes, dermal perivascular lymphocytic infiltration, apoptotic keratinocytes and dermal edema.[3]

The pattern of presentation appears to be drug-dose dependent. Several theories have been suggested regarding pathogenesis of HFS. The chemotherapeutic drugs secreted through sweat ducts accumulate and cause local direct damage to the keratinocytes. This theory explains the localization of the cutaneous changes to the hands and feet.[4] Some hypothesize increased expression of metabolizing enzyme thymidine phosphorylase that converts capecitabine to 5 fluorouracil and increases toxic injury to the tissue.[4]

The manifestations of HFS are classified into 3 grades according to their severity by the National Cancer Institute Cancer Therapy Evaluation Program.[5] Grade 1 shows erythema of lateral aspects of fingers, progressing to thenar and hypothenar eminences, with swelling, numbness, dysesthesia/paresthesia, and tingling, especially over the pads of distal phalanges. A similar picture may also be seen on the feet. However, this does not interfere with the patient's normal daily activity. Grade 2 shows a progression of manifestations of grade 1, with the pain,

tenderness and discomfort affecting daily activities. In grade 3, along with severe pain, there is also development of blisters, moist desquamation and ulcer formation.[6]

Along with HFS our patient also presented with palmoplantar keratoderma which has not been mentioned in the grading. It has been suggested that keratoderma can develop during capecitabine chemotherapy as a sequential event of HFS.[7]

Treatment includes cold compresses, topical emollient, antibiotics to prevent secondary infection, topical steroid, oral pyridoxine[8] and topical retinoids[9] in mild to moderate cases. Severe cases may require reduction of dose or discontinuation of the drug. In such cases, capecitabine may be cautiously re-introduced in a lower dose, which may gradually be stepped up.

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Sharma A. Capecitabine induced Hand- Foot Syndrome. JDA Indian Journal of Clinical Dermatology 2018;1:89-90.

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CASE REPORTS
Year: 2018 I Volume: 1 I Issue: 3 I Page: 91-93

CHONDROID SYRINGOMA – A RARE MIXED APPENDAGEAL TUMOUR AT MULTIPLE SITES

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Abstract

Chondroid syringomas are rare, generally benign, mixed tumors of the skin, composed of sweat gland elements. The tumor usually presents as benign asymptomatic, slowly-growing, subcutaneous or intradermal, single nodule occurring commonly over head and neck [3],[4]. We report a case of multiple, benign eccrine variant of Chondroid Syringoma, occurring on the scalp in 72yr old man, for its rarity and unique mode of presentation.

Key words: Chondroid syringoma, multiple, eccrine

Introduction

Chondroid syringomas are rare, generally benign, mixed tumors of the skin which were first described by Billroth (1859), that have both a benign and malignant form with an incidence of 0.01%–0.098% and of unknown etiopathogenesis. Hirsch and Helwig (1961) gave them the appellation chondroid syringoma, because of the presence of sweat gland elements which are set in a cartilaginous stroma.[1,2] Morphologically it is considered to be the cutaneous counterpart of the pleomorphic adenoma of salivary glands, but differs from it in that it rarely recurs.[3] Chondroid syringoma is often overlooked because of its rarity and the unremarkable clinical presentation. Hence, the tumour is typically diagnosed retrospectively from histopathological examination, and further classified as – apocrine and eccrine variants, apocrine being more common[3,4].

Case Report

A 72 yr old man presented with multiple, asymptomatic, painless, hard raised skin lesions on scalp of 30 years duration (fig 1-3). Lesions started as a few, small pea sized ones which progressively increased to the present size and were seen over scalp and forehead. There was no h/o any other skin or systemic complaints. Cutaneous examination revealed multiple (12), polysized (0.5cm to 2cm), round to oval shaped, skin coloured, hard to soft, non tender papules and nodules over vertex (fig 1), forehead(fig 2), right temporal area of scalp(fig 3).

