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G1, Medical Academics of Malaysia
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GENERAL DERMATOLOGY - Original Article

TOPICAL CORTICOSTEROID PHOBIA AMONG ATOPIC ECZEMA PATIENTS AND THEIR CAREGIVERS: SURVEY IN TWO DERMATOLOGY OUTPATIENT CLINICS IN MALAYSIA

Noorlaily MN[1], Baba R[2]

Abstract

Introduction: Steroid phobia has been recognized as a hindrance to successful treatment in patients with atopic eczema. Data on this phenomenon is lacking in Malaysia. This study investigates the prevalence and reasons for topical corticosteroid phobia in patients with atopic eczema and their caregivers.

Methods: A questionnaire-based study was conducted in both children and adult patients suffering from atopic eczema in two government dermatology clinics at Hospital Kuala Lumpur and Hospital Kota Bharu between May and July 2011.

Results: Of the 77 respondents, 39% were worried about using topical corticosteroids for their eczema with 13% totally avoiding steroids. The most common reason for this fear was concern about skin thinning (27.3%) followed by concern on changes in skin colour (14.3%), scarring (13%) and stretch marks (13%). Steroid phobia was significantly higher in those with tertiary education (p=0.005).

Conclusion: Steroid phobia appears to be a significant problem in Malaysia especially in those with higher educational background. Despite these concerns, the patients' compliance to topical corticosteroids is still reasonably good.

Keywords: *Steroid, phobia, eczema*

Introduction

Topical corticosteroid is one of the most commonly used treatment modalities in atopic eczema patients. It was first introduced in 1952 and remained the mainstay of treatment for atopic eczema because of its potent anti-inflammatory and antiproliferative effects.[1]

Corresponding Author and Reprint Request
Dr Noorlaily Mohd Noor
Department of Dermatology, Hospital Kuala Lumpur,
Jalan Pahang 50586 Kuala Lumpur
Email: laily124@hotmail.com

[1] Department of Dermatology, Hospital Kuala Lumpur
[2] Department of Dermatology, Hospital Melaka

Recently a phenomenon known as steroid phobia has been recognized as a hindrance to successful treatment in patients with atopic eczema in a few developed countries. Steroid phobia is irrational fear and anxiety about using corticosteroids.[2,3] Some centres reported prevalence of steroid phobia to be as high as 80.7%.[4] However data on steroid phobia is lacking from the developing countries. Thus we embarked on this study to investigate the prevalence and the reasons for topical corticosteroid phobia in patients with atopic eczema and their caregivers. We also explored whether the level of education and family income have any effect on this phenomenon.

Materials and methods

TA questionnaire-based study was conducted in both children and adult patients suffering from atopic eczema in two government dermatology clinics at Hospital Kuala Lumpur and Hospital Kota Bharu between May and July 2011. This questionnaire was prepared with reference to an almost similar study done in Queen's Medical Centre, Nottingham.[2] The diagnosis of atopic eczema was made by dermatologists and senior medical officers in all cases. The questionnaire was given out to successive patients, then completed by patients in those over 18 years of age or a parent or guardian in those less than 18 years of age.

The first section of the questionnaire was intended to capture the demographics of the patients. In the second section, the level of education and family income were determined. The level of education was divided into primary (up till primary six or 12 years old), secondary (up till Form 5 or 17 years old) and tertiary (educational level following secondary education). The family income was divided into different categories based on the monthly income as follows: low (<RM 1500), lower middle (RM 1501-5000), upper middle (RM 5001-10000) and high income group (>RM10000).

The next section looked into steroid phobia and the cause of the phobia with the following questions:

Do you apply the steroid creams as advised by your doctor?
Are you worried about applying steroids on your skin/your child's skin?
If the answer is yes, why do you worry?
Have the worries stopped you from applying the steroid creams (on your child)?

The effect of the educational background and family income on steroid phobia was analysed using the chi-square test.

Results

There were 77 respondents, 59 from Hospital Kuala Lumpur and 18 from Hospital Kota Bharu. Table 1 shows the demographics of the patients.

Table 1. Patients' demographics

		Number of patients (%), n=77
Age	< 18	51 (66.2%)
	≥ 18	26 (33.8%)
Gender	Male	22 (28.6%)
	Female	55 (71.4%)
Race	Malay	61 (79.2%)
	Chinese	7 (9.1%)
	Indian	7 (9.1%)
	Others	2 (2.6%)
Income	< RM 1500	26 (33.8%)
	RM 1501 - 5000	40 (51.9%)
	RM 5001 - 10000	7 (9.1%)
	> RM 10000	4 (5.2%)

Table 2. The reasons for patients'/caregivers' concerns about using topical corticosteroids.

Cause of topical corticosteroid phobia	Number of patients (%), n=77
Skin thinning	21 (27.3%)
Change in skin colour	11 (14.3%)
Scarring	10 (13%)
Stretch marks	10 (13%)
May worsen eczema	9 (11.7%)
May become dependent	8 (10%)
Increased risk of infection	7 (9%)
Cancer	5 (6.5%)
Cataract	5 (6.5%)
Growth delay	5 (6.5%)
Skin ageing	5 (6.5%)
Increased body hair	5 (6.5%)
Sunburn	4 (5.2%)
Bruising	3 (3.9%)

Table 3. The effect of educational level on steroid phobia.

	Yes	No	P value
Level of education			
Primary and secondary	12/46 (26.1%)	34/46 (73.9%)	0.005
Tertiary	18/31 (58.1%)	13/31 (41.9%)	> 0.05

Out of these, 39% (30/77) were worried about using topical corticosteroids for their eczema. The reasons for these worries are shown in Table 2. A total of 13% (10/77) avoided using topical corticosteroids because of these worries. The most common reason for this fear was their concern about skin thinning (27.3%). They were also worried about topical corticosteroids causing changes in skin colour (14.3%), scarring (13%) and stretch marks (13%). The number of patients with steroid phobia was significantly higher in those with tertiary education, p=0.005 (Table 3). Family income has no effect on steroid phobia.

Discussion

Steroid phobia has become an increasingly difficult problem especially in the developed countries such as United Kingdom, Japan and Australia.[2,4-6] Our study showed that 39% of patients with atopic eczema has steroid phobia. This figure is relatively small compared with data from developed western countries such as United Kingdom and France where 72.5% and 80.7% of atopic eczema patients have steroid phobia respectively.[2,4] On the other hand a study in Japan indicated that the incidence of steroid phobia was 38.3% although a slightly different methodology was used.[7] More studies should be done in Asian countries to see whether fear for steroid is less common among Asian patients compared to the west. If this is so, it would be interesting to see whether cultural differences and belief play any role in this observation.

