ADVERSE DRUG REACTIONS - Case Report

Cetirizine induced bullous fixed drug eruptions

Tan WC, *MRCP*, Chan LC, *MMed*

Keywords *cetirizine, fixed drug eruptions*

Introduction

A fixed drug eruption (FDE) is a distinct drug induced reaction pattern that characteristically recurs at the same site on the skin or mucosa. We report a case of bullous FDE following ingestion of cetirizine, a common treatment for allergic disorders but a rare causative agent for cutaneous adverse drug reaction.

Case report

A 30 year old lady presented with multiple painful blisters in the oral cavity and over the right wrist and left breast for 4 days duration. The lesions developed within 3-4 hours after taken a single tablet of Cetirizine Hydrochloride which was prescribed for her allergic rhinitis. This was the 2nd episode. She had a similar problem about 1 month ago. She took Cetirizine for allergic rhinitis. The similar blistering lesions occurred in the oral cavity and over the right wrist which resolved after 1 week with minimal residual hyperpigmentation. She did not seek medical attention during the first episode. In the past she had taken multiple courses of "clarinase" and "clarityn" without any complaint.

On examination, she was afebrile and well. There were multiple tense bullae at the buccal mucosa and right wrist of various sizes ranging from 4mm - 10mm (Figure 1 & 2). A hyperpigmented patch was noted over the left breast. The blisters were surrounded by an erythematous rim. There was no lesions elsewhere. No target lesion were seen and no eye and genital involvement were detected. Skin biopsy, patch test and oral provocation test were not done in this patient because of the clear drug history and the patient did not consent for the tests.

Correspondence
Tan Wooi Chiang, *MRCP*
Department of Dermatology, Penang Hospital
Jalan Residensi, 10990 Penang
E-mail : tanwooichiang@yahoo.com

The patient was counselled on medication avoidance and possible cross-reactions of similar medications such as cetirizine, hydroxyzine, levocetirizine and ethylenediamine. She was given a short course of oral prednisolone, in a tapering dose, for 2 weeks. The lesions improved and healed with residual hyperpigmentation.

Figure 1 Tense bullae with erythematous rim over the right wrist

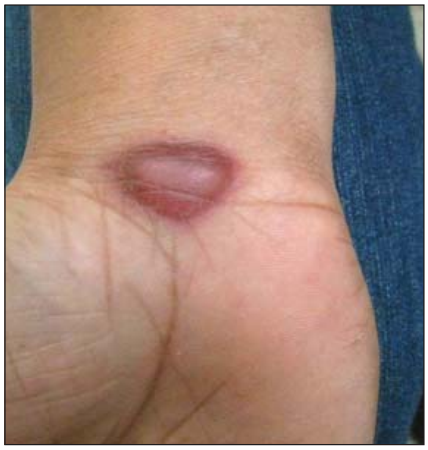
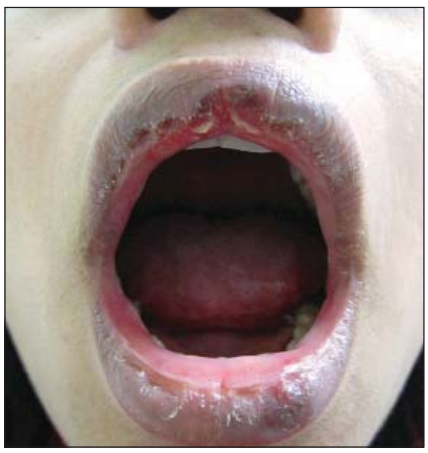


Figure 2 Circular hyperpigmented lesions over the lip with superficial erosions



Discussion

Brocq first introduced the term "fixed drug eruption" in 1894[1]. The term FDE describes the development of one or more annular or oval erythematous patches as a result of systemic exposure to a drug. These reactions normally resolve with hyperpigmentation and may recur at the same site with re-exposure to the drug[2]. Repeated exposure to the offending drug may cause new lesions to develop in addition to "lighting up" the older hyperpigmented lesions.

The pathogenesis of FDE is still enigmatic. The most commonly accepted hypothesis is the persistence of memory T cell in the affected skin[3]. CD8+ cells phenotypically resembling effector memory T cells have been shown to be greatly enhanced in the lesions of FDE.

The skin lesion of FDE usually starts as an erythematous macule then subsequently evolves into a plaque. Vesicles and bullae develop at a later stage. The lesions can occur on any part of the skin and mucous membranes. The sites of predilection are the genitalia, limbs, sacral region, palmar and plantar area. The oral mucosa may be involved in association with skin lesions or in isolation.

More than 100 drugs have been implicated in FDE but the causative drugs that are commonly associated with FDE include co-trimoxazole, tetracycline, non-steroidal anti inflammatory drugs (NSAIDs), phenytoin, griseofulvin, salicylates, penicillin and phenolphthalein[4]. It has also been described as a side-effect of some anti-H1-antihistamine drugs, such as cyclizine lactate, diphenhydramine hydrochloride, phenothiazines, hydroxyzine[5] and loratadine[6]. To the best of our knowledge, there were only a few cases of FDE secondary to cetirizine being reported[7-8].

Cetirizine is a specific histamine H1-receptor antagonist and a second-generation antihistamine and generates the lowest rate of cutaneous reactions. Cetirizine is used worldwide in the treatment of allergic disorders and is generally well tolerated without much problem. Although topical antihistamines commonly leads to sensitization and can cause contact and photo-contact dermatitis[9], skin reactions provoked by their systemic use is rare[10-11].

Cross-reactions among ethylenediamine, cetirizine and hydroxyzine had been reported by Bark-Lin Lew et al[12]. Cutaneous reactivity to the H1-antihistamines is caused by the fact that they share the same chemical piperazine structure and similar pharmacologic profiles[12-13]. In vivo, 45% of hydroxyzine is transformed into cetirizine and levocetirizine is the active (R)-enantiomer of cetirizine[5].

Oral challenge can be used to confirm the etiology of FDE[14]. But there are risks involved in this approach, mainly anaphylactic reactions or intense lesional reactivation with a significant increase in the number of lesions.

Patch testing is not regularly performed. Reappearance of skin lesions with re-challenge identifies offending agent. Furthermore, reactivity of patch tests in FDE is variable. Some study showed patch testing at the site of a previous lesion yields a positive response in up to 43% of cases[15], but the reactivity depends on the drug and the vehicle. Reactivity is usually seen before 24 hours and is observed exclusively on the lesional skin. Patch testing is safer than oral provocation tests. It also allows the study of several drugs at the same time.

Physicians should watch carefully for eruptions from antihistamines and not to misinterpret them as unresponsiveness to the medications. Because oral antihistamines are one of the most common medications used to treat itchy dermatoses, it is prudent to listen to patients when they indicate that these medications seem to worsen their condition.

This report highlights an uncommon causative agent of FDE.

References

1. Brocq I. Eruption erythemato-pigmentee fixe due al' antipyrine. Ann Dermatol Venereol. 1894; 5: 308-13. Quoted from: Shiohara T, Nickoloff BJ, Sagawa Y, Gomi T et al. Fixed drug eruption. Expression of epidermal keratinocyte intercellular adhesion molecule-1 (ICAM-1). Arch Dermatol 1989; 25: 1371-6
2. Lee AY. Fixed Drug Eruptions: Incidence, recognition, and avoidance. Am J Clin Dermatol 2000; 1(5):277-85

3. Shiohara T, Nickoloff BJ, Sagawa Y, Gomi T et al. Fixed drug eruption. Expression of epidermal keratinocyte intercellular adhesion molecule-1 (ICAM-1). Arch Dermatol 1989; 25: 1371-6
4. Magee P. Drug-induced skin disorders. In: Walker R, Edwards C 'editors'. Clinical Pharmacy and Therapeutics. 3rd edition. Phildelphia: Churchill Livingstone 2003; 843-52
5. Mahajan VK, Sharma NL, Sharma VC. Fixed drug eruption: a novel side-effect of levocetirizine. Int J Dermatol 2005; 44: 796-798
6. Pionetti CH, Kien MC, Alonso A. Fixed drug eruption due to loratadine. Allergol Immunopathol (Madr) 2003; 31: 291-293
7. Inamadar AC, Palit A, Athanikar SB, Sampagavi VV, Deshmukh NS. Multiple fixed drug eruptions due to cetirizine. Br J Dermatol 2002; 147: 1025-6
8. Kranke B, Kern T. Multilocalized fixed drug eruption to the antihistamine cetirizine. J Allergy Clin Immunol 2000; 106: 988
9. Kulthanan K, Tiprungkorn P, Linpiyawan R. Cutaneous reaction to oral antihistamine. Clin Exp Dermatol 2003; 28: 229-30
10. Spencer CM, Faulds D, Peters DH. Cetirizine: a reappraisal of its pharmacological properties and therapeutic use in selected allergic disorders. Drugs 1993; 46: 1055-80
11. Campoli-Richards DM, Buckley MMT, Fitton A. Cetirizine: a review of its pharmacological properties and clinical potential in allergic rhinitis, pollen-induced asthma, and chronic urticaria. Drugs 1990; 40: 762-81
12. Lew BL, Haw CR, Lee MH. Cutaneous drug eruption from cetirizine and hydroxyzine. J Am Acad Dermatol. 2004; 50(6): 953-6
13. Assouere MN, Mazereeuw-Hautier J, Bonafe JL. Cutaneous drug eruption with two antihistaminic drugs of the same chemical family: cetirizine and hydroxyzine. Ann Dermatol Venereol 2002; 129: 1295-1298
14. Kauppinen K, Stubb S. Fixed eruptions: causative drugs and challenge tests. Br J Dermatol 1985; 112: 575-578.
15. Barbaud A, Reichert-Penetrat S, Trechot P, et al. The use of skin testing in the investigation of cutaneous adverse drug reactions. Br J Dermatol 1998; 139: 49-58.

ADVERSE DRUG REACTIONS - Update

Intravenous Vitamin K Administration

Norkasihan Ibrahim, Fadilah Othman, Norliza Mat Ariffin, Zainon Abudin

Vitamin K injection is a common treatment for the prevention of bleeding. It acts by promoting the formation of liver coagulation factor II, VII, IX and X. Anaphylactic reaction with the use of this product is rare and suspected to be caused by its non-ionic solubilizing and emulsifying agent; Cremophor EL formulated in Vitamin K Injection (KISAN® 10mg/ml)[1,2]. The main component of Cremophor EL is glycerol-polyethylene glycol ricinoleate. It is widely used as a formulation vehicle for various poorly-water soluble drugs including the lipid soluble vitamin A, D, E and K. Extra precautions should be taken when administering pharmaceutical products containing Cremophor EL. This agent has been found to cause severe anaphylactoid-like reaction especially when high doses are administered via bolus infusion[1,2]. It is recommended to dilute Vitamin K Injection (KISAN® 10mg/ml) in normal saline, dextrose 5% or dextrose saline to a concentration not exceeding 1mg/ml. The diluted product should be infused for not less than 30 minutes[1,2].