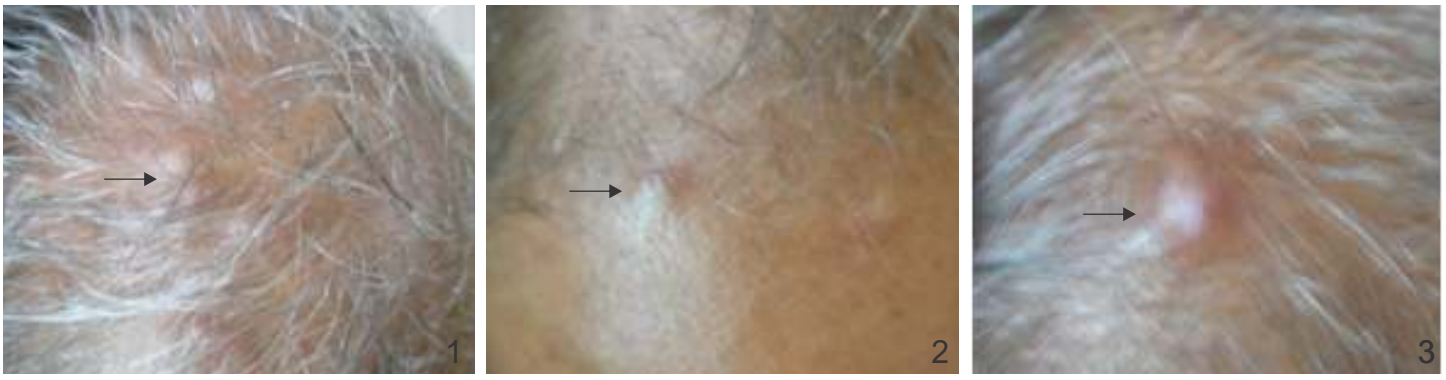
Skin over the lesions is not pinchable, and is mobile horizontally not vertically - not attached to the underlying structures. Excision biopsy was done and sent for histopathological examination keeping differential diagnoses:

(1)Sebaceous cyst (calcified) (2) Cylindroma (3) Spiradenoma.

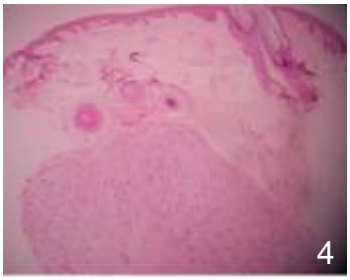
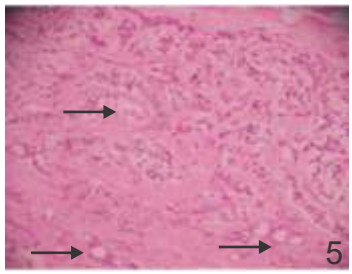
(4) Hidradenoma (5) Metastatic nodules (6) Calcinosis cutis.

Microscopic examination showed skin with underlying lesion proper, comprised of cells arranged in cords and trabecular pattern and with focal glandular pattern(fig 4-8).

Cords are separated by abundant mucoid stroma (fig 10). The cells are polygonal with bland nuclei and clear cytoplasm (fig 5 - 9). Few glands show bilayering of cells (fig: 6-8) No mitosis and no atypia. Eccrine variant- tadpole like acrosyringium (fig: 5-8) and negative for PAS diastase (rules out the apocrine variant). Positive Special stains for Chondroid Syringoma (fig: 9-12) S/O Benign Chondroid Syringoma having Eccrine variant.



Figs 1-3: Polysized, round to oval shaped, Papules and nodules.

Figs 4,5: Revealing tubule-cystic spaces in dermis (H&E;10x,40x)

Discussion

Hirsch & Helwig[1] proposed 5 criteria for diagnosis of chondroid syringoma: 1]Nests of polygonal/cuboidal cells. 2] Intercommunicating tubuloalveolar structures lined with 2 or more cuboidal cells. 3] Ductal structures composed of 1/2 rows of cuboidal cells. 4] Occasional keratinous cysts. 5] Matrix of of mixed chondroid and myxoid composition. In the present case it almost fulfilled the criteria.

Headington[3,4] divided CS into apocrine and eccrine variants based on luminal morphology.

1)The apocrine type: Is the most common type and demonstrates irregular branching tubules (tubulocystic pattern) lined by at least 2-cell-thick epithelium.

2) The eccrine type: characterized by rather uniform, all, round tubules that are evenly spaced within a myxoid-chondroid matrix. The present case was of eccrine type according to the Headington's criteria.

With constellation of clinical, histopathological and immunhistochemistry findings, following diagnosis was concluded: "Benign Multiple Chondroid Syringomas on scalp with Ecrrine differentiaton" . Because of malignant potential , the usual first-line treatment is total excision of tumor without destroying aesthetic and functional structure and regular follow up.[2]

We are presenting this rarest case of benign chondroid syringoma, with multiple nodules without any atypia over long duration. Our case is of interest because histopathologically both the luminal variants and the tadpole (comma shaped) appearance of tubules are classically seen in eccrine variant of Chondroid Syringoma, which itself is a rare entity.