Only a third of our patients with steroid phobia avoided steroids which was similar to the study done in the UK.[2] Thus the majority of patients were still compliant to their treatment despite this fear. However without proper counselling the figure may rise and potentially cause treatment failure as a result of noncompliance. Thus it is important for doctors and other healthcare workers to spend more time with patients and their caregivers to ensure dissemination of correct information. One of the reasons suggested was the inappropriate use of the words 'sparingly' or 'thinly' to describe how corticosteroid creams should be applied on the skin. This misled patients to be extremely cautious and anxious of the adverse side effects during application. The more suitable advice would be 'apply enough to cover affected areas'.[8]

Skin thinning is the most common reason for the fear of topical corticosteroids in this and other studies.[2,6] One of the reasons is that this adverse effect is probably emphasized the most by doctors or perceived as the worst by patients. Change in skin colour is the next most worrisome side effects in patients with steroid phobia. As the majority of patients were Asians with generally darker skin, hypopigmentation especially is not an acceptable side effect. The other thing to look into is how the information on adverse effects is phrased in the steroid information leaflet of the creams.

Steroid phobia was significantly higher in those with tertiary education. However family income has no effect on steroid phobia. Patients and caregivers are more well informed nowadays, and this is especially so with the availability of vast material on the media and internet. Those with tertiary education would probably search for more information and do not rely solely on advice from the doctors. Future studies should look into the source of information in these patients and their caregivers.

Among the limitations of this study was the small number of patients and their selection from subsidized government hospitals only. Further studies should include more centres and include patients in private healthcare settings as well as those from rural areas.

Conclusion

Steroid phobia appears to be a significant problem in Malaysia although its prevalence is not as high as that of other developed countries. Steroid phobia is also more common in those with tertiary or higher educational background. Despite these concerns, the patients' compliance to topical corticosteroids is still reasonably good.

Acknowledgement

The authors would like to thank Dr Zulrusydi Ismail and Dr Felix Yap for their contribution.

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LEARNING POINTS FROM THIS STUDY

1. Steroid phobia is not uncommon in Malaysia. However, the phenomenon is not as common as other developed countries. This might be due to the belief, upbringing and treatment seeking behavior of Malaysian patients. Most patients seek treatment from doctors without much hassle and do not query about the medications given.

2. Despite the concern about steroid, only a third of patients totally abstain from using steroid. It might indicate that they have heard about the side effects of steroid that might raise some concern. Most still use steroid as they has trust in their doctors.

3. Topical steroid if used properly will benefit patients with atopic eczema. Doctors should always educate patients with eczema about steroid and how to use them properly. They should allay patients' fear about steroid side effects and offer alternative treatment if necessary.

4. Information about steroids on the media especially social media will cause widespread phobia and thus compromise the care of patients with eczema. Thus, it is important for doctors especially dermatologists to properly educate patients regarding proper use and side effects of topical steroids. Paramedical personnel and medical students should also be educated about proper judicious use of topical steroids.

Yap FBB
Editor-in-Chief, Malaysian Journal of Dermatology

GENERAL DERMATOLOGY - Original Article

THE EFFECT OF ORAL CLINDAMYCIN AND RIFAMPICIN COMBINATION THERAPY IN PATIENTS WITH HIDRADENITIS SUPPURATIVA IN SINGAPORE

Ochi H, Tan L X, Oon H

Abstract

Introduction: Staphylococcus spp. Are frequently isolated from lesions of hidradenitis suppurativa (HS) although it is not an infectious disease. Here, we review 11 patients with HS treated with combination of oral clindamycin and rifampicin.

Methods: Retrospective review assessing the efficacy of a 10 week course of oral clindamycin 300 mg twice daily and oral rifampicin 300 mg twice daily in the treatment of HS

Results: Seven patients (63.6%) reported clinical improvement. Three patients (27.3%) achieved clear, minimal or mild scoring from all sites after completion of therapy and 2 patients (18.2%) reported a 2-grade improvement relative to baseline from at least one site. One patient (9.1%) who reported side effects of nausea and vomiting and 1 patient (9.1%) who defaulted follow-up.

Conclusion: Combination of oral clindamycin and rifampicin is safe and efficacious in the treatment of HS.

Keywords: *Hidradenitis suppurativa, clindamycin, rifampicin*

Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory disease of follicular occlusion characterized by abscesses, draining sinuses and scarring. It mainly affects apocrine gland-bearing areas, including the axilla, groin and anogenital regions. Although HS is not primarily an infectious disease, Staphylococcus aureus and Staphylococcus epidermidis are pathogens most frequently isolated as secondary colonizers.[1]

Corresponding Author and Reprint Request
Dr Ochi Harumi
National Skin Centre, 1 Mandalay Road
Singapore 308205
Email: ochi.harumi@mohh.com.sg

Methodology

This retrospective review assessed the efficacy of a 10 week course of oral clindamycin 300 mg twice daily and oral rifampicin 300 mg twice daily in the treatment of HS. Patients who received this combination therapy between 1 December 2012 and 31 July 2013 in a tertiary dermatological center in Singapore were included.

Results

Eleven patients (9 males) had a mean age of 24.5± 8.8. There were 6 Chinese (54.5%), 4 Malays (36.3%) and 1 Indian (9.1%). Five were smokers (45.5%), 6 were obese (54.5%) and 1 had a family history of HS (9.1%). The duration of HS prior to commencement of oral clindamycin and rifampicin ranged from 2 to 20 years. Eight patients (72.7%) had previous treatments, including retinoid and antibiotics, with limited effect and persistent disease.

At the end of 10 weeks of treatment, 7 of the 11 patients (63.6%) reported clinical improvement. Four patients had digital photography documenting response before and after treatment and 2 blinded assessors evaluated the improvement using the HS physician's global assessment (PGA) score. Three patients achieved clear, minimal or mild scoring from all sites after completion of therapy and 2 patients reported a 2-grade improvement relative to baseline from at least one site. There was only 1 patient (9.1%) who reported side effects of nausea and vomiting and 1 patient (9.1%) who defaulted follow-up. (Table 1)

Discussion

The efficacy and tolerability of this combination treatment had previously been assessed in 4 studies. Overall results are promising with reported improvement rates between 71.4 % and 85.7%.[1, 2, 3, 4] Statistically significant improvements in all dimensions of the quality of life Skindex-France questionnaire was also described in 1 study.[2]

It is hypothesized that both the antibacterial and anti-inflammatory properties of clindamycin and rifampicin are responsible for the beneficial effects in treating HS. Clindamycin is a lincosamide

Table 1. Demographics of patients, previous treatments, response and side effects of combination therapy.