References

1. KISAN® Injection 10mg/ml Product Information Leaflet
2. Cremophor EL the drawbacks and advantages of vehicle selection for drug formulation.

Correspondence
Norkasihan Ibrahim
Department of Pharmacy, Selayang Hospital Selangor
E-mail : NrKasihan@selayanghospital.gov.my

ADVERSE DRUG REACTIONS - Short Communication

Hypersensitivity reaction to intramuscular Vitamin K

Raoul RS[1], *MBBS*, Rohna R, *MRCP*, Shalini Kumar[2], *MPath*

Dear Editor,

We have encountered a 42 year old Malay lady with antiphospholipid syndrome for the past 3 years presented with red cutaneous swelling at both deltoid regions a week after given intramuscular Vitamin K injection. She was referred to us 6 weeks following the Vitamin K injection. Psoriasiform plaques with perilesional warm erythematous skin were noted over both deltoid regions (Figure 1). A skin biopsy (Figure 2a & 2b) showed subepidermal vesicular change with formation of bullae including basal cell vacuolation and necrotic keratinocytes. The upper dermis was infiltrated with mononuclear inflammatory cells. The features are in keeping with a hypersensitivity reaction.

A diagnosis of hypersensitivity reaction to Injection Vitamin K was made based on the history and skin biopsy. The skin lesions flattened and became hyperpigmented after two weeks application of potent topical steroid and oral antihistamine.

Figure 1 Lesion at right deltoid

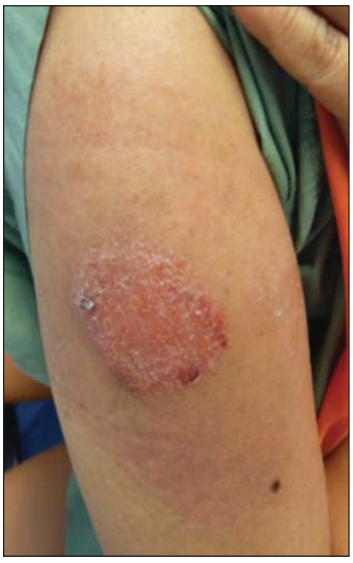


Figure 2A HPE of skin biopsy at 4X magnification

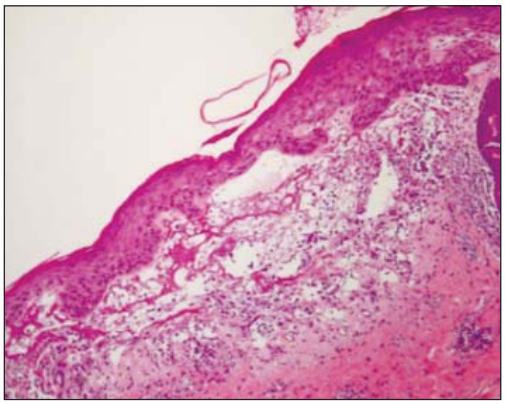
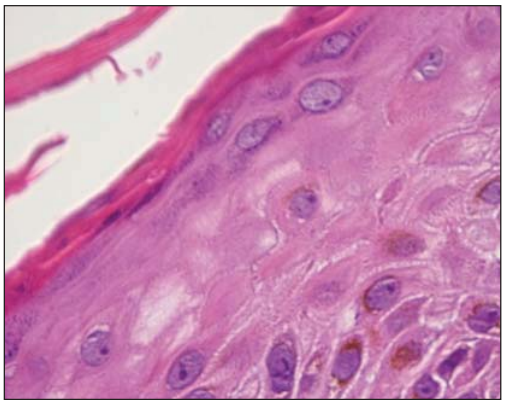


Figure 2B HPE of skin biopsy at 40X magnification



Correspondence
Raoul Roger Sibert, *MBBS*
[1]Department of Dermatology
Selayang Hospital, Selangor
E-mail : raoul@selayanghosp.gov.my

[2]Department of Pathology, Selayang Hospital

Discussion

Vitamin K1 (Phytomenadione) is a fat soluble, naturally occurring vitamin used to treat certain coagulation disorders[1]. Injectable vitamin K can cause skin reactions whatever the dose and mode of injection[2]. The first few cases of hypersensitivity to vitamin K were reported in patients with liver disease. The pathophysiological mechanism of the acute form would involves type IV allergy to Phytomenadione[3]. There are three distinct types of cutaneous reactions to vitamin K1: localized eczematous, localized morphea-form[4-5], and, very rarely, diffuse maculopapular eruption[2]. The eczematous type appears at the site of injection.

The morphea-form type is a localized morphea-form patch that appears at the site of injection. The diagnosis of an adverse cutaneous reaction to vitamin K can be made if the possibility is considered. Many of these reactions are very slow to clear up and some may persist as a chronic sclerodermoid change[4-5]. Managing may be frustrating for both the patient and the clinician.

In our patient, the localized psoriasiform dermatitis with perilesional erythema and oedema was treated with topical steroids and resolved with post-inflammatory hyperpigmentation.

References

1. Gimenez-Arnau, A.M., A. Toll, and R.M. Pujol, Immediate cutaneous hypersensitivity response to phytomenadione induced by vitamin K in skin diagnostic procedure. Contact Dermatitis 2005; 52(5): 284-5
2. Moreau-Cabarrot, A., F. Giordano-Labadie, and J. Bazex, [Cutaneous hypersensitivity at the site of injection of vitamin K1]. Ann Dermatol Venereol, 1996; 123(3): 177-9
3. Bruynzeel, I., et al., Cutaneous hypersensitivity reactions to vitamin K: 2 case reports and a review of the literature. Contact Dermatitis 1995; 32(2):78-82.
4. Finkelstein, H., M.C. Champion, and J.E. Adam, Cutaneous hypersensitivity to vitamin K1 injection. J Am Acad Dermatol, 1987; 16(3 Pt 1): 540-5.
5. BK Pang, V Munro, S Kossard. Pseudoscleroderma secondary to phytomenadione (vitamin K1) injections: Texier's disease. Australasian Journal of Dermatology, 1996 - interscience.wiley.com

AUTOIMMUNE DISORDERS - Case Report

Pemphigus foliaceous and thymoma: a report of 2 cases

Tang MM[1], *AdMDerm*, Lee YY[2], *AdMDerm*, Suganthi T[1], *MMed (UKM)*

Keywords *autoimmune bullous disease, pemphigus, thymoma, paraneoplastic*

Introduction

Pemphigus foliaceous (PF) is an autoimmune blistering disease resulting from acquired immunoglobulin G autoantibodies against desmoglein 1 of the skin, which is one of the adhesion molecules of keratinocytes. Clinically patients with PF develop crusted and scaly erosions mainly over the seborrhoeic distribution i.e. the face, scalp and upper trunk. Mild cases of PF may be localized but in some cases it may progress to erythrodermic exfoliative dermatitis. There is however no mucosal involvement in PF in contrast to pemphigus vulgaris and paraneoplastic pemphigus. Light microscopy of lesional biopsy shows subcorneal acantholysis. Direct immunofluorescence study of perilesional skin reveals presence of intraepithelial intercellular deposit of IgG and C3. We describe 2 cases of PF in the presence of thymoma, a relatively rare association, which could further support the fact of thymoma associated autoimmune disease.

Case report 1

A 35-year-old Malay painter, presented to us in August 2008 with a month history of flaccid blisters starting over the anterior chest, progressively involving the face, arms, abdomen, legs and then became generalized. There was no oral or genital involvement. The lesions were not aggravated by sun exposure. There was no significant drug history. There were no other constitutional symptoms. On further questioning, the patient was diagnosed to have a thymoma after being investigated for difficulty in swallowing and changes of voice in 2006. He was advised for surgical resection of the mediastinal mass by cardiothoracic surgeon. He however declined any form of surgery and defaulted subsequent follow up.

On examination, the patient was afebrile. His blood pressure was normal but has tachycardia. He was erythrodermic with erosions and crusts over the face, scalp, chest, back, arms and thighs sparing the oral mucosal and genitalia. Examination of other systems revealed no abnormality.

Skin biopsy of lesion demonstrated subcorneal bullae with acantholytic cells (Figure 1). The direct immunofluorescence study showed strong intraepidermal IgG deposits (Figure 2).

These confirmed the clinical diagnosis of pemphigus foliaceous. His indirect immunofluorescence study showed a titre of 1:320. His blood counts were normal with ESR of 35mm/hr. Full blood picture showed leucocytosis with predominant neutrophils, toxic granulation were seen but immature cell were absent. His alanine aminotransferase was 283U/l (normal 0-41 U/l) Serology tests for HepBsAg, Anti Hep C antibody, anti-HIV-1&2, anti-smooth muscle antibody were negative. Ultrasound of abdomen showed normal liver echogenicity and echotexture with no other abnormality. His chest radiograph revealed a mass at the right mid zone, obliterating the right heart border (Figure 3).