Though 400 cases of chondroid syringoma are reported worldwide there are very few cases of eccrine variant; still

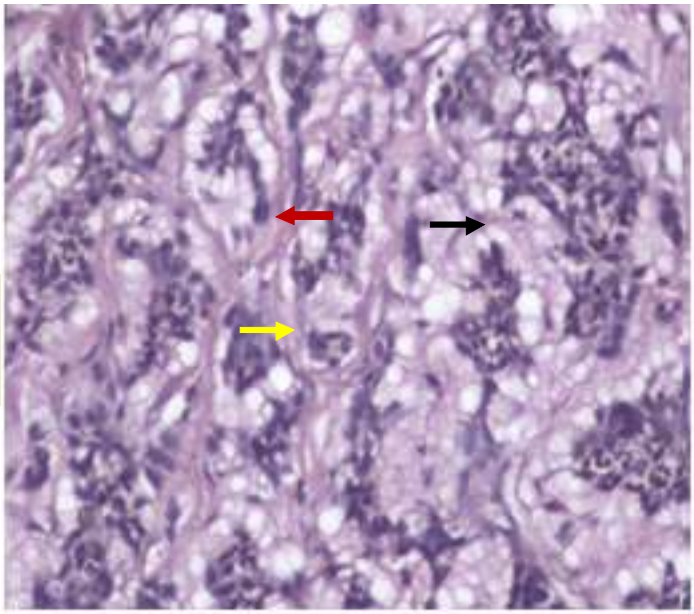


Fig9: Epithelial cells (black) and myoepithelial clear cells (red), against chondrmyxoid matrix (yellow) (H&E, 100x)

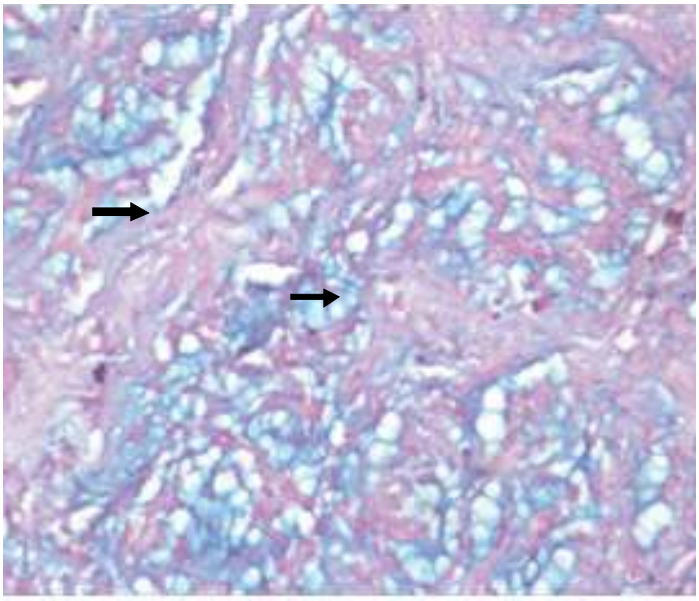
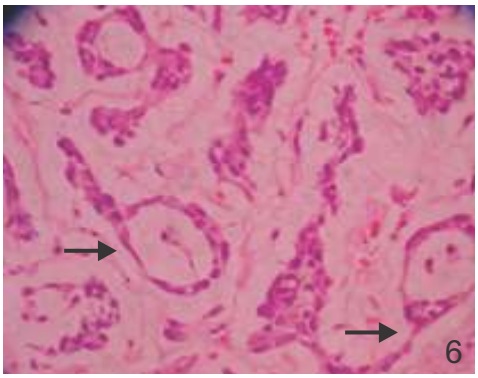
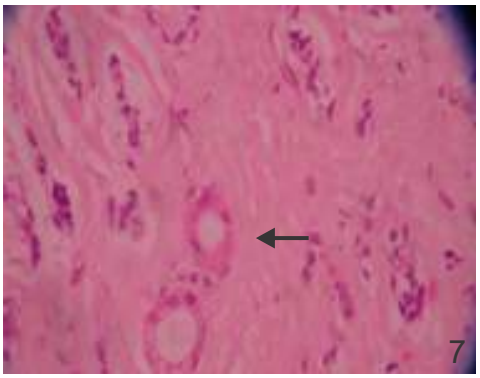
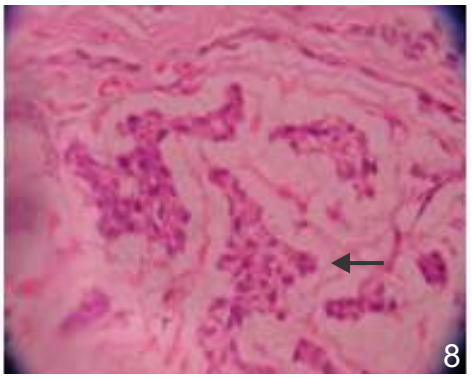


Fig10: Highlighting chondromyxoid matrix (ALCIAN PAS;100X)

further, eccrine variant presenting with multiple lesions are sparse.[5,6,7]

When an elderly male patient presents with multiple asymptomatic lesions over scalp, neck or forehead, chondroid

Figs 6-8: Tadpole like acrosyringium- [eccrine variant (red)] and trabecular pattern (blue) (H&E;100x)