Case No.	Age (years)	Gender	Duration of disease (years)	Affected Area (s)	Prior Therapy	Physician clinical assessment	Pre-treatment Physician Global Assessment (PGA)	Post-treatment Physician Global Assessment (PGA)	Reported Side Effects
1	18	Male	2	Axilla, neck	Doxycycline, topical clindamycin	Improved	Nil	Nil	Nil
2	18	Male	4	Perineal	Doxycycline, erythromycin, isotretinoin, minocycline	Improved	Nil	Nil	Nil
3	19	Male	9	Perineal	Bactrim, cephalexin, doxycycline, erythromycin, isotretinoin, minocycline	Improved	2.75	1.50	Nil
4	20	Male	6	Perineal, axilla	Augmentin, topical clindamycin	Non-responder	Nil	Nil	Nil
5	21	Male	13	Perineal, axilla	Doxycycline, topical clindamycin	Improved	2.67	1.00	Nil
6	21	Male	3	Perineal, axilla, Neck	Nil	Improved	1.83	1.75	Nil
7	21	Male	3	Perineal, back	Defaulted	Defaulted	Nil	Nil	Nil
8	22	Male	5	Perineal	Isotretinoin, minocycline, topical clindamycin	Improved	Nil	Nil	Nil
9	48	Male	20	Perineal, axilla	Augmentin, acitretin. ciprofloxacin, clindamycin, ceftriaxone, isotretinoin, infliximab	Non-responder	3.13	3.00	Nil
10	27	Female	7	Perineal, axilla	Doxycycline, isotretinoin	Non-responder	Nil	Nil	Nausea, vomiting
11	35	Female	2	Perineal, axilla	Nil	Improved	Nil	Nil	Nil

antibiotic that is active against Gram-positive cocci and anaerobic bacteria. It mediates inflammation by suppressing complement-derived chemotaxis of polymorphonuclear leukocytes. Rifampicin is a lipid soluble, broad-spectrum antibiotic highly effective against Staphylococcus aureus. Additionally, it modifies cell-mediated hypersensitivity by suppressing antigen-induced transformation of sensitized lymphocytes.

Rapid emergence of bacterial resistance may result with rifampicin monotherapy.[5] Hence, combination therapy is synergistic with reduced resistance rates and increased anti- inflammatory properties. Although a longer duration of treatment appears warranted in chronic diseases like HS, no large differences in outcome between patients treated for 10 weeks or more and those treated for a shorter period have been reported.[4]

Other studies have similarly described good tolerability with low rates of side effects between 13.0% and 38.2%. Gastrointestinal complaints were most commonly reported but there were no cases of clindamycin associated Clostridium difficile colitis.[1, 2, 3, 4]

In a recent systematic review of HS treatment, only combination clindamycin-rifampicin regimen, infliximab, Nd:YAG laser and surgical excision were considered effective treatments. However, some of these modalities have limitations. Infliximab has reported adverse events including severe allergic reactions, multifocal motor neuropathy and drug-induced lupus reactions. Recurrence rates of up to 42.8% after surgical excision have also been described.[6]

Conclusion

In conclusion, oral clindamycin and oral rifampicin combination therapy is safe and efficacious in the treatment of HS in groups of Caucasian and Asian patients in Singapore.

Acknowledgments

Special thanks to Dr Heng Yee Kiat for assisting with the PGA scoring.

Conflicts of interest

There are none to declare. Dr Hazel Oon has received research grant from Pfizer, Novartis and acted as a speaker for Novartis, Galderma and Abbvie.

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LEARNING POINTS FROM THIS STUDY

1. Combination of oral rifampicin and clindamycin for 10 weeks seems promising in the treatment of HS. However, success of treatment is very dependent on the selection of patients.

2. Those who do not respond to this treatment cocktail seemed to have longer duration of disease. It is likely that they have been on multiple courses of antibiotics and anti-inflammatory agents which might cause treatment failure

3. It will be interesting to know how many courses of this cocktail can be given with good treatment outcome for patients with HS.

Yap FBB
Editor-in-Chief, Malaysian Journal of Dermatology

GENERAL DERMATOLOGY - Short Case

ADULT XANTHOGRANULOMATOSIS: A CASE REPORT

Ramalingam Rajalingam[1], Johar Asmah[1], Lee Bang Rom[2]

Introduction

Xanthogranulomatosis (XG) is the simultaneous occurrence of multiple xanthogranulomas, and is usually seen in the pediatric population. Adult XG (AXG) has many similarities to the childhood variant, including the association with hematologic disorders.

Herein, we report a case of xanthogranulomatosis in an adult without evidence of hematologic disease.

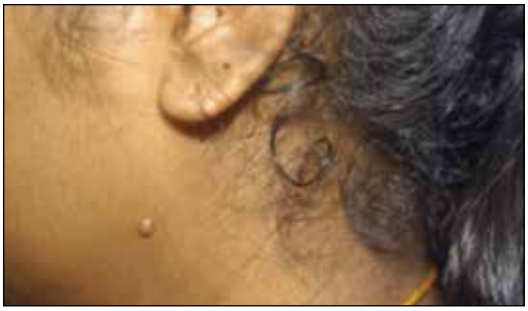

Figure 1a. yellowish papules on the left anterior neck and left earlobe.

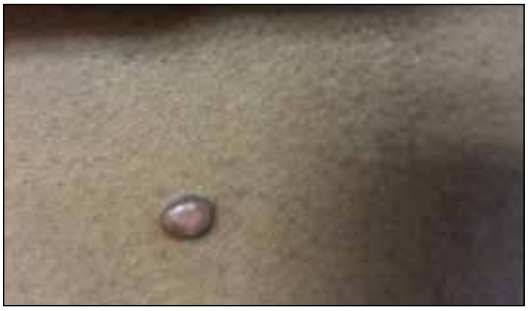

Figure 1b. brownish nodule on the anterior abdominal wall.

Corresponding Author and Reprint Request
Dr Ramalingam, Rajalingam
Department of Dermatology
Hospital Kuala Lumpur, Malaysia
Email: raj.blueheart@gmail.com

[1] Department of Dermatology, Hospital Kuala Lumpur
[2] Department of Pathology, Universiti Putra Malaysia

Case Report

A 27-year-old Indian housewife gave a 4-month history of spontaneous, multiple, discreet, yellowish, papular eruption over her left ear lobe, anterior neck, anterior abdominal wall, right forearm and left leg. The papules were not pruritic and not painful, but were increasing gradually in number and size. There was no history of trauma.

On examination, multiple discreet fleshy and yellowish papules and nodules were noted over the left earlobe, left anterior neck (Figure 1a), anterior abdominal wall (Figure 1b), right anterior upper arm (Figure 1c) and left medial calf (Figure 1d).