Due to the severity of his skin lesions, the patient was admitted and initially given intravenous hydrocortisone 100mg 8hrly. Multiple courses of intravenous antibiotic were given for the superimposed bacterial infection of the eroded skin. The skin lesions progressed further despite with above treatment and aggressive skin nursing care. Steroid sparing immunosuppresants such as azathioprine, mycophenolate mofetil and methotrexate were not considered due to hepatitis. Due to the poor response after 3 weeks of intravenous hydrocortisone, we initiated pulse

Correspondence
Tang MM, *AdMDerm*
[1]Department of Dermatology, Hospital Kuala Lumpur
E-mail : minmoon2005@yahoo.com

[2]Department of Dermatology
University Malaya Medical Centre

Figure 1 The skin biopsy showed superficial subcorneal bulla with collection of neutrophils. Haematoxylin and Eosin stain x400

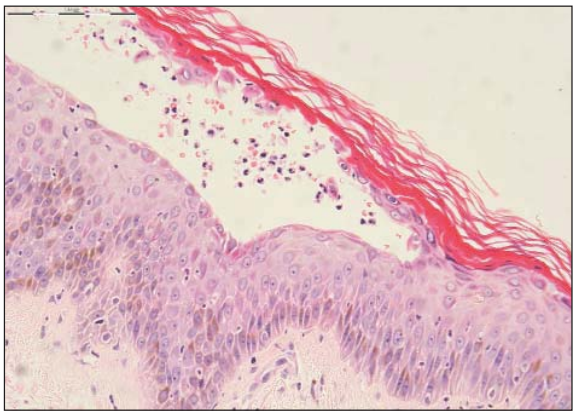


Figure 2 Positive deposition of Ig G of the intraepithelial and intercellular spaces. (Direct immunofluorescence stain IgG x200)

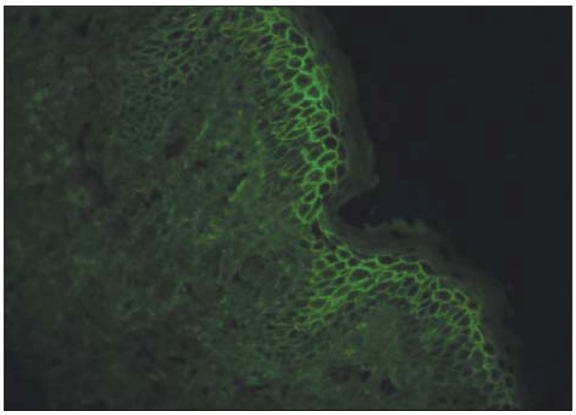


Figure 3 Presence of a mass at the right mid zone on the chest radiograph

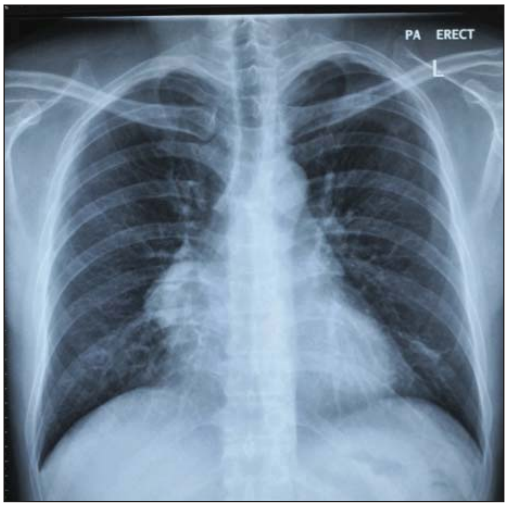
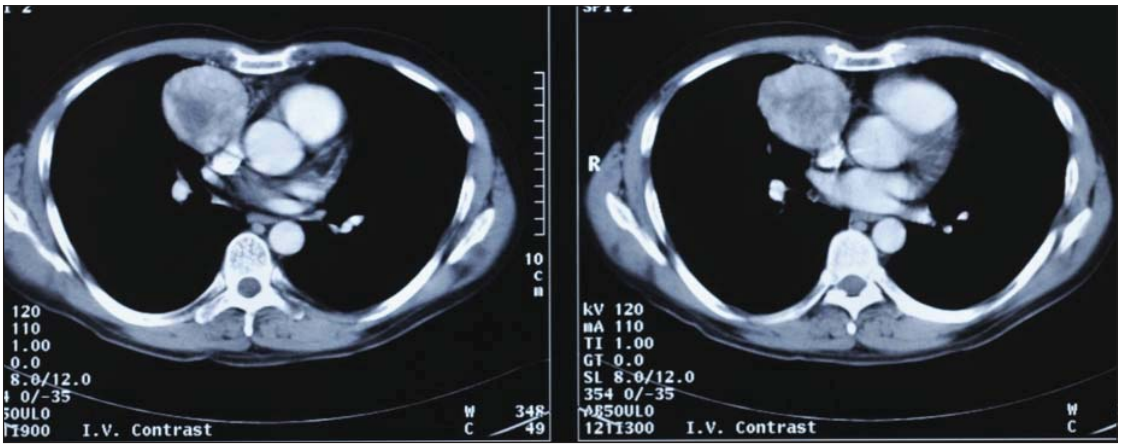


Figure 4 Computed tomography of thorax showed a heterogeneously enhancing anterior mediastinal mass measuring 5.5x5.6cm on the right mid thoracic level



dexamethasone-cyclophosphamide (DCP) regime after obtained consent from the patient. This regime was modified to include oral prednisolone at 0.5mg/kg/day, after the first 2 pulses, the skin lesions stopped progressing and healed gradually.
Further investigation included a CT thorax which showed a heterogeneously enhancing anterior mediastinal mass measuring 5.5 x 5.6cm on the right mid thoracic level. There was calcification within the mass. There was clear demarcation with the adjacent vessels. The mediastinal lymph nodes were not enlarged and there were no lung nodules or pleural effusion (Figure 4). Fatty liver was also noted in the CT scan. His carcinoembryonic antigen (CEA) was 11.1U/l Units (twice above the upper normal limit) Other tumour markers included lactate dehydrogenase and alpha fetoprotein were not elevated. Computed tomography guided biopsy confirmed a type A thymoma.

The patient was repeatedly counseled on the need for surgical excision of the thymoma as the pemphigus foliaceous which could be a paraneoplastic manifestation may probably resolve post-thymectomy. However the patient adamantly refused surgical intervention. He subsequently underwent 11 pulses of DCP and he has been lesion free for the past 4 months.

Case report 2

A 43-year-old Chinese lady presented to us in March year 2004 with 2-week history of erosions started over the neck which became generalized. There were however no oral or genital ulcers. She was diagnosed to have malignant thymoma in 1997 and had thymectomy done in the same year. She was also suffering from end stage renal failure due to chronic glomerulonephritis in 1999 and had a cadaveric renal transplant done in China in 2001. After the renal transplant, she was prescribed a few immunosuppressants which included oral prednisolone, high dose of oral cyclosporine and oral mycophenolate mofetil. She had recurrence of thymoma in 2002 and was re-operated followed by 30 fractions of radiotherapy. Unfortunately she developed another recurrence of the thymoma at the end of 2003 and the tumour was deemed inoperable. She was referred to Oncology team and was offered chemotherapy. However she declined. The dosage of cyclosporine was reduced and mycophenolate mofetil was stopped.

Clinically she was anemic. She was not jaundiced. There were no lymph nodes palpable. She had erosions with crust over the face, chest, back, abdomen and limbs. There was crust over the lower lip but there were no oral or genital ulcers. There was also no hepatosplenomegaly or lymphadenopathy.

Skin biopsy demonstrated subcorneal blister with presence of intraepithelial intercellular deposits of immunoglobulin G and C3 and these confirmed the diagnosis of pemphigus foliaceous. Her haemoglobin was 6.7g/dl with reticulocyte count of 6.8%. Her direct Coomb's test was positive but the serum bilirubin was not raised. Her full blood picture revealed normocromic normocytic anaemia with features of combined iron and folate deficiency, and there was no active haemolysis noted. Her upper endoscopy and colonoscopy showed normal findings. She had a positive homogenous antinuclear antibody test but her extractable nuclear antibody and anti ds-DNA antibody were non reactive. Her renal profile and liver function tests were also normal. Computed tomography of the thorax showed a solid mass at anterior lower mediastinum with multiple right paracardiac lymph nodes.

Prednisolone at the dose of 1mg/kg/day was started initially and new erosions ceased to form and pre-existing lesion healed slowly. There were no other steroid sparing agents added while the cyclosporine was maintained at 25mg bd. Nevertheless she developed new lesions each time when the prednisolone was tapered below 20mg/day. She was at the same time co-managed by oncology team and nephrology team. The malignant thymoma had increased in size in 2007 and she had right pleural effusion, enlarged mediastinal lymph nodes and liver metastasis. She was given 6 cycles of palliative chemotherapy consisting of Doxorubicin, Cisplatin and Cyclophosphamide every 3 weeks. At the end of the chemotherapy, the thymoma shrank, the liver metastasis resolved and the right lower thorax pleural effusion was slightly improved. The skin condition was well controlled at that point of time with oral prednisolone 15-20mg a day.

Unfortunately she deteriorated rapidly in September 2008 when she developed ascites, ankle oedema and right pleural effusion. She was dependant on continuous nasal oxygen therapy and her symptoms were only slightly controlled with symptomatic treatment and oral dexamethasone. She was then finally succumbed to the disease at home in October 2008.

Discussion

Thymoma is the commonest primary mediastinal tumour in the adult population with those associated with paraneoplastic syndromes, tend to occur at younger age group[1]. It has been associated with many paraneoplastic syndromes such as the neuromuscular syndrome with Myasthenia Gravis being the most common (30-45%); hematologic syndromes; collagen and autoimmune disorders; immune deficiency syndromes; dermatologic disorders; endocrine disorders; gastrointestinal disorders and renal disease[2]. Pemphigus, alopecia, chronic candidiasis. have rarely been reported to sporadically occur in the presence of thymoma.

Both our patients presented with history of thymoma a few years prior to the onset of cutaneous manifestations. They developed superficial erosions without any mucous membrane involvement. There was no history to suggest myasthenia gravis. The histopathologic and direct immunofluorescence features confirmed pemphigus foliaceus. The clinical manifestations were not suggestive of a typical natural history paraneoplastic pemphigus (PNP). There was no intractable stomatitis which is the most constant feature of PNP[3]. Besides, the cutaneous lesions in PNP are usually polymorphic including macules, tense blisters, erosions, EM-like, lichenoid and GVHD-like[3]. In both our cases, there were only erosions with no other form of lesions. In addition, the histopathology of PNP is also variable including a combination of PV-like, EM-like, LP like histological features i.e. intraepidermal suprabasal acantholytic blister; interface dermatitis with vacuolar degeneration of basement membrane zone, necrotic keratinocytes, superficial perivascular lymphocytic infiltrates and lichenoid dermatitis[3]. Direct immunofluorescence shows IgG and complement on intercellular spaces of keratinocytes and basement membrane zone. These were not present in our patients. Positive staining of rat urothelium on indirect immunofluorescence is another diagnostic criteria for PNP but this test is not available in this country.