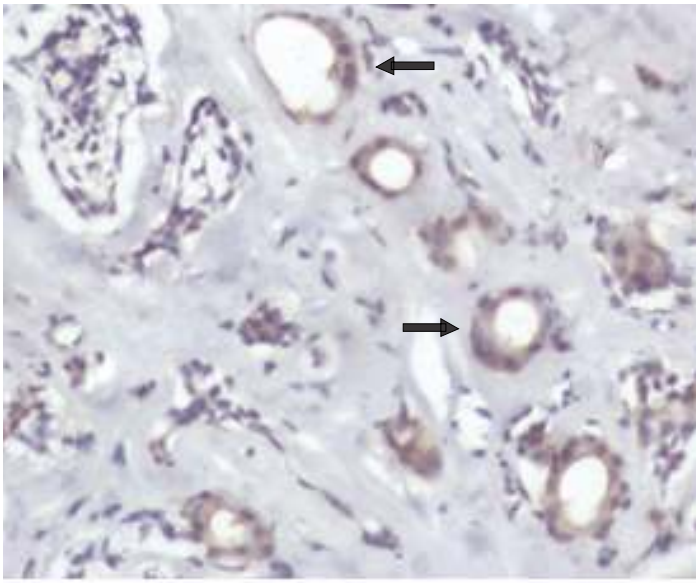


Fig 11: Highlighting the epithelial component (IHC with CYTOKERATIN;100X)

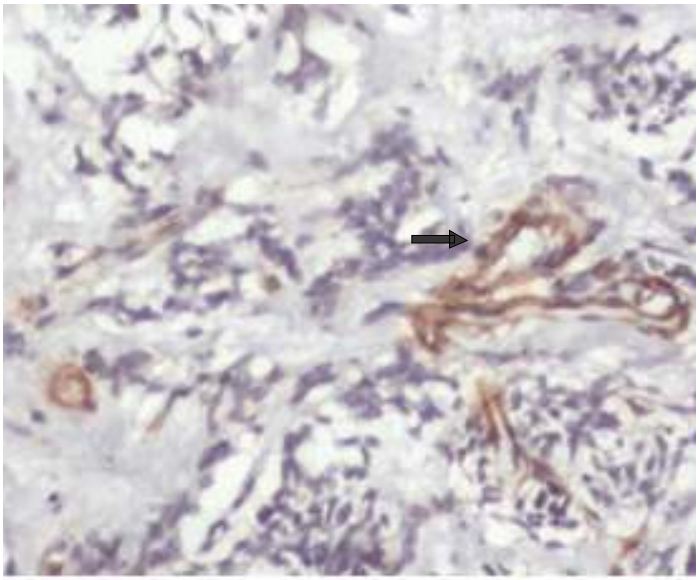


Fig 12: Highlighting the myoepithelial component (IHC with SMA;100X)

syringoma has to be considered as one of the diagnosis. Histopathology helps in confirmation and treatment of the disease.

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CASE REPORTS
Year: 2018 I Volume: 1 I Issue: 3 I Page: 94-95

A RARE CASE OF DOWLING-DEGOS DISEASE AND RETICULATE ACROPIGMENTATION OF KITAMURA

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Abstract

Dowling-Degos disease and Reticulate acropigmentation of Kitamura are autosomal dominant form of pigmentary disorders. Dowling-Degos disease is characterized by reticulate pigmentation usually in flexural areas with pitted perioral acneform scars. Reticulate acropigmentation of Kitamura shows reticulate hyperpigmentation on dorsa of hands and pits on the palms and soles. Here we are reporting a case which has the findings of both the diseases .

Keywords: : Reticulate pigmentation, Dowling Degos, Kitamura

Introduction

Dowling-Degos disease (DDD)is an autosomal dominant form of reticulate pigmentary genodermatosis with variable penetrance. The reticulate pigmentation usually has a flexural distribution. Comedo like lesions in neck and pitted perioral acneform scars has also been described. The disorder usually appears and/or worsens after puberty. DDD, dyschromatosis symmetrica hereditaria (DSH), dyschromatosis universalis hereditaria (DUH) and reticulate acropigmentation of Kitamura (RAPK) share clinical features with each other; yet, they have different pathology findings.[1,2] Histopathology is a diagnostic testing with a distinctive form of acanthosis, characterized by downward elongations of thin rete ridges with reticulated or fenestrated patterns, with a concentration of melanin at the tips and occasional follicular plugging and horn cysts.[3]

Reticulate acropigmentation of Kitamura(RAPK) is a rare pigmentary disorder that has an autosomal dominant pattern of inheritance. Typical features include reticulate hyperpigmentation , atrophic macules on dorsa of hands and pits on the palms and soles in the first or second decade of life.