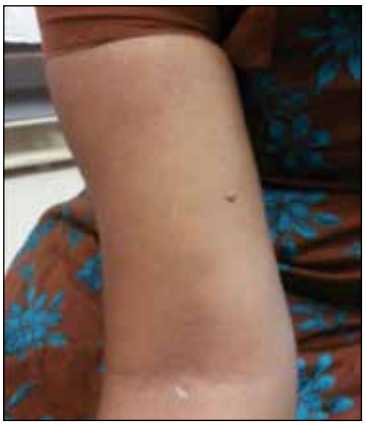

Figure 1c. yellowish papule on the right anterior upper arm.

Figure 1d. brownish nodule on the left medial calf.

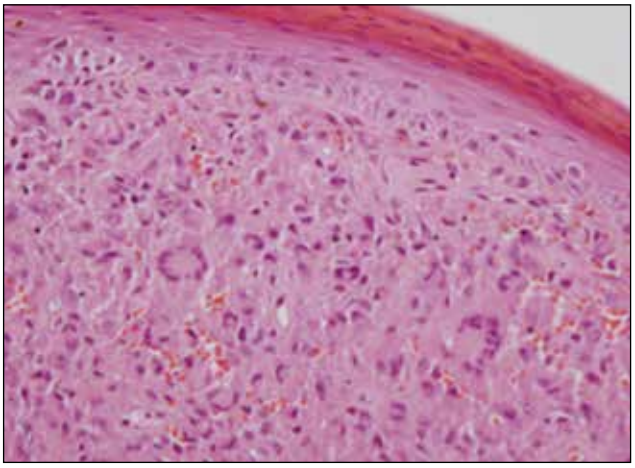

Figure 2a. Touton giant cells, hematoxylin & eosin stain, 100X magnification (from left earlobe nodule).

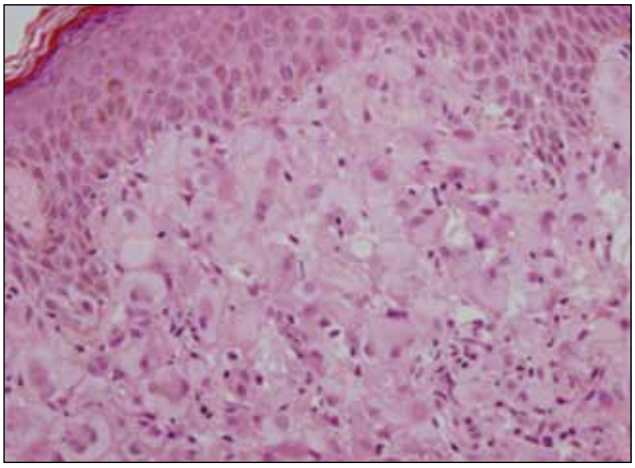

Figure 2b. Sea of histiocytes in the papillary and reticular dermis, hematoxylin & eosin stain, 400X magnification (from left anterior neck papule).

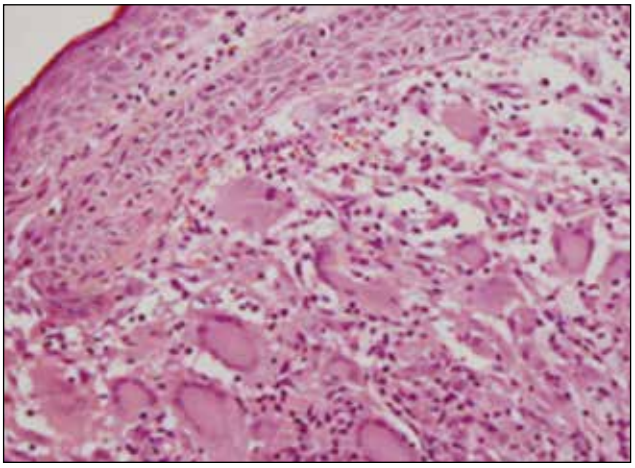

Figure 2c. Touton giant cells, hematoxylin & eosin stain, 400X magnification (from anterior abdominal wall nodule).

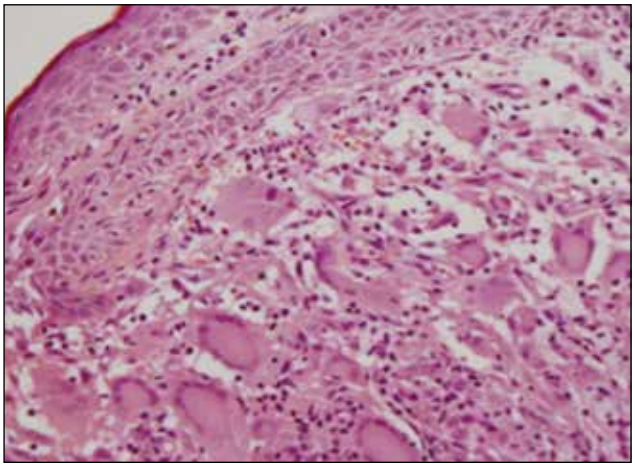

Figure 2d. Touton giant cells, hematoxylin & eosin stain, 400X magnification.

There was no lymphadenopathy or hepatosplenomegaly noted. Examination of all other organ systems were unremarkable.

We entertained several possible diagnoses at this point, including Eruptive Xanthomata and Hypertrophic Scars.

Blood investigations and imaging studies excluded the presence of malignancy. Glaucoma and retinal haemorrhage were also excluded. Excision biopsies showed dense histiocytic infiltrate admixed with Touton cells and foam cells of variable number, with lymphocytes, neutrophils and plasma cells seen infiltrating into the lesion. These findings are consistent with xanthogranuloma. Special immunohistochemical stains were not done.

Hence, we diagnosed her as having cutaneous xanthogranulomatosis. We initiated monthly intralesional triamcinolone injection for her papules. On subsequent follow-up, the lesions had shown only minimal flattening, but there were no new eruptions.

Discussion

Xanthogranulomatosis (XG) is the commonest non-Langerhans cell histiocytosis whose etiology is poorly understood.

Adult XG (AXG) accounts for 10% of all cases of XG, having a peak incidence ranging from 30 - 40 years. Initially pink to red papules, the lesions progress to yellow-brown firm, dome-shaped papules and nodules. They are most common on the

head, neck, and trunk, as seen in our patient. Ocular involvement is the most common extra-cutaneous manifestation which can lead to hemorrhage and glaucoma. Spontaneous resolution has been noted in several cases[1].

AXG is associated with hematologic disorders such as essential thrombocytosis, chronic lymphocytic leukemia, large B cell lymphoma and monoclonal gammopathy.[2-4] The development of XG lesions occurs before, during or following the hematologic disorder. Thus, it is important for us to follow up our patient long-term.