The coexistence of thymoma and pemphigus was first reported in 1964 by Kough & Barners and since then case reports have appeared sporadically in the literature. The subtypes of pemphigus that were indentified to be associated with thymoma in the literature include pemhigus vulgaris, pemphigus foliaceous and pemphigus erythematosus. The types of thymoma reported ranged from benign to malignant. It is still not understood exactly how thymic neoplasms are associated with pemphigus. The thymus gland is important in the immunological make-up of an individual. It is postulated that the altered constituents of the thymus may act as antigens which may mimic epidermal intercellular adhesion molecules in pemphigus, which induce autoantibodies that react against the epidermal intercellular adhesion molecules. This is supported by the development of antibodies against intercellular adhesion molecules in 3 patients with thymomas without clinically apparent pemphigus reported by Imamura et a[l4]. Takeshita K et al in 2000 also reported a case of thymoma associated with pemphigus foliaceous who had underwent total thymectomy which resulted in the resolution of cutaneous lesions and reduction of serum anti-Dsg 1 antibody[5].

In our first patient, his skin lesions were not well controlled with high dose of systemic steroid. Pulse dexamethasone-cyclophosphamide (DCP) was used not only because of the severity of the skin condition but also because other agents such as azathioprine and MMF were contraindicated in the face of hepatitis. The skin lesions resolved on the DCP regime with oral prednisolone and oral cyclophosphamide in between pulses. The patient refuses surgical intervention despite extensive counseling. We are currently exploring other modality of treatment such as chemotherapy or radiotherapy for the thymoma. Interestingly, Loehrer PJ et al reported a 50% response rate in patients with unresectable / advance thymoma treated with the PAC regime (combination of cisplatin, doxorubicin and cylcophosphamide)[6]. This paper made us wonder if the cyclophosphamide, which is a known agent in the treatment of thymoma administered in our patient, halted the progression of his thymoma through its antimitotic property.

Our second patient had a rather complex combination of malignant thymoma, chronic glomerulonephritis leading to end stage renal failure with renal transplant and pemphigus foliaceus. It is unclear whether the chronic glomerulonephritis could be part of the disorders associated to epithelial thymic tumour. Besides, her treatment with immunosuppressive agents such as high dose cyclosporine, mycophenolate mofetil and

prednisolone post renal transplant might play a role in escalating her tumour progression as she experienced the first relapse of thymoma a year after the renal transplant. Studies have shown that organ-transplant recipients have an increased incidence of cancer as compared with an age-matched healthy population or with patients undergoing dialysis. London NJ et al found that after 20 years of immunosuppressive therapy, 40 percent of recipients had cancer[7]. Sasaki et al reported immune suppression may have been a contributing factor in the induction of thymoma[8]. In our patient, the dose of cyclosporine was reduced and the MMF was taken off successfully without compromising the renal graft function. However the thymoma progressed for the next 4 years since the diagnosis of pemphigus folicaceous and she succumbed to her malignancy with metastases. The aggressive nature of her malignant thymoma was the main contributing factor to the mortality. Her skin lesions were well controlled few months before she passed away, probably because she received a cycle of palliative chemotherapy consisting of Doxorubicin, Carboplatin and Cyclophosphamide and also dexamethasone in her palliative care period.

In conclusion, we reported 2 cases of pemphigus foliaceous associated with thymic neoplasms which could be the manifestations of immune system instability. Based on our experience managing these 2 patients, it is pertinent for us to seriously consider the possibility of thymoma if there is abnormal mediastinal widening or mass in the chest radiograph of patients presenting with pemphigus foliaceous. Further studies are needed to analyze the pathogenesis of the natural course of pemphigus foliaceous co-existing with, or after discovery of thymoma and also the role of thymectomy in relation to the natural course of the cutaneous manifestations.

References

1. Detterbeck FC, Parsons AM. Thymic tumors. Ann Thorac Surg 2004;77: 1860-9
2. Venuta F, Anile M, Diso Daniele et al. Thymoma and thymic carcinoma. Eur J Cardiothorac Surg (2009), doi:10.1016/ j.ejcts.2009.05.038
3. Camisa C, Helm TN. Paraneoplastic pemphigus is a distinct neoplasia-induced autoimmune disease. Arch Dermatol 1993;129:883-6
4. Imamura S, Takigawa M, Ikai K, Yoshinaga H, Yamada M. Pemphigus foliaceus, myasthenia gravis, thymoma and red cell aplasia. Clin Exp Dermatol 1978; 3: 285-291
5. Takeshita K, Amana M, Shimizu T et al. Thymoma with pemhigus foliaceous. Int Med 2000; 39:742-747
6. Loehrer Sr PJ, Chen M, Kim K et al. Cisplatin, doxorubicin and cyclophosphamide plus thoracic radiation therapy for limited stage unresectable thymoma: and intergroup trial. J Clin Oncol 1997; 15:3093-9
7. London NJ, Farmery SM, Will EJ, Davison AM, Lodge JP. Risk of neoplasia in renal transplant patients. Lancet 1995;346:403-6
8. Sasaki J, Saito A, Hoshino M, Yanagawa S, Mizoguchi M, Takahashi H. A case of pemphigus vulgaris with mediastinal tumor, a case report and literature review. Hifukano Rinsho (Rincho Derma) 31: 479-483, 1989

AUTOIMMUNE DISORDERS - Case Report

Mimicry of the great mimicker

Lee YY, *AdMDerm*, Priya GP, *MRCP*, Suganthi T, *MMed*

Introduction

Pemphigus erythematosus (PE) is an autoimmune bullous disease where the antibody is directed at desmoglein 1, a desmosomal protein important in keratinocyte adhesion, resulting in intraepidermal bullae. Pemphigus erythematosus also known as Senear-Usher syndrome, is a variant of superficial pemphigus with features of both lupus erythematosus and pemphigus. The skin biopsy exhibits histopathological and direct immunofluoresence features of both lupus eythematosus and pemphigus i.e. granular IgG and C3 at the basement membrane zone and intercellular IgG and C3 on the cell surface of keratinocytes with circulating antinuclear antibodies in the blood. We describe an interesting case of a Myanmar refugee with pemphigus erythematosus presenting with cutaneous features resembling lupus erythematosus.

Case report

A 26-year-old Myanmar refugee presented in April 2009 with four months history of erythematous papules, pustules and blisters over both her cheeks (Figure 1). The lesions progressed to involve her whole face, anterior chest wall, arms and trunk with no oral or genital involvement. There was no previous similar skin rash. The lesions were pruritic but not painful.

The erythema was aggravated by sun exposure. However, she was systemically well. There were no symptoms suggesting an underlying connective tissue disease, such as joint pain, hair loss, oral ulcers or muscle weakness. She denied recent intake of new medicines, traditional medications, or supplements.

Figure 1 Extensive, erythematous papules and pustules involving the malar regions bilaterally

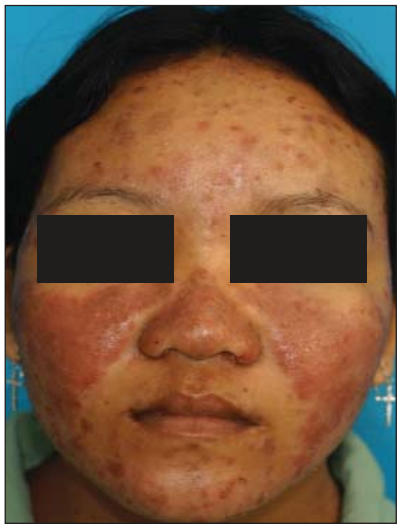
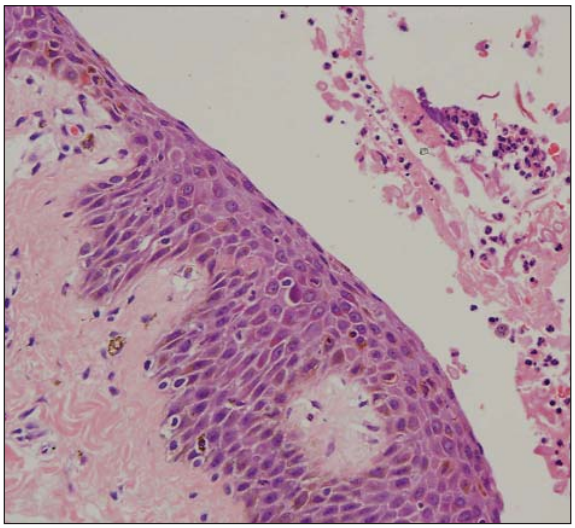


Figure 2 Pemphigus Erythematosus
Histopathology: A subcorneal blister is filled with acantholytic keratinocytes and numerous neutrophils. The granular layer is diminished but dyskeratotic acantholytic granular cells are not seen in this case. (H&E 200X)



Correspondence
Priya Gill, *MRCP (UK)*
Department of Dermatology, Hospital Kuala Lumpur
Wilayah Persekutuan, Malaysia
E-mail : drpriya74@yahoo.com

Clinically, there were extensive erosions with erythematous crusts and scales involving the malar region, anterior trunk and entire back. There were also a few intact flaccid bullae on the upper limbs and back. However, there was no Raynaud's phenomenon or cutaneous vasculitic changes. Examination of other systems was normal. The provisional diagnosis was pemphigus erythematosus with a differential of pemphigus foliaceus and acute cutaneous lupus erythematosus.

Skin biopsies were performed from 2 sites; an erythematous, scaly patch from the left cheek and a flaccid bulla from the right forearm. The first biopsy showed follicular plugging, mild oedema of the upper dermis with perivascular and periappendegeal infiltration by chronic inflammatory cells plus focal basal cell vacuolation. The second biopsy demonstrated subcorneal bullae containing few neutrophils and acantholytic cells (Figure 2). Direct immunofluorescence showed moderately strong intraepidermal and dermo-epidermal junction IgG deposits. These confirmed the clinical diagnosis of pemphigus erythematosus.

The full blood counts, renal profile, liver function and serum complements were normal. Anti nuclear antibody and extractable nuclear antibody were non reactive. There was microscopic hematuria which could be attributed to a Klebsiella urinary tract infection.

The patient was admitted to the dermatology ward and subjected to intensive skin nursing. She was prescribed intravenous hydrocortisone 100mg 8 hourly and oral Cefuroxime Axetil (for urinary tract infection) and improved dramatically over the next 5 days. The patient requested an early discharge and was discharged on Prednisolone 45mg daily, calcium carbonate 600mg twice daily, _-calcidiol 0.5mg daily, titanium dioxide as a sun block and topical betamethasone valerate 0.025% twice daily. We planned to add on oral Azathioprine 100mg daily as a steroid sparing agent on follow up.

Unfortunately, she defaulted her follow up and was not contactable thereafter.