Case Report

A 22-year-old female presented with tiny brown flat and few depressed lesions over her face, neck, axillae, dorsa of hands and feet and bilateral ulnar aspects of forearm since the age of 10 years. She even complained of tiny scars around the mouth since the age of ten years. There was no history of consanguinity. There was no history of similar illness in family members. On cutaneous examination, hyperpigmented macules with atrophy in some lesions over face, neck, upperchest, upper back, bilateral axillae (Figure no.1), flexures of bilateral forearm, dorsa of bilateral hands and feet were present (Figure no.2). Comedo like lesions and perioral acneform scars were present over face (Figure no.3). There were pits over bilateral palms (Figure no.4). Hair and nails were normal.

Histopathological examination showed circumscribed foci of epidermal proliferation and hyper pigmentation. The epidermis showed subtle proliferation in the form of delicate elongated and confluent rete ridges that showed antler like branching at places. The pigment was seen to be concentrated at the bottoms of rete ridges and also in the melanophages in papillary dermis (Figure no.5). Based on the clinical findings and histopathological

Figure 1: Reticulate pigmentation over axilla.

Figure 2: Reticulate pigmentation of dorsum of hands.

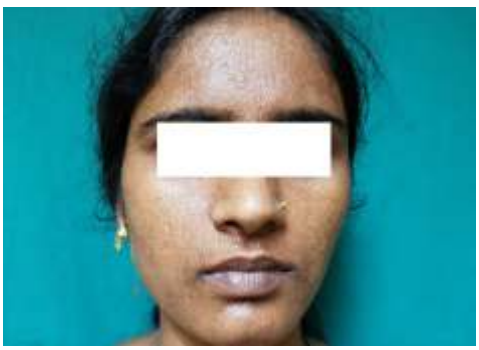

Figure 3: Perioral and facial acneform scars.

Figure 4 : Pits over the palm.

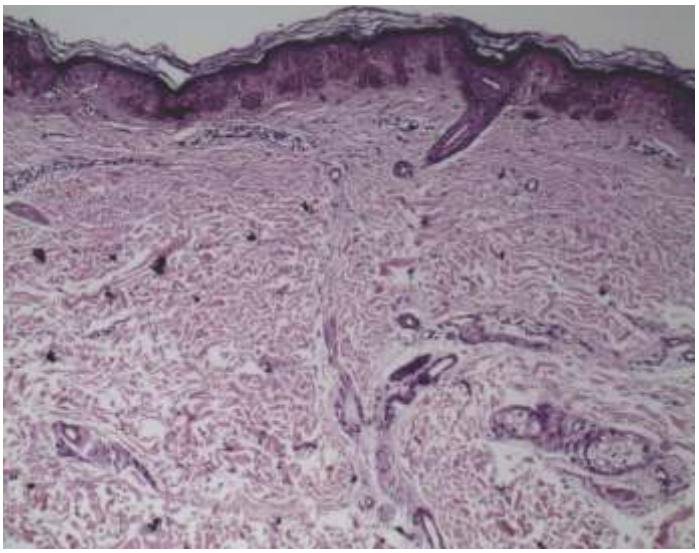

Figure 5 : epidermis with subtle proliferation in the form of delicate elongated and confluent rete ridges that showed antler like branching at places. The pigment was seen to be concentrated at the bottoms of rete ridges and also in the melanophages in papillary dermis (H&E;100X)

findings diagnosis of Dowling-Degos disease with over lapping features of reticulate acropigmentation of Kitamura was made.

Discussion

DDD and RAPK are autosomal dominant reticulated pigmentary disorders. In the past, there has been a few case reports of patients exhibiting features of both DDD and RAPK, indicating that they may be the same disease with variable phenotypic expression.[4] DDD was first described by Dowling in 1938 and Degos in 1954. It is mainly characterized by reticulated hyperpigmented macular lesions predominantly distributed over the flexures (neck, axilla, cubital fossa, groin).[5] It may also show open comedolike lesions of the face and neck and pitted perioral acneiform scars. The genetic defect in DDD is due to the loss of function mutations in the keratin 5 gene (KRT5) situated in the keratin gene cluster on chromosome 12q13, resulting in haploinsufficiency.[6] Another genetic defect of DDD has been reported in the gene locus mapping to chromosome 17p13.3 and chromosome 1q21 with mutations located in the DSRAD gene.[7] Galli- Galli disease is a rare acantholytic variant of DDD.[8]

RAPK has mostly been reported in Asian ethnic groups. It usually develops during the first and second decades of life and is characterized by reticulate hyperpigmented macules over the dorsa of hands and feet with few palmoplantar pits. It has been postulated that sunlight may play a role in aggravating this condition and therefore lesions gradually darken over time.[9]

Our patient had diffuse reticulate pigmentation of face, flexures with dark comedo-like lesions and perioral pitted acneiform scars resembling DDD and reticulate acral pigmentation over the dorsa of both hands and feet with few palmar pits resembling RAPK. Treatments options include topical adapelene, 20% azelaic acid, systemic retinoids and Erbium doped yttrium aluminum garnet(Er:YAG).[10] None of the treatment options available are effective. Our patient had an overlap of features of DDD and RAPK, which has been rarely mentioned in the literature.