Histopathology typically shows a dense infiltrate of foam cells and Touton giant cells in the upper and mid reticular dermis, with variable extension into the subcutis. With immunohistochemical staining, histiocytes are positive to antibodies against factor XIIIa, HAM56, HHF35, KP1 (CD68), Ki-M1P and Vimentin, but negative to CD1a and S-100.[5]

Topical[6] and intralesional corticosteroid injection[7] have not been shown to have a significant effect. However, our patient shows some improvement with the latter treatment. Methotrexate on the other hand has shown some benefit, and might be considered should our patient not respond to current treatment.

Conclusion

Xanthogranulomatosis (XG) is the commonest non-Langerhans cell histiocytosis that can rarely occur in adults. The disease course remains uncertain with some cases demonstrating spontaneous resolution. In view of its association with hematologic disorders, we recommend screening adult patients with XG for those conditions.

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GENERAL DERMATOLOGY - Short Case

MYCOBACTERIUM CHELONAE INFECTION IN A 19-YEAR-OLD IMMUNOCOMPETENT PATIENT SUCCESSFULLY TREATED WITH ORAL CLARITHROMYCIN AND LINEZOLID

Ochi H, Pan JY

Introduction

Mycobacterium chelonae is a rapidly growing non-tuberculous mycobacteria (NTM) predominantly affecting immunocompromised hosts. We report a case of disseminated NTM cutaneous infection occurring in a healthy individual successfully treated with combination of oral clarithromycin and linezolid.

Case Report

A 19-year-old Chinese female with a past medical history of allergic rhinitis first presented with multiple abscesses on the sacrum, back and shoulder for 5 months. The abscesses developed when she was working in a microbiology lab preparing media and culturing bacteria. She denied any preceding trauma to the affected areas and was not on any immunosuppressive medications.

On examination, there were 2 discrete subcutaneous indurated nodular lesions over the mid-back. The largest lesion measured 5cm by 4cm with ulceration and underlying granulation tissue. There were multiple fleshy nodules over left shoulder, right infra-clavicular region and left iliac fossa. (Figure 1)

First set of fungal, bacterial and acid-fast bacillus cultures obtained from incision and drainage of back abscesses were negative. Skin biopsy showed granulomatous dermatitis (Figure 2). She was treated with multiple courses of oral amoxicillin-clavulanate, ciprofloxacin, clarithromycin, clindamycin, rifampicin and ofloxacin on separate occasions. Her disease was refractory to treatment and suppurative abscesses continued to develop on her trunk. Serology for human immunodeficiency virus and diabetic screening were negative. Subsequently, mycobacterial cultures from needle aspiration grew M. Chelonae sensitive to clarithromycin and linezolid. She was treated with combination oral clarithromycin 500mg and linezolid 600mg twice daily for 2 weeks with clinical improvement and healing. She will be reviewed in 3 months to check for any recurrence.

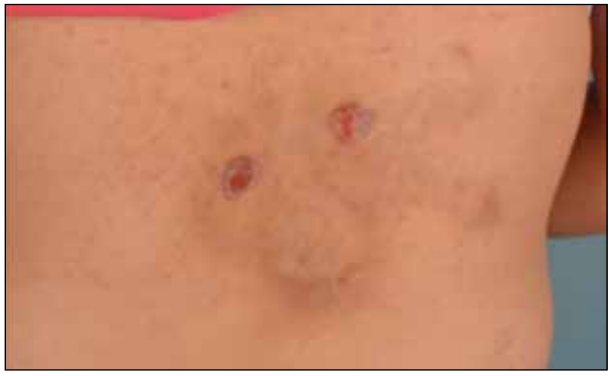

Figure 1. On examination, there were 2 x discrete subcutaneous indurated nodular lesions over mid-back.

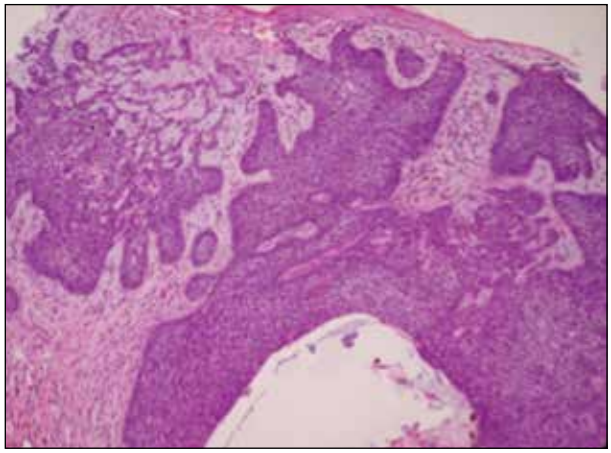

Figure 1. On examination, there were 2 x discrete subcutaneous indurated nodular lesions over mid-back.

Corresponding Author and Reprint Request
Dr Ochi Harumi
National Skin Centre, 1 Mandalay Road,
Singapore 308205
Email: ochiharumi89@gmail.com

Discussion

Non-tuberculous mycobacteria (NTM) are slender, non-motile, acid-fast bacilli classified into 4 categories based on growth rates and colony pigmentation.[1,2] Cutaneous presentations of NTM infection include localized non-healing ulcers and chronically draining subcutaneous nodules in immunocompetent patients. Disseminated disease is more frequently seen in immunocompromised hosts.[3] Mycobacterium chelonae and abscessus are responsible for 95% of disseminated cutaneous infections caused by rapidly growing mycobacteria.[4]

Clarithromycin 500mg daily monotherapy was previously recommended for treatment of M. chelonae. However, in vivo efficacy may lack correlation with in vitro sensitivity and clarithromycin resistance and therapeutic failure in patients with disseminated cutaneous infection have been reported.[5] Hence, more recent studies have suggested the use of oral linezolid combination therapy.[6]

Linezolid exhibits in vitro bacteriostatic activity against Mycobacterium tuberculosis and is increasingly used in the treatment of patients with multidrug-resistant (MDR) and extensively drug-resistant (XDR) strains. It blocks bacterial ribosomal protein synthesis by a novel mechanism: binding to the 50S bacterial ribosomal subunit and preventing formation of the initiation complex for protein synthesis. Linezolid has high bioavailability, low protein binding (31%) and exhibits no cross-resistance with other antituberculosis drugs.[7] Koh et al[8] reported that daily 300 mg dose of linezolid was useful in the treatment of patients with intractable multidrug-resistant (MDR) and extensively drug-resistant (XDR) tuberculosis with reduced neurotoxicity, compared with a daily 600 mg dose. In vitro synergy between linezolid and clarithromycin against Mycobacterium tuberculosis has been reported.[9]

To date the position of linezolid in the treatment of cutaneous nontuberculous mycobacterial infections is still unclear. Chetchotisakd et al10 has reported complete resolution of symptoms in 50% of patients with disseminated NTM infections on linezolid therapy. However, 31% developed adverse reactions to linezolid, 3 of whom received 600mg twice daily. Various adverse drug reactions have been described with long-term use of linezolid, primarily bone marrow suppression and peripheral and optic neuropathy. Hematologic adverse reactions ensuing from the prolonged use of linezolid are dose dependent and reversible secondary to inhibition of mitochondrial protein synthesis, while peripheral neuropathy might be irreversible depending on the prolonged duration of the therapy rather than dosage; however, optic neuropathy appears to resolve after stopping linezolid.[11,12] In view of promising clinical response and growing resistance to first line clarithromycin, the role of linezolid should be further evaluated.