Discussion

Pemphigus erythematosus (PE), also known as Senear-Usher syndrome is an antibody-mediated autoimmune bullous disease with combined features of pemphigus foliaceous and lupus erythematosus. The term 'pemphigus erythematosus' was initially coined to describe patients with immunological features of both lupus erythematosus and pemphigus foliaceous. PE represents approximately 8% of all cases of pemphigus. In pemphigus foliaceous, antibodies were directed at desmoglein 1, a desmosomal protein important in keratinocyte adhesion.

Histopathology and direct immunofluorescence examination of PE elucidate features of both lupus erythematosus and pemphigus, i.e. granular IgG and C3 at the basement membrane zone and intercellular IgG and C3 on the cell surface of keratinocytes in a fishnet appearance 1.

PE affects mainly middle-aged adults and manifests itself on sun-exposed areas as flaccid bullae with scales and crusts. The main areas of distribution include the scalp, face, upper chest and back. Facial manifestations are localized to the typical butterfly distribution as seen in lupus erythematosus (LE)[2,3]. Pemphigus and LE have been reported to coexist in the same patient. However, the incidence of the co-existence is low. Most of these patients are non-Caucasian females in their reproductive age groups, which is similar to LE. In a review conducted by Mohsin Malik et al, majority of these patients had pemphigus vulgaris (PV) and less commonly the other variants of pemphigus[4].

Our patient, presented with a history and clinical examination suggestive of LE such as photosensitivity and malar rash. The bullae also led us to think of bullous lupus erythematosus. However, the connective tissue screening was unremarkable with a negative anti nuclear factor (ANA) and extractable nuclear antibody (ENA). The confirmatory diagnosis of PE was only elucidated from the skin histopathological and direct immunofluorescence examination whereby there were features of both pemphigus foliaceous and lupus erythematosus.

The natural course of PE is relatively milder, more steroid sensitive and carries a better prognosis[5]. Treatment for PE has relied heavily on the use of systemic steroids, with adjuvant steroid sparing drugs such as dapsone, azathioprine, cyclophosphamide and methotrexate. In our patients, oral prednisolone at 0.5mg/kg/day was

successful in controlling the initial eruption. Unfortunately, we are unable to monitor her long term disease progress. This patient is an excellent example of how the great mimicker (LE) is mimicked (PE).

References

1. Amagai M. Pemphigus. In: Dermatology (Bolognia JL, Jorizzo J, Rapini RP, eds). London: Mosby, 2003; 449-62
2. Senear FE, Usher B. An unusual type of pemphigus: coming features of lupus erythematosus. Arch Dermatol Syphilol 1926; 13: 761
3. Chorzelski J, Jacblonska S, Blaszczyk M. Immunopathological investigations in the Senear-Usher syndrome (coexistence of pemphigus and lupus erythematosus). Br J Dermatol 1968; 80: 211)
4. Mohsin Malik, A. Razzaque Ahmed. Concurrence of Systemic Lupus Erythematosus and Pemphigus: Coincidence or Correlation? Dermatology 214:231-239, 2007
5. Ahmed AR, Salm M. Juvenile pemphigus. J Am Acad Dermatol 1983; 8: 799-807

CONTACT DERMATITIS & OCCUPATIONAL DERMATOSES - Original Article

Clinical pattern and causative allergens of hand and / or feet eczema identified from patch test - a retrospective 5 year study

Priya G, *MRCP*, Asmah J, *MMED*, Gangaram HB, *FRCP*, Suraiya HH, *FRCP*

Abstract

Background Hand and/or feet eczema may be due to contact dermatitis, either irritant or allergic in nature. Difficulties often arise in distinguishing purely endogenous eczema from the possibility of contact dermatitis clinically. Patch test is carried out to detect the presence of allergic contact dermatitis. This is important for optimum patient care and to obtain a favourable outcome.

Objectives To identify the demography, clinical characteristics and causative allergens of hand and/or feet eczema among patients from the patch test clinic.

Methods Patients who attended the patch test clinic in the Department of Dermatology, Hospital Kuala Lumpur from 2003 to 2007 were evaluated retrospectively. All of them were having hand and/or feet eczema. Data were collected for their demography, sites affected and patch test findings.

Results 379 patients were included in the study. The age of patients ranged from 6 years to 78 years with an average of 36.7 years. Their occupations ranged from blue collar (20.3%) and white collar (38.3%) workers, housewives (9.5%), pensioners (7.1%) and students (20.3%). Clinical presentations included isolated hand eczema (34.6%), isolated feet eczema (21.9%), hand and feet eczema (19.0%), and hand and/or feet with eczema with involvement of other parts of the body (24.5%).The mean duration of eczema was 3.8 years. The rate of positive patch test was 58.0 %(n = 220/379).Clinically relevant allergens were identified in 123 (32.5%) patients only. Fifty two percent of the clinically relevant allergens were identified from the European Standard Series patch test, 9.0% from the Specific Series patch test and 39.0 % from the patients' own personal products that were tested. The most common source was metal items containing nickel (33.3%), followed by toiletries (14.6%) and detergents (10.6%). Other sources include fragrance, cosmetics, rubber, medicaments and hair dye. In 256 (67.5%) patients, there were no underlying causes detected, and they were managed as endogenous hand and feet eczema. There is a possibility that the causative allergen was not suspected/tested and hence not detected.

Conclusion Hand and/or feet eczema can affect any age group and patch testing forms a very important diagnostic tool in the management.

Keywords *Clinical pattern, allergens, hand and/or feet eczema, patch test*

Introduction

The hand and feet are often exposed to various potential allergens and irritants. Exclusive hand eczema affects 9.1% of patients and another 9.0% have exclusive feet eczema. Difficulties often arise in distinguishing endogenous eczema from the probability of contact dermatitis clinically and patch testing is of much help in these situations[1,2].

The aim of this study is to determine the demography, clinical pattern and causative allergens identified from patch test in hand and / or feet eczema.

Correspondence
Priya Gill, *MRCP (UK)*
Department of Dermatology, Hospital Kuala Lumpur
Wilayah Persekutuan, Malaysia
E-mail : drpriya74@yahoo.com

Materials and methods

This is a retrospective study on patients from the Patch Test clinic in the Department of Dermatology, Hospital Kuala Lumpur. All patients with hand and/or feet eczema who underwent Patch Test from January 2003 to December 2007 were included. Patch Test was carried out according to the recommendations made by the International Contact Dermatitis Research Group using Trolab® allergens. All patients were patch tested against the European Standard Series allergens and if necessary, with an additional specific series e.g. hairdressing chemicals, shoe allergens, rubber chemicals etc and their own products. The readings were done on day 3 and day 5.

Data was analyzed using SPSS. The patients were assessed in terms of demographic data, clinical presentations, patch test results, clinically relevant patch test results and source of allergens.

Results

A total of 379 patients were included in this review (259 female,120 male). Two hundred and twenty patients had a positive patch test (164 female, 56 male). The racial distribution of patients in each category mimicked the racial attendance of the Outpatient specialist clinic, Department of Dermatology, Hospital Kuala Lumpur in 2007 with 58.6% Malays, 21.9% Chinese, 17.4% Indian and 2.1% of patients belonging to other races. They were categorized into patients with only hand eczema or only feet eczema, hand and feet eczema and hand and / or feet eczema with generalization. The demography of patients is shown in Table 1. There is a female preponderance and the mean age groups were in their thirties for all categories. The age distribution is shown in Figure 1.

Table 2 details the patients' occupations. They were classified into white collar workers (e.g. supervisors, clerks, nurses, professionals, teachers, secretaries, executives, film producers etc), blue collar workers (soldiers, labourers, machine operators, welders, attendants, taxi drivers, baby sitters, cleaners etc), housewives, pensioners and students.The white collar patient subgroup presented mainly with hand and feet eczema while those who held blue collar jobs had mainly feet eczema. Interestingly, the many of the students had feet eczema.

The working diagnosis for all the patients is shown in Table 3. Patch Test was undertaken only when a contact element was suspected.

Two hundred and twenty patients had a positive Patch Test (58%). The positive Patch Test was clinically relevant in only 123 patients (32.5 %) (Table 4). Fifty two percent of the relevant allergens were identified from the European Standard Series, 39% from the patients' personal products and 9% from the Specific Series.

Table 1 Patient demographics

Type	Hand Eczema	Feet Eczema	Hand & Feet Eczema	Hand and/or Feet Eczema with generalization
Number	131 (34.6%)	83 (21.9%)	72 (19.0%)	93 (24.5%)
Male: Female ratio	1: 2.4 (38 male, 93 female)	1:1.4 (35 male, 48 female)	1:2.0 (24 male, 48 female)	1:3.0 (23 male, 70 female)
Age (years)				
Range	7 - 72	6 - 72	7 - 66	7 -78
Mean	34.4	34.4	36.0	38.0

The top 10 allergens detected from the European Standard Series were nickel, fragrance mix, balsam of Peru, paraben, chromium, neomycin, colophony, cobalt, thiuram mix and formaldehyde which were clinically relevant in 41.3%, 31.0%, 42.9%, 35.0%, 41.2%, 31.3%, 33.3%, 28.6%, 50.0% and 42.9% respectively. (Table 5). Only the top 20 allergens identified from the European Standard Series and their clinical relevance are shown in Table 5.

Fifty four patients were tested with rubber chemicals; clinically relevant in 4 patients (9%) (Table 6). A total of 26 patients were tested with shoe allergens; clinically relevant in 2 patients [n = 2/26, (8%)]. Twenty nine patients were tested with the textile and leather dyes allergens; relevant in only 1 patient [n = 1/29, (3%)]. Only 4 patients were tested with hairdressing allergens and two of them were clinically relevant. [n= 2/4, (50%)].