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CASE REPORTS
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A CASE REPORT WITH REVIEW OF LITERATURE ON PYODERMA FACIALE IN PREGNANCY – A THERAPEUTIC DILEMMA

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Abstract

Pyoderma faciale is a rare facial dermatosis. It is characterized by a fulminating course of facial inflammation consisting of numerous pustules, cystic swellings and coalescing sinuses in young women. It has to be differentiated from acne and rosacea. The aetiopathogenesis of pyoderma faciale is not yet identified. It has been associated with pregnancy in a few cases. We report a case of a primigravida who presented with sudden onset of pustules and cystic swellings over the face with no prior similar history which was diagnosed as pyoderma faciale. In view of her pregnancy, systemic retinoids which is the treatment of choice was contraindicated and so was treated with tapering doses of oral steroids in combination with topical therapy. There was complete resolution of symptoms with treatment and a good obstetric outcome.

Key words: pyoderma faciale, pregnancy

Introduction

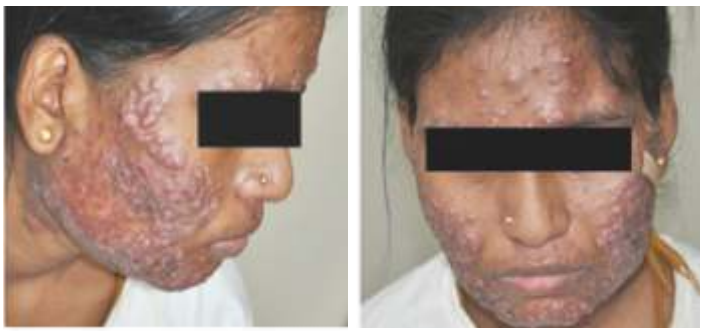
Pyoderma faciale was formerly described by O'Leary and Kierland in 1940.[1] It is a rare facial dermatosis characterized by the sudden onset of severe facial inflammation consisting of numerous pustules, cystic swellings and coalescing sinuses. Plewig et al considered it as an extreme form of rosacea and termed it as rosacea fulminans.[2] But it is not yet clear whether this condition is a variant of rosacea or acne vulgaris or a separate entity. It is not a pyoderma; nor a variant of acne conglobata.[3,4] We report a case of pyoderma faciale in pregnancy due to its rarity and for its therapeutic dilemma.

Case Report

Twenty four years old primigravida, presented at eight weeks of gestation with abrupt onset of pustules, nodules and cystic swellings over the face of three weeks duration. There was no significant past medical history and no history of similar lesions in the past. The lesions were tender and disturbed her sleep. Two weeks prior to presentation to our hospital, the patient was diagnosed to have acne vulgaris and treated elsewhere. As there was no improvement she was referred to us. There is also history of intake of vitamin B complex supplements after the onset of lesions, which aggravated the lesions. She also had hyperemesis gravidarum for which she was admitted to give supportive care.

General and systemic examination was normal. Dermatological examination revealed multiple tender nodules, abscesses, pustules and cystic swellings over the face sparing the central part and temporal region. There were no lesions elsewhere in the body (Figure 1,2).

All investigations were normal. Biopsy was consistent with pyoderma faciale, which showed follicular plugging, dense perivascular and periadnexal infiltrate. Dermis showed granulomatous reaction pattern with infiltrate including neutrophils, eosinophils, lymphocytes, epithelioid histiocytes, plasma cells and multinucleate giant cells. (Figures 5, 6 and 7)



Figs 1,2: Figure 1,2: multiple tender nodules, abscesses, pustules and cystic swellings over the face sparing the central part and temporal region

As there was minimal crusting of the lesions she was given a course of cloxacillin for 1 week. Then she was initiated on topical steroid along with low dose of systemic steroid (Tab Prednisolone 20 mg) and topical clindamycin. Oral prednisolone was maintained at the dose of 20 mg for 4 weeks then it was tapered by 2.5 mg every week. As all of her lesions subsided, oral prednisolone was tapered and stopped over 12 weeks (Fig. 8,9). There was no recurrence of lesions during the follow up for the next 5 months. She had a full term normal vaginal delivery.