Conclusion

In summary, we report a case of disseminated cutaneous Mycobacterium chelonae infection in an immunocompetent patient successfully treated with combination oral clarithromycin and linezolid. Data concerning the use of linezolid in nontuberculous mycobacterial infections is promising and should be further investigated.

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GENERAL DERMATOLOGY - Short Case

CONTACT DERMATITIS TO A TURMERIC COATED WEDDING THALI: A CASE REPORT

Wan Syamee Afira WAK[1], Tarita T[1], Rohna R[2], Zuraini A[3]

Introduction

Turmeric is a spice derived from the rhizome of the plant Curcuma longa, which is cultivated in India and Southeast Asia. Curcumin is the component of turmeric that gives the spice its unique taste and yellow colour and is thought to be responsible for turmeric's biologic activities[1]. Curcumin is used as a spice in foods, treatment in alternative medicine, dye for fur, and for traditional and religious purposes especially in the Indian culture. Rare cases of allergic contact dermatitis from curcumin have been reported among workers who dye animal furs, a worker at a pasta factory, in an Indian spice miller and from use of the Chinese herbal cream Chuu-ou-kou[2,3,4,5]. We report a case of allergic contact dermatitis to turmeric in an Indian lady who applied it on her string necklace for religious purposes (wedding thali) . We also describe the way turmeric is prepared as an allergen for patch test, as it is not readily available in the standard and extended series patch tests.

Case Report

We report a 51-year-old Indian housewife who presented with a three year history of an erythematous and pruritic rash on the neck and upper chest. For the past 23 years, she has been wearing a yellow 'thali' (string necklace) with a gold pendant, as a symbol of her being married. This 'thali' was coated with turmeric powder three monthy. Clinical examination showed a scaly, pruritic, erythematous curvilinear patch around the neck and the upper chest, with its distribution following the area of contact with 'thali'. There was relative sparing of the surrounding skin unexposed to the necklace.

Corresponding Author and Reprint Request
Dr Ramalingam, Rajalingam
Department of Dermatology
Hospital Kuala Lumpur, Malaysia
Email: raj.blueheart@gmail.com

[1] Department of Dermatology, Hospital Kuala Lumpur
[2] Department of Pathology, Universiti Putra Malaysia

Patch test was done with the European Standard Series, plant and textile series (from Chemotechnique Diagnostics), turmeric 'as is', turmeric extract, patient's own turmeric powder and patient's thali 'as is' with and without turmeric. The turmeric was extracted based on the work process practiced in the Department of Occupational and Environmental Dermatology, Malmo Hospital, Sweden. The turmeric was first cut into small pieces, then immersed in acetone solution as a solvent. The extraction procedure was carried out in an ultrasonic bath, whereby the mixture was sonicated for 10 minutes continuously. At the end of sonication, the suspension was cooled to room temperature and then transferred to a rotating evaporator for it to be evaporated under reduced pressure. This resulted in a dry form of turmeric extract. The turmeric extract and turmeric powder were then diluted with petrolatum to 5%, 1% and 0.1% diluents.

The patch test was positive for nickel sulphate, turmeric 'as is' and the turmeric extract. This patient also had a flare of her dermatitis on the chest during patch test. She however tested negative for the 'thali' itself and turmeric powder. Aside from avoiding the use of turmeric and nickel containing products, she was also treated with topical streoids, emmolients and antihistamine, after which her symptoms improved.

To exclude a false positive reaction, further patch tests to the turmeric extract were done on 20 normal controls. None had any positive reaction to the turmeric extract.

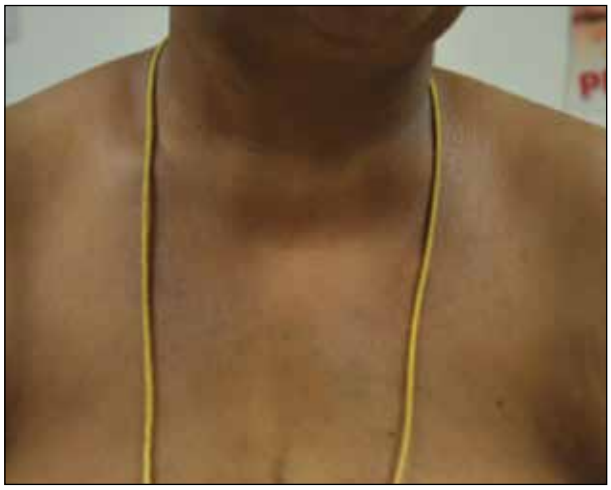

Figure 1. Evidence of a curvilieniar dermatitis on the upper chest.

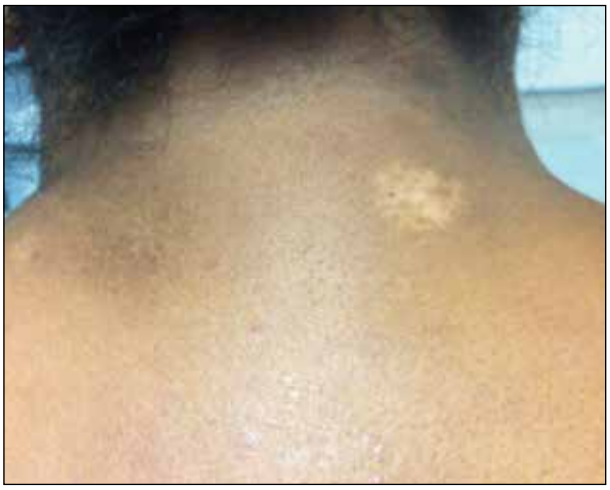

Figure 2. Dermatitis with post inflammatory hyperpigmentation at nape and upper back.

Figure 3. Tumeric immersed in acetone.