Figure 1 Age distribution

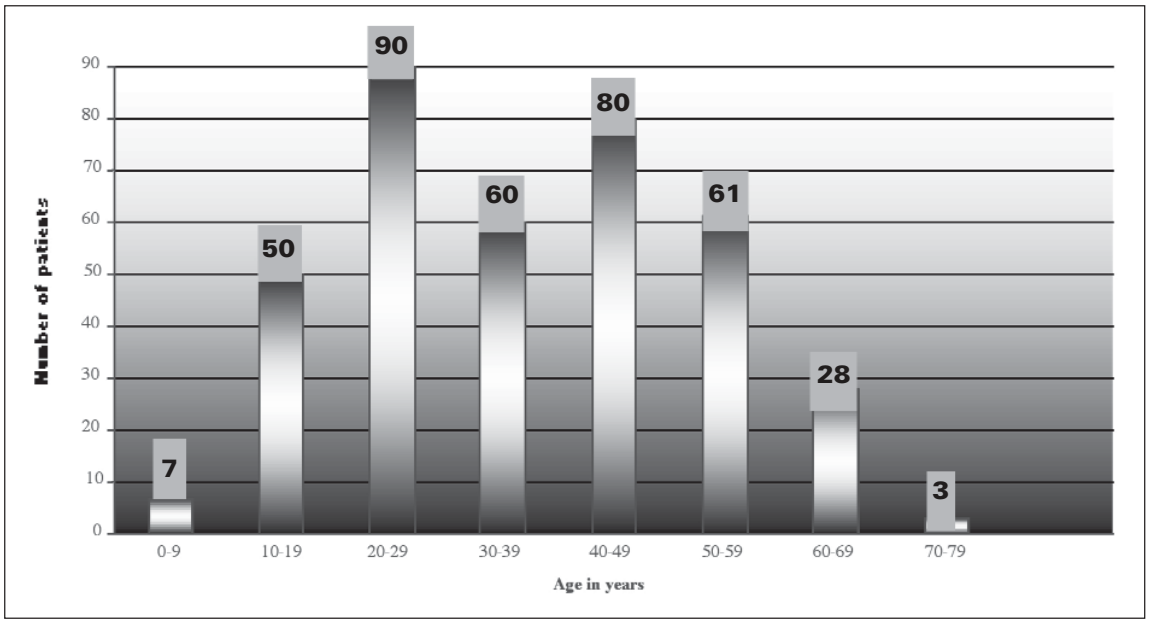


Table 2 Occupation

Occupational field	N=379	Hand Eczema N = 131	Feet Eczema N=83	Hand & Feet Eczema N = 72	Hand & / or Feet Eczema with eczema elsewhere N=93
White Collar	145 (38.3%)	57 (43.5%)	18 (21.7%)	32 (44.4%)	38 (40.9%)
Blue Collar	77 (20.3%)	27 (20.6%)	21 (25.3%)	9 (12.5%)	20 (21.5%)
Housewife	36 (9.5%)	9 (6.9%)	7 (8.4%)	8 (11.1%)	12 (12.9%)
Pensioner	27 (7.1%)	13 (9.9%)	5 (6.0%)	5 (7.0%)	4 (4.3%)
Student	77 (20.3%)	16 (12.2%)	29 (35.0%)	17 (23.6%)	15 (16.1%)
Not available	17 (4.5%)	9 (6.9%)	3 (3.6%)	1 (1.4%)	4 (4.3%)

Table 3 Working diagnosis and Patch test results

Diagnosis	All patients N=379	Relevant Allergen identified N=123
Contact Dermatitis	163 (43.0%)	72 (58.6%)
Hand Eczema	75 (19.8%)	16 (13.0%)
Feet Eczema	62 (16.3%)	13 (10.6%)
Hand & Feet Eczema	59 (15.6%)	18 (14.6%)
Discoid Eczema	6 (1.6%)	0 (0%)
Atopic Eczema	8 (2.1%)	2 (1.6%)
Photodermatitis	5 (1.3%)	2 (1.6%)
Juvenile Plantar Dermatitis	1 (0.3%)	0 (0%)

Table 4 The top 20 allergens from the European Standard Series

Allergens	+ve PT (%)	Clinically Relevant (%)
Nickel	104 (47.3)	43 (41.3)
Fragrance mix	42 (19.1)	13 (31.0)
Balsam of Peru	21 (9.5)	9 (42.9)
Paraben	20 (9.1)	7 (35.0)
Chromium	17 (7.7)	7 (41.2)
Neomycin	16 (7.3)	5 (31.3)
Colophony	15 (6.8)	5 (33.3)
Cobalt	14 (6.4)	4 (28.6)
Thiuram Mix	14 (6.4)	7 (50.0)
Formaldehyde	14 (6.4)	6 (42.9)
Isothiazolin	10 (4.5)	3 (30.0)
Flavin	8 (3.6)	1 (12.5)
Wool Alcohol	7 (3.2)	4 (57.1)
Mercaptobenzothiazole	5 (2.3)	3 (60.0)
Mercapto Mix	4 (1.8)	4 (100.0)
Sesquiterpene Lactone Mix	3 (1.4)	3 (100.0)
Paratertiary Phenol Formaldehyde Resin (PPFR)	1 (0.5)	1 (100.0)
N-Isopropyl-N2-phenyl paraphenylenediamine (PPD)	1 (0.5)	1 (100.0)
Hydroxymethylpentylhexenecarboxyaldehyde	2 (0.9)	0
Benzocaine	3 (1.4)	0

Table 5 Patch Test result for rubber allergens

Rubber Chemicals, n = 54, clinically relevant in 4 patients		
Allergens	**Frequency**	**%**
Hexamethylenetetramine	1	25
Diphenylthiourea	2	50
Dibutylthiourea	1	50
1, 3 - Diphenylguanidine	1	25
Bis (diethyldithiocarbamato) Zinc	1	25
N, N - Diphenyl Paraphenylenediamine	2	50
Bis (dibuhyldithiocarbamato)Zinc	1	25
Cyclohexyl Thiophtalimide	2	50
4, 4'- Dihydroxybiphenly	1	25
Zinc dibenzyldithiocarbamate	1	25

Table 6 Patch Test result for Hand eczema

Allergens	+ve PT (%) (N = 82)	Clinically relevant (%)	Source
Nickel	32 (39.0)	10 (31.3)	Coins, Costume jewellery, Watch Strap, Belt buckle, pins etc
Fragrance mix	20 (24.4)	4 (20.0)	Perfume & After shave Perfume & After shave / Cosmetics
Balsam of Peru	9 (11.0)	2 (22.2)	Cosmetics
Paraben	8 (9.8)	2 (25.0)	Rubber Gloves
Thiuram Mix	7 (8.5)	3 (42.9)	-
Cobalt	7 (8.5)	0	Medicament
Colophony	6 (7.3)	1 (16.7)	Cosmetics
Formaldehyde	6 (7.3)	2 (33.3)	Cosmetics /Detergents
Isothiazolin	6 (7.3)	2 (33.3)	Medicament
Neomycin	4 (4.9)	2 (50.0)	-
Chromium	4 (4.9)	0	Cosmetics
Wool Alcohol	4 (4.9)	3 (75.0)	-
Benzocaine	3 (3.7)	0	-
Flavin	3 (3.7)	0	Cosmetics
Sesquiterpene Lactone Mix	2 (2.4)	1 (50.0)	Rubber Gloves
Special Series: Rubber Chemicals	4 (4.9)	3 (75.0)	Hair Dye
Special Series : Hairdressing	2 (2.4)	2 (100)	

Table 7 Patch Test result for Feet eczema

Allergens	+ve PT (N = 39) (%)	Clinically relevant (%)	Source
Nickel	18 (46.1)	8 (44.4)	Costume jewellery, Shoe buckle, Footwear
Neomycin	3 (7.7)	1 (33.3)	Medicaments
Colophony	3 (7.7)	1 (33.3)	Footwear
Thiuram Mix	3 (7.7)	1 (33.3)	Footwear
Paraben	2 (5.1)	1 (50.0)	Cosmetics
Chromium	2 (5.1)	2 (100)	Footwear
Isothiazolin	2 (5.1)	1 (50)	Cosmetics
Fragrance mix	2 (5.1)	1 (50)	Perfume & AfterShave
Balsam of Peru	2	0	-
Cobalt	2	0	-
Flavin	2	0	-
N-Isopropyl-N2-phenyl paraphenylenediamine (PPD)	1	0	-
Paratertiary Phenol Formaldehyde Resin (PPFR)	1	0	-
Wool Alcohol	1	0	-
Special Series: Textile and Leather Dyes	2 (5.1)	2 (100)	Footwear
Special Series: Shoe Allergens	2 (5.1)	2 (100)	Footwear

On further analysis looking at the source of clinically relevant allergens, the most common source of nickel were coins, costume jewellery, watch straps, belt buckles, pins, zippers and shoe buckles. Balsam of Peru and fragrance mix were mainly found in perfumes, after shave lotions and cosmetics. (Tables 7, 8, 9, 10)

In this study, nickel was the main source of allergens (33%), followed by toiletries (15%), detergents (11%), rubber (8%), 6% each from cosmetics, fragrance and hair dye and finally 5% each from medicaments, footwear and miscellaneous. (Figure 2)

Discussion

In centres with patch test clinics the overall rate of positive patch tests was 40-50%[3]. These were clinically relevant in 16 - 40 % of total patch tests done. Forty to eighty five percent of those with positive patch tests were clinically relevant. This study has similar results with a positive patch test rate of 58% (n = 220 / 379).The clinical relevance was found in 56% of patients and 32.5% of all patch tests carried out.

Table 8 Patch Test result for Hand & Feet eczema

Allergens	+ve PT (%) (N = 40)	Clinically relevant (%)	Source
Nickel	17 (42.5)	6 (35.3)	Coins, Costume jewellery, Zipper, Watch Strap, Belt & shoe buckle etc
Fragrance mix	8 (20.0)	2 (25.0)	Fragrance/Cosmetics
Balsam of Peru	6 (15.0)	4 (66.7)	Toiletries/Cosmetics/ Perfume & After shave
Paraben	6 (15.0)	4 (66.7)	Cosmetics
Chromium	5 (12.5)	2 (40.0)	Cement/Industrial oils
Isothiazolin	2 (5.0)	1 (50.0)	Cosmetics
Formaldehyde	2 (5.0)	2 (100)	Cosmetics
Cobalt	2 (5.0)	2 (100)	Personal Items/Industrial oils
Mercaptobenzothiazole	2 (5.0)	2 (100)	Rubber Gloves
Mercapto Mix	2 (5.0)	2 (100)	Rubber Gloves
Flavin	2 (5.0)	0	-
Colophony	2 (5.0)	0	-
Wool Alcohol	1 (2.5)	0	-
Neomycin	1 (2.5)	0	-
Benzocaine	1 (2.5)	0	-
Special series: Rubber Chemicals	1 (2.5)	1 (100)	Rubber Gloves
Special Series: Shoe Allergens	1 (2.5)	1 (100)	Footwear

Figure 2 Sources of clinically relevant positive Patch test in Hand & / or Feet Eczema