Discussion

Pyoderma faciale is an uncommon disorder of unknown etiology that mainly affects post adolescent women, with abrupt onset and disfiguring sequelae if left untreated. There is sudden onset of severe facial inflammation consisting of numerous pustules, cystic swellings and coalescing sinuses. Edema and at times an

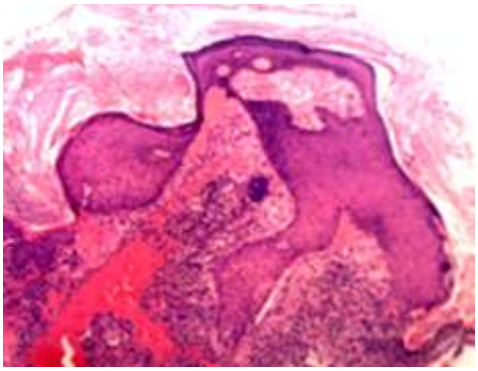


Figure 5: Pseudoepitheliomatous hyperplasia with dense dermal inflammation (H/E;20X)

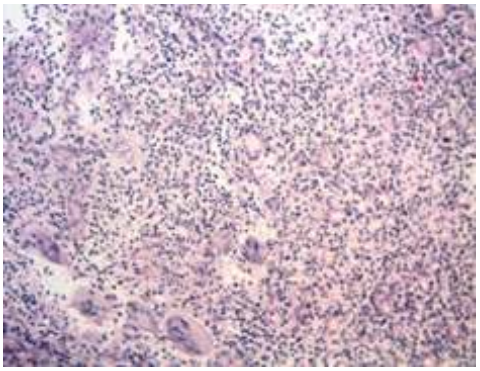


Figure 6: Dense dermal infiltrates of lymphocytes, neutrophils, plasma cells & few multinucleate giant cells (H/E;20X)

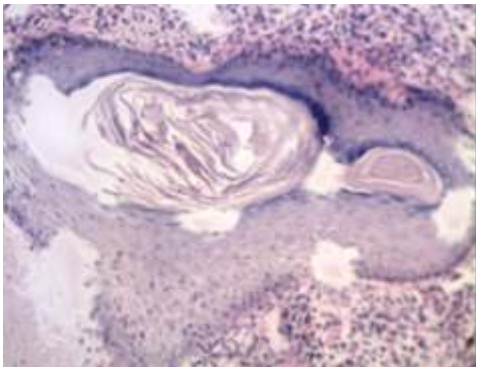
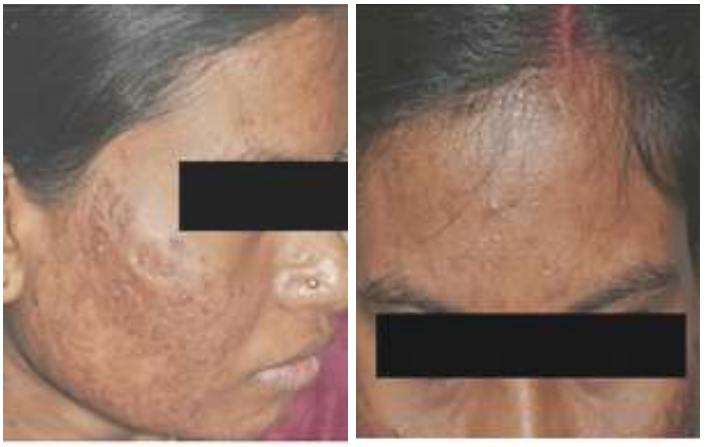


Figure 7: Periadnexal inflammation (H/E;20X)



Figs 8,9: Post treatment

intense reddish or cyanotic erythema accompanies this pustular process. [2,4]

The aetiopathogenesis of pyoderma faciale is unidentified. It is associated with multiple conditions (table 1).[5-12]

No.	Condition
1	Pregnancy[5]
2	Erythema nodosum[6]
3	Crohn's disease[7]
4	Ulcerative colitis[8]
5	interferon 2B and ribavirin therapy for hepatitis C[9]
6	High-dose vitamin B supplements[10]
7	Emotional distress[11]
8	Thyroid disorders[12]

Table 1: Associations of pyoderma faciale

Hormonal imbalance has been proposed in view of its almost exclusive occurrence in women; furthermore, the eruption has been associated with pregnancy in a few cases.[4,5,12] Usually no recurrence of pyoderma faciale is seen except few case reports.[3,12] This condition has to be differentiated from acne vulgaris and rosacea. As compared to acne vulgaris it is abrupt in onset with devastating sequelae of scarring if not treated early. But it does not last more than a year. It is confined to the face and does not arise from comedones. Except for two case reports in males, it occurs exclusively in females.[13, 14] It should also be differentiated from rosacea. There is no consistent history of flushing in cases of pyoderma faciale. Furthermore the features such as pre-existing erythema or telangiectasia of the convex portions of the face are absent. The lesions in pyoderma faciale are characteristically large abscesses and nodules. It is not associated with sun exposure, Helicobacter pylori and Demodex. In pyoderma faciale induration and rhinophyma never develop as a sequelae.[3,4,15]

The diagnosis of pyoderma faciale is often exclusively made based on clinical findings, but can be aided by biopsy.