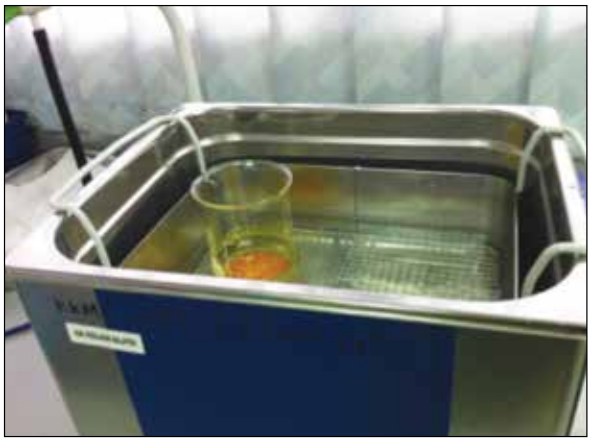

Figure 4. Beaker placed in an ultrasound bath.

Figure 5. Use of a rotating evaporator to remove solvent.

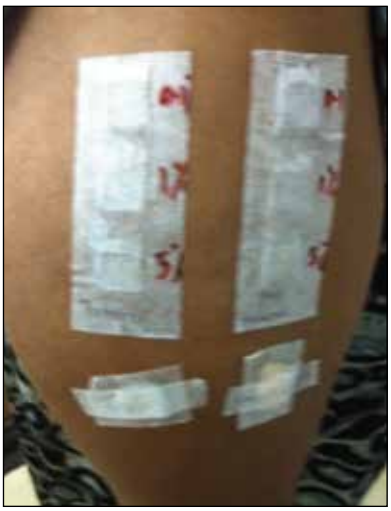

Figure 6. Patch Test.

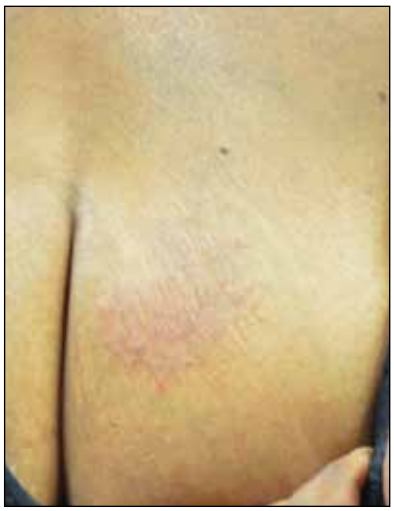

Figure 7. : Flare of dermatitis during patch test.

Discussion

Turmeric, which has been used widely for religious and traditional purposes especially in the Asian culture, is also becoming increasingly popular in the modern society, as a health supplement. It is thus important for physicians to recognize the complications of turmeric, including its effect on the skin, in causing allergic contact dermatitis, as demonstrated in this case.

Turmeric contains up to 5% essential oils and 3% curcumin a polyphenol which is the active substance of turmeric. Curcumin is also known as C-175300 or natural yellow[3]. This oleoresin is prepared via extraction from the turmeric with an organic solvent. The technique of extracting curcumin in this case used ultrasound or sonication to break the cell membranes, which has the advantage of reducing the extraction time considerably and increasing the extract yield. The application of ultrasound disrupts the cell wall structure of turmeric and accelerates diffusion through membranes; thus, the cell lyses and hence facilitates the release of its active contents. We used a rotating evaporator to gently and effectively remove the solvents by evaporation. Following the evaporation of the solvent, the extracted turmeric olioresin may contain 15-40% curcuminoids, of which curcumin predominates. The other two curcuminoids are demethoxycurcumin, and Bis - dimethoxycurcumin.

This patient developed a classic delayed-type hypersensitivity to turmeric. By definition, as it is mediated by immune cells rather than by antibodies, the reaction can be thought of as occurring in two phases, initially a sensitization and then an elicitation response. It is the sensitization phase that is the basis for its classification as an immune mediated reaction. The capacity for sensitization various from person to person, but certain individuals are more prone to developing sensitivity to a particular substance as compared to others. On the other hand, some individuals are actually more resistant to sensitization due to repeated exposure to sub-sensitizing doses of an allergen. Women have stronger cell mediated immunity responses than men. The reason for female preponderance in clinical patch test studies is mainly explained by the large number of metal sensitive females which is largely the result of ear piercing and the greater exposure to fragrances, cosmetics and hair dyes[6]. Number of positive patch-test reactions also tend to increase with age due to accumulation of allergies acquired over lifetime[7]. Aside from the above factors, UVB exposure has been shown to diminish the skin's immune response to contact allergens. Indian women were found to become sensitized to dyes and adhesives in kumkum and bindi. which also contain turmeric[8]. Sensitization and subsequent contact dermatitis may occur due to repeated exposure to the allergen after years of contact. This patient developed a dermatitis after almost 20 years of repeated exposure to turmeric. She has been wearing the 'thali' persistently even during bathing. When exposed to water, the 'turmeric' might have leached out to be in direct contact with skin. During this period sensitization has been accomplished, and the residues of the allergens in the skin react with newly formed T lymphocytes. If a sensitized person is re-exposed to a specific allergen in sufficient concentration, the clinical reaction subsequently develops much more quickly within 24-48 hours, depending on the degree of sensitivity, penetration and other factors. A flare of her dermatitis demonstrates this during patch test when she was again exposed to turmeric. It is worth to note that the negative patch test to turmeric powder was possibly because the allergen was too diluted, as the exact concentration of the turmeric powder that the patient used to coat her 'thali' might have been higher than our allergens She also tested positive to nickel on patch test. The gold pendant on her 'thali' could have contained nickel. This could be confirmed by further analyzing the pendant or simply by doing a dimethylglyoxime (DMG) spot test which would indicate nickel release of >0.5µg/ cm^2 per week.

Conclusion

This is a unique case of allergic contact dermatitis which could have been dismissed as a simple nickel allergy. As demonstrated in this intriguing case of turmeric coated wedding 'thali' allergy, a thorough clinical evaluation and performing patch tests beyond the readily available commercial series is sometimes crucial in obtaining the correct diagnosis. This case report illustrates how the extraction of turmeric and its preparation as an allergen for patch test is performed.

Contact allergic dermatitis to turmeric, or rather curcumin, its active ingredient, is likely to be under-reported in this country. In view of its wide and common use, it is thus imperative for physicians especially dermatologists to recognize its possible effect in causing allergic contact dermatitis in susceptible individuals. The incorporation of turmeric extract in our local plant or even standard series should be the way forward if a bigger study of the prevalence and incidence of this condition showed significant results.

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GENERAL DERMATOLOGY - Short Case

LUPUS TUMIDUS IN A CHINESE MALE: A CASE REPORT

Anisha B, Norashikin S

Introduction

Lupus erythematosus tumidus (LET) was first reported in 1909 by Hoffman, and then again in 1930 by Gougerot and Burnier 1, 2. Thought to be a rare variant of chronic cutaneous lupus erythematosus (CCLE), it presents as succulent erythematous non-scarring plaques on sun exposed areas. Histology shows perivascular and periappendageal lymphocytic infiltrate with large amounts of interstitial mucin deposition. The disease follows a benign course, and the prognosis is favorable. Most reports of LET are of Caucasian patients. Herein we describe a case of LET in a Chinese patient and review the relevant literature.