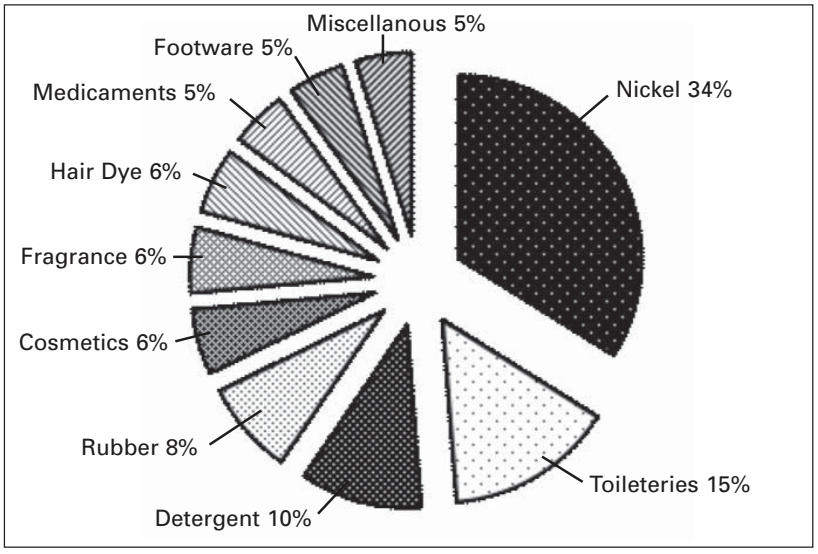


Table 9 Patch Test result for Hand & / or Feet Eczema with generalization

Allergens	+ve PT (%) (N = 59)	Clinically relevant (%)	Source
Nickel	37 (51.4)	19 (51.4)	Coins, Costume jewellery, Zipper, Watch Strap, Belt & shoe buckle etc
Fragrance mix	12 (20.3)	1 (8.3)	Perfume & After shave
Neomycin	8 (13.6)	3 (38.0)	Medicaments
Chromium	7 (11.9)	2 (28.6)	Dental Material/ Leather Strap
Formaldehyde	6 (10.2)	0	-
Paraben	4 (6.8)	2 (50.0)	Medicaments
Colophony	4 (6.8)	0	-
Balsam of Peru	4 (6.8)	0	-
Thiuram Mix	4 (6.8)	2 (50.0)	Gloves
Mercaptobenzothiazole	3 (5.1)	1 (33.3)	Leather Strap
Cobalt	3 (5.1)	0	Dental Material
Hydroxymethylpentylhexenecarboxyal-dehyde	2 (3.4)	1 (50.0)	Perfume & After shave
Mercapto Mix	2 (3.4)	1 (50.0)	Leather Strap
Sesquiterpene Lactone Mix	1 (1.7)	1 (100)	Flower
Flavin	1 (1.7)	0	-
Wool Alcohol	1 (1.7)	0	-
Special Series: Metal Compounds	1 (1.7)	1 (100)	Factory machinery

The predominance of young women corresponds to the cohort of Meding et al[6]. In the South India study looking at lower leg and feet eczema, the average patient age was 40.49 years with a female to male ratio of 1.6:1[4]. However there are studies that with a male preponderance. Smith et al reported a male to female ratio of 1.25:1 in their study on descriptive epidemiology of hand eczema[7]. Goh CL from Singapore observed a male preponderance as well in the prevalence of hand eczema in his cohort of patients. (Male 56.0%, Female 44.0%)[8]. The difference between studies could be explained by different jobs being carried out by men and women and their tolerance to develop hand and / or feet irritant contact dermatitis with different types of exposure to allergens / irritants.

The working diagnosis for our patients was contact dermatitis (43.0%), hand eczema (19.8%), feet eczema (16.3%), hand and feet eczema (15.6%) with about 5% of patients having a diagnosis of discoid eczema/ atopic eczema/photodermatitis/ juvenile dermatitis. As these patients were identified from patch test, those with one type of endogenous eczema do not have to undergo this procedure unless suspected to have contact dermatitis. Therefore, those with hand and feet eczema are more likely to undergo patch test.

Chougule et al reported that among patients with lower leg and foot eczema in South India, the most common working diagnosis was lichen simplex chronicus (36%), followed by discoid eczema (18.5%), stasis eczema (7.5%), juvenile plantar dermatoses (5%), hyperkeratotic eczema (3%) and unclassified endogenous eczema (3%)[4]. The North American Contact Dermatitis Group (NACDG) looked at 6953 patients with hand eczema only (1994-2004) and their common working diagnosis were allergic contact dermatitis (27.8%), irritant contact dermatitis (19.7%), psoriasis (3.3%), atopic eczema (2.5%), pompholyx (1.7%) and other dermatitis/dermatoses (8.9%)[5]. In this paper, however, the authors were not specifically looking for contact dermatitis but rather at the range of conditions that presented with hand eczema. Different centres will have different working diagnosis prevalence as a lot of factors will influence their decision to carry out patch test.

Majority of patients were in the white collar group (38.3%), followed by the blue collar group and students (20.3% each), housewives (9.5%) and finally pensioners (7.1%). Our results are different compared to studies by Cherry et al and Smith et al. Cherry concluded that contact dermatitis is by far the most commonly reported occupational skin disease, especially in the blue collar group. Their study showed that women were most likely to have dermatitis attributed to wet work, and men to oils and related substances. In Smith's paper, the most frequent types of employment associated with occupational hand eczema were caterers, metal workers, mechanics, builders and printers (blue collar workers)[7,9]. Unfortunately, as this is a retrospective review, we are unable to ascertain for sure the relationship of eczema in our group of patients to the occupation. The majority of patients in our review were from white collar occupations. This can be attributed to our patient population that underwent patch testing in general being from the white collar group as this department is located in the heart of Kuala Lumpur, the biggest city in Malaysia. Smith et al also suggested that low office humidity as an irritant factor and the recommendation of office work as a "clean, safe job" might need to be reviewed[7].

The most common presentation for white collar workers, blue collar workers and pensioners was hand eczema. Feet eczema was the most common subtype among students and not surprisingly, housewives presented most commonly with hand and/ or feet eczema with eczema elsewhere. This could be due to the possible contact to various potential irritants in everyday household chores like washing clothes and utensils with detergents and water. Many housewives as well squat on the ground while doing their chores; and their feet are continuously exposed to detergents and water while other areas are affected by splashing of the water.

The highest proportion of clinically relevant allergens were identified in patients who were being managed as contact dermatitis (44.2%), compared to hand eczema (21.3%), feet eczema (21.0%), hand & feet eczema (30.5%) and atopic dermatitis (25.0%).This could be due to a lower index of suspicion for a contact element in those diagnosed to have endogenous eczema. However, studies have shown that endogenous eczema is commonly complicated by a contact element and eliminating the causative factor will greatly facilitate the management[10,11].

The most common positive patch test allergens in this study were nickel (47.3%), fragrance mix (19.1%) and balsam of Peru (7.9%). However, for those with feet eczema, the most common allergens were nickel, chromium, shoe allergens and textile and leather dyes. The results in our centre differed slightly compared to Osmania General Hospital, Hyderabad, India where a similar study was done using the Indian Standard Series Patch test allergens. Their most common allergens were nickel and chromium (25% each), followed by fragrance mix (21.4%) and wool alcohol (14.3%).These results were probably due to the different series being used for Patch test and the different patient factors, commercial products and environmental factors the two populations are exposed to[2].

The most common sources of allergens were personal items containing nickel (33%), toiletries (15%) and detergents (15%). Our findings are similiar that of the North American Contact Dermatitis Group. They found the most common sources to be soaps, cleaners, detergents, solvents, oil and lubricants[5]. These are common everyday items individuals are exposed to.

There are several limitations to this study. The data is retrospective with some even incomplete information; therefore a more definite causal relationship cannot be determined. The study

sample was drawn only from patients with hand and/or feet eczema who were patch tested; as such, they are not representative of the general population or the general dermatology population. More research in a clinical setting is needed to address some of the issues raised in our review, especially prospective research.

Conclusions

Hand and/ or feet eczema affects all ages and different types of occupations. This disease can cause significant disability and economic loss to both individuals and society. Patch testing is a very important diagnostic tool to exclude allergic contact dermatitis in those suspected as having endogenous eczema or other types of dermatoses. The specific series patch test allergens and the patient's own products are of added benefit if clinically indicated. Contact avoidance remains the most important measure in the prevention and management of allergic and irritant contact dermatitis. Therefore, education about avoidance of, or protection from, the most common antigens is critical, especially in high risk occupations

References

1. Ng SK, Goh CL, M Shahidullah. Clinical patterns of Feet Eczema in Singapore. Bulletin for medical practitioners 1998; 9 (1)
2. G Vani , Rao GA, V Gowri TSS Lakshmi. Allergic contact dermatitis of the hands and/or feet - common sensitizers. Contact Dermatitis 2005; 52 (1):50-52
3. Ormond P, Hazelwood E et al. The importance of a dedicated patch test clinic. Br J Derm 2002; 146: 304-307
4. Chougule A, Thappa DM. Patterns of lower leg and foot eczema in south India. Indian J Dermatol Venereol Leprol 2008; 74:458-61
5. Warshaw ME, Ahmed AL, Belsito DV et al. Contact dermatitis of the hands: Cross-sectional analyses of North American Contact Dermatitis Group Data,1994-2004; J Am Acad Dermatol 2007; 57; 301-14)
6. Veien NK, Hattel T, Laurberg G. Hand eczema: causes, course, and prognosis I. Contact Dermatitis 2008; 58; 330-334
7. Smith HR, Armstrong DKB, WakelinSH et al. Descriptive epidemiology of hand dermatitis at the St John's contact dermatitis clinic. Br J Dermatol 2000; 142: 284-287
8. Goh CL. An epidemiological comparison between occupational and non-occupational hand eczema. Br J Dermatol 1989; 120: 77-82
9. Cherry N, Meyer JD, Adidesh A et al. Surveillance of occupational skin disease: EPIDERM and OPRA. Br J Derm 2000; 142:1128-1134
10. Shackelford KE, Belsito DV. The etiology of allergic-appearing foot dermatitis: a 5-year retrospective study. J Am Acad Dermatol 2002; 47 (5): 715-721
11. Fowler JF, Ghosh A, Sung J et al. Impact of chronic hand dermatitis on quality of life, work productivity, activity impairment and medical costs. J Am Acad Dermatol 2006; 54 (30): 448-457

GRANULOMATOUS DISEASE - Case Report

Cutaneous Sarcoidosis Mimicing Tuberculoid Leprosy

Chong YT, *MRCP*, Tey KE, *MRCP*, Choon SE, *FRCP*

Keywords *Uveitis, lymphadenopathy, leprosy, sarcoidosis*

Introduction

Sarcoidosis is a chronic systemic disorder of unknown etiology, characterized histopathatologically by non-caseating, epithelioid granulomatous infiltration in various organs.[1,2] Cutaneous sarcoidosis is also known as a dermatologic masquerader because the lesions can exhibit many different morphologies.[3] We report a patient who was initially diagnosed as having tuberculoid leprosy based on histological findings. He was treated with multi-drug therapy for 18 months without clinical improvement. In addition, he had left panuveitis and mediastinal lymphadenopathy.