The differential diagnoses of pyoderma faciale include gram negative folliculitis,acne conglobata, acne fulminans, fungal and mycobacterial infections, iododerma, bromoderma, and neutrophilic dermatosis like sweet's syndrome only affecting the face.[16]

It has been recommended that treatment should begin with potent topical corticosteroids for no more than two weeks, oral prednisolone at 1 mg/kg daily for 1–2 weeks followed by a gradual tapering along with oral isotretinoin at 0.2–0.5 mg/kg daily for three to four months until complete healing occurs. Oral tetracycline antibiotics and dapsone have been found to be effective as in many case reports.[17, 18] But during pregnancy retinoids and tetracylines are contraindicated. Pyoderma faciale is the only indication for topical or systemic corticosteroids in the treatment of rosacea.[18]

To our knowledge, this is the twentieth reported case of pyoderma faciale associated with pregnancy in literature, thus contributing further evidence that pregnancy can aggravate this condition and this is the first case report of the same from India. Previous reported cases of pyoderma faciale (rosacea fulminans) associated with pregnancy and their comparisons with our case are given in the table 2.[4,5,12,19-25]

Most of the cases of pyoderma faciale presented in first trimester. Out of the eighteen cases of pyoderma faciale in pregnancy, where treatment details were available eight cases were treated with oral steroids and topical antibiotics. All of the patients had an improvement with treatment except two cases which includes one case who had persistence of lesions throughout the pregnancy and other case had recurrence of symptoms on tapering of steroids.[21, 23] Out of the eleven cases where obstetric outcome details were available, two had intrauterine death, one

Table 2: Case reports of pyoderma faciale (rosacea fulminans) in pregnancy

Serial no.	Authors	No. of cases	Development of pyoderma faciale(PF)	Treatment given	Outcome of treatment	Outcome of pregnancy
1	Massa MC and Su WP [12]	5	Third trimester / postpartum	Not specified	Improved	Not specified
2	Marks VJ and Briggaman RA [19]	1	Second trimester	Topical antibiotics, intralesional triamcinolone acetonide and prednisolone	Improved	Full term healthy baby
3	Plewig G, Jansen T, and Kligman AM [4]	4	Two cases in first trimester; one case in third trimester,one case developed in postpartum	Topical antibiotics including clindamycin and erythromycin	Improved	Not specified
4	Haugstvedt A and Bjerke JR [20]	1	Not specified	Not specified	Not specified	Not specified
5	Lewis et al[21]	1	First trimester	Prednisolone and erythromycin	Poor response Lesions and recurrence of lesions on tapering prednisolone	Intrauterine death
6	Fehrabas et al[22]	1	First trimester	Oral steroids, surgical drainage, topical antibiotics	Moderate response	Full term delivery
7	Cisse et al[23]	1	First trimester	Topical and oral macrolides, topical metronidazole, amoxicillin, oxacillin and fusidic acid	Skin disease persisted throughout pregnancy	Not specified
8	Jarrett R, Gonsalves R and Anstey AV [5]	3	All three patients first trimester	Prednisolone and azithromycin	First patient had moderate improvement with persistence of symptoms till 2 months postpartum. Second and third patients had complete resolution of lesions.	First case intrauterine death, Second case termination of pregnancy, and third case had a normal vaginal Delivery
9	Fuentelsaz et al[24]	1	First trimester	Oral azithromycin, topical antibiotics and steroids	Resolution by sixth month of pregnancy	Full term normal delivery
10	Fernanda et al[25]	1	Second trimester	Oral erythromycin and prednisolone	Improved	Full term caesarean delivery
11	Current case	1	First trimester	Topical and systemic steroids, Topical and systemic antibiotics,benzoyl peroxide face wash, incision and drainage	Complete resolution of the lesions after 12 weeks of therapy	Full term normal delivery

had medical termination of pregnancy while others had full term delivery. Cisse et al reported a case of pyoderma faciale during pregnancy which was initially thought to be due to administration of follicle stimulating hormone (FSH) and luteinizing hormone releasing inhibitor, but later this was thought to be unlikely since lesions persisted throughout pregnancy even after withdrawal of the drugs.[23] Fernanda et al reported a case of pyoderma faciale in second trimester associated with relentless ocular involvement which ended up with ocular perforation. She was treated with oral erythromycin,

predinisolone and successful corneal transplant. She had a full term normal delivery.[25]

It is imperative to recognize this entity and its importance in relation to pregnancy. It should be diagnosed and treated without delay as the sequelae of scarring can have a negative psychosocial impact on the patient. Our patient received immediate attention and was started on specific therapy, with complete resolution of lesions without much scarring. So it highlights the significance of early diagnosis of this condition and initiation of steroids in the early phase of the disease to bring remission.

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