Case Report

A 27 year old Chinese with a history of papular eczema presented with a five month history of mildly pruritic plaques on both cheeks and left upper arm. There were no preceding history of foreign body injections or insect bites nor symptoms of systemic lupus erythematosus. Examination revealed symmetrical erythematous, indurated plaques on the mandible and left deltoid (figure 1). There was minimal scaling and slight atrophy (figure 2).

Skin biopsy from the left deltoid revealed patchy perivascular and periappendageal lymphocytic infiltrates (figure 3) with copious dermal mucin deposition on Alcian blue (Figure 4) consistent with LET. Blood investigations including antinuclear antibody (ANA) were unremarkable.

He was given topical steroids and oral hydroxychloroquine 200mg daily. On follow up visit, the condition improved.

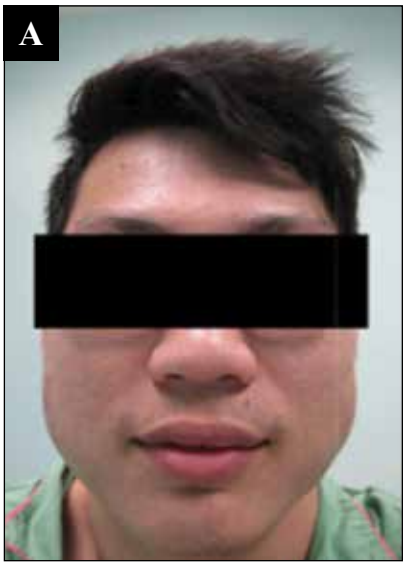

Figure 1. Symmetrical erythematous, indurated ill-defined plaques on bilateral mandibular regions.

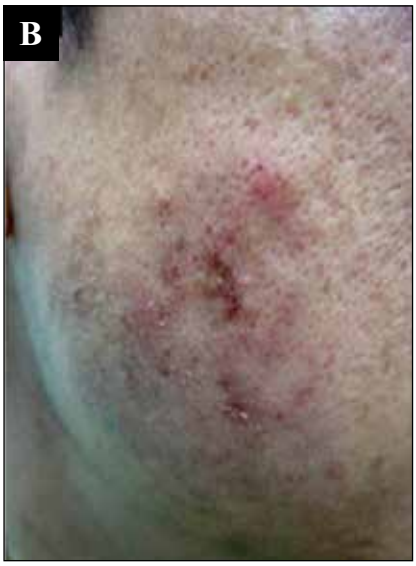

Figure 2. Minimal scaling and slight surface atrophy overlying the erythematous plaques.

Corresponding Author and Reprint Request
Dr. Anisha Bhullar
Department of Medicine, Facuty of Medicine and Health Sciences, Universiti Putra Malaysia
43300 Serdang, Malaysia
Email : anishabhullar@gmail.com

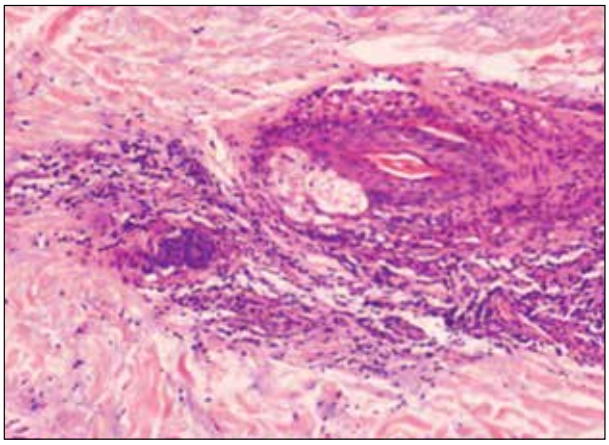

Figure 3. Patchy dermal lymphocytic infiltrates around the perivascular and periadnexal structures (H&E, X10).

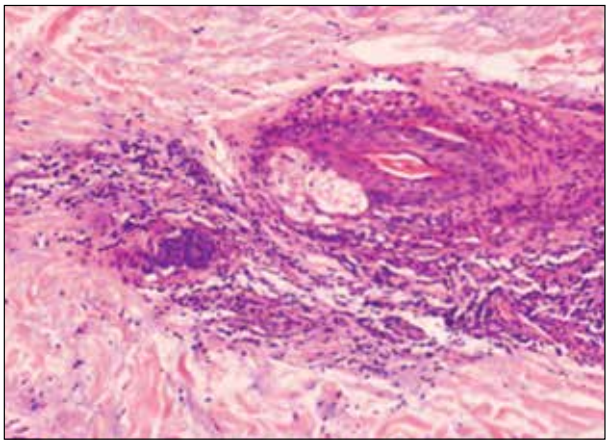

Figure 4. Extensive dermal mucin deposition seen within the collagen bundles (Alcian blue, X10).

Discussion

Lupus erythematosus tumidus (LET) has recently been accepted as a separate subtype of CCLE3. It is a rare dermatological entity and well documented in the Caucasian with sporadic cases in Asia. The pathogenesis of LET has not been clearly established but there has been a prevailing increase in the CD4+/ CD8 inflammatory infiltrates and expression of endothelial cell adhesion molecules with significant decrease in FOXP3+ immunopositivity and CD39+ immunoreactivity.4, 5

The lesions are oedematous, erythematous, non-scarring plaques with very minimal surface atrophy with a predilection for sun exposed areas. Occasionally indurated papules and linear tumid lesions following Blaschko lines can be seen.6,7 The histological characteristics are distinct from other forms of CCLE with well-circumscribed lymphocytic dermal infiltrate in a perivascular and periadnexal pattern and abundant interstitial mucin deposition without epidermal atrophy and follicular plugging7. Basement membrane vacuolation is usually absent 8,9 It is rarely associated with systemic lupus erythematosus.

Photosensitivity has been implicated to play a role in LET. In a case series by Alexiades-Armenakas et al, 86.6 % of the cases were photo-distributed 10 . In a series done in Germany of 62 Caucasian patients with LET, 72% developed characteristic tumid lesions after UV irradiation. A large proportion of their cases had positive reactions to either UVA (50%), UVB (48%) or both wavelengths.11 In Asia, the role of UV ray is not as pronounced. A small case series in Thailand demonstrated only 50% having positive photoreactions 12. Antimalarial is the mainstay of treatment. Other treatment modalities include topical tacrolimus and pulsed dye laser therapy13,14. Sun protection is also vital.

In summary, LET is a disease with a favorable prognosis with the lesions regressing over time. In order to gain a better understanding of the clinical and histological features of this disease in the Asian population, larger scale studies or case series are warranted.

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