Case report

A 28 year old gentleman with no previous medical illness first presented to the ophthalmologist with complaint of acute onset of left eye redness and pain. He was diagnosed and treated for left panuveitis. He was referred to us two months later for further management of non-pruritic erythematous skin rashes of 1 year duration. At the same time, he was referred to the respiratory team for further work up of possible tuberculosis.

The skin eruption first started over the upper limbs and subsequently involved the trunk, sparing the face, palm and soles. There was no associated limb numbness or weakness and no fever, cough or shortness of breath. His appetite was normal and there was no significant weight loss. Review of other systems was unremarkable.

Clinically, he was pink and not jaundiced. His height was 108 cm and weight 102 kg. There were multiple well-defined erythematous papules and plaques distributed symmetrically over the trunk and upper limbs. Sensation was normal and there was no peripheral nerve thickening. Lymph nodes were not palpable. Examinations of the cardio-respiratory system and abdomen did not review any abnormalities.

Blood investigations revealed hypochromic microcytic anaemia with a hemoglobin of 13.1gm/dl and eosinophilia. His erythrocyte sedimentation rate (ESR) was 30mm per hour. He has hyperglobulinaemia (51 g/L) and slightly raised alanine aminotransferase (40 IU/L). The renal profile, serum calcium and fasting blood glucose were normal. Serological screen for viral hepatitis, rapid reagent test for syphilis (RPR) and anti-HIV were negative. Mantoux test was negative (2mm). The slit skin smear was negative for acid fast bacilli.

The skin biopsy was reported as tuberculoid leprosy which showed numerous epitheliod granuloma around neurovascular bundles with occasional Langerhan's giant cells, many epitheliod cells and lymphocytes. There was no caseation necrosis and the Fite stain was negative for acid fact bacilli. Tissue culture for mycobacteria and fungus was also negative.

Based on the histology report, multi-drug therapy (MDT) for paucibacillary leprosy (rifampicin, clofazimine and dapsone) was commenced. At the same time, he was treated with prednisolone for panuveitis by the ophthalmologist. His skin lesions improved initially. However, two months into treatment, he developed raised liver enzymes. Ultrasound of the liver showed fatty changes. The MDT was withheld for one month and later restarted with minocycline and dapsone. After one year of treatment, his skin lesions did not show much improvement despite the initial response. A repeat skin biopsy showed similar findings as the initial one.

Further investigations with CT scan and endoscopic ultrasound showed multiple small mediastinal lymph nodes. Fine-needle aspiration cytology of the sub-carinal lymph node was reported as a granulomatous lesion. PCR test from a repeat skin biopsy was negative for Mycobacteria DNA.

Correspondence
Chong Yew Thong
Department of Dermatology
Hospital Sultanah Aminah
80100 Johor Bahru, Johor
E-mail : cytusm@yahoo.com

He was diagnosed as having cutaneous sarcoidosis and referred back to the respiratory physician for further management. He was treated empirically with anti-tuberculous drugs (ethambuthol, isoniazid, rifampicin and pyrazinamide) for another two months with no response. At the same time, high dose prednisolone was restarted by the ophthalmologist for a flare-up of his left panuveitis. His skin lesions resolved after one month of treatment.

Discussion

This gentleman presented with a diagnostic challenge. He had erythematous papules and plaques over the upper limbs and trunk of one year duration. The cutaneous eruption preceded the development of uveitis. Based on cutaneous findings, various differential diagnosis were entertained including cutaneous atypical mycobacteria infection, fungal infection, leprosy and cutaneous lymphoma.

He was treated for tuberculoid leprosy based on histological findings. The initial response was most likely due to the steroids prescribed for uveitis. However, the skin lesions relapsed when the steroid was tapered and was subsequently stopped.

Sarcoidosis is a chronic systemic disorder of unknown etiology, characterized histopathatologically by non-caseating, epithelioid granulomatous formation.[1,2] It can affect various organs, with lungs, lymph nodes, skin and eyes being more commonly affected.[4]

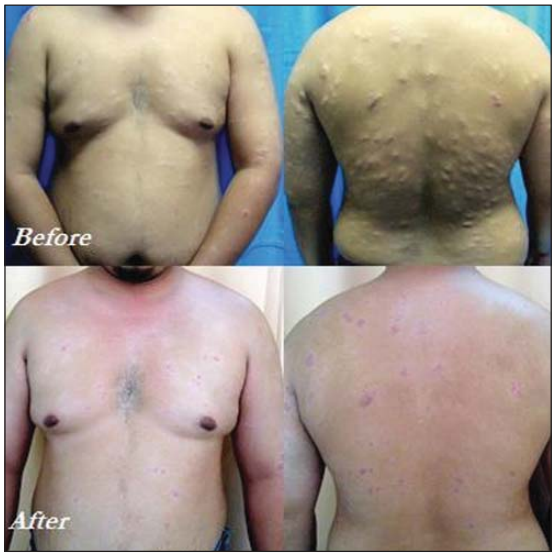


Figure 1-4 Multiple symmetrical erythematous papules and plaques seen on the upper limbs and trunk before treatment with steroid (Figure 1 & 2) and after treatment with steroid (Figure 3 & 4)

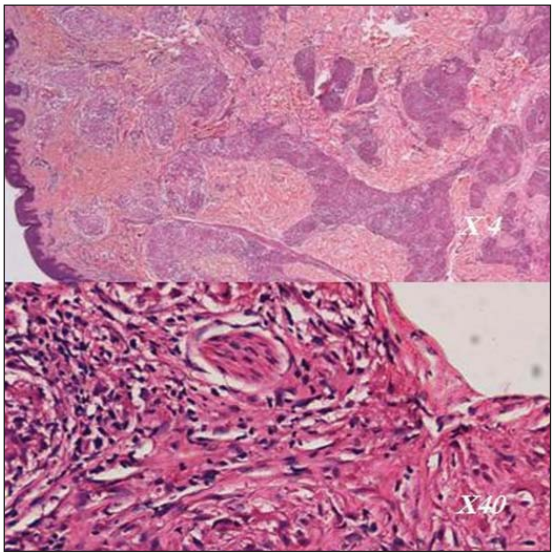


Figure 5&6 Multiple well-formed non-caseating granulomas seen in the dermis (Figure 5) which consist of numerous epitheliod cells and lymphocytes around neurovascular bundles with occasional Langerhan's giant cells (Figure 6)

Sarcoidosis affects all races, both sexes and all ages. Incidence reported ranged from 64/100, 000 in Sweden to 1.4/100, 000 in Japan.[1]

Cutaneous involvement occurs in 20 to 35 percent of patients with systemic sarcoidosis but it may occur without systemic involvement.[3] It is most commonly seen at the onset of the disease process.[1] Various morphologies have been described, including papules, follicular papules, plaques, nodules, ulcerative lesions and alopecia.[1]

The eye and adnexa are involved in 25 to 80% of patients with sarcoidosis and anterior uveitis is the most common manifestation, occurring up to 65% of patients with ophthalmologic involvement. In about 10 to 15%, both the anterior and posterior (panuveitis) segments may be involved.[11]

In developed countries, sarcoidosis was reported to be the second most common non-infectious cause of uveitis, after sero-negative spondyloarthropathy, especially in US, Netherland and Japan.[10]

There is no single test to confirm the diagnosis of sarcoidosis.[3] Sarcoidal granulomas have no unique histologic features to differentiate them from other granulomas. Patients are diagnosed with sarcoidosis on the basis of compatible clinical, radiologic findings, supported by histologic evidence of non-caseating granulomas, and when other potential causes, such as infections, are excluded.[11]

Cutaneous sarcoidosis has quite often been misdiagnosed as leprosy because of near similar skin lesions and histologic findings of non-caseating granuloma. Diagnosis of sarcoidosis was suspected when the patient did not respond to a course of anti-leprosy drugs.[6,7,8,9]

Cutaneous sarcoidosis is rare in South-East Asia, including Malaysia. Liam et al 12 reported 14 cases of sarcoidosis from a single centre in eighteen years (1972 to 1990), out of which, twelve patients had pulmonary involvement and five developed erythema nodosum. A case series of 25 patients with cutaneous sarcoidosis in twenty three years (1980 to 2003) was also reported in Singapore by Chong et al.[5] 10 out of 25 patients had extra cutaneous manifestation.

As this condition is rare, the clinical diagnosis of cutaneous sarcoidosis is often not suspected and is made on subsequent biopsy excluding other causes, in particular tuberculoid leprosy and other mycobacterial infections which are more prevalent.[5]

In summary, our patient presented with erythematous skin eruption associated with uveitis and bilateral hilar lymphadenopathy. His skin biopsy revealed a non-caseating granulomatous reaction. He was treated with anti-leprosy and anti-tuberculous drugs without clinical improvement. Thus, he was diagnosed as having sarcoidosis after the infectious causes were ruled out. Cutaneous sarcoidosis is rare and a high index of suspicion with clinical correlation of various features is important to make a correct diagnosis.

References

1. English III JC, Patel PJ, Greer KE. Sarcoidosis. J Am Acad Dermatol 2001; 44:725-743
2. Young RJ III, Gilson RT, Yanase D, Elston DM. Cutaneous sarcoidosis. Int J Dermatol 2001; 40:249-253
3. Katta, R. Cutaenous sarcoidosis: A dermatologic masquerader. Am Fam Physician 2002; 65:1581-1584
4. Mahajan VK, Sharma NL, Sharma RC, Sharma VC. Cutaneous sarcoidosis: Clinical profile of 23 Indian patients. Indian J Dermatol Venereol Leprol 2007; 73:16-21
5. Chong WS, Tan HH, Tan SH. Cutaneous sarcoidosis in Asians: a report of 25 patients from Singapore. Clin Exp Dermatol 2005; 30:120-124
6. Singh K, Raina V, Narulla AK, Singh R. Sarcoidosis masquerading as leprosy, pulmonary tuberculosis and urolothiasis. J Assoc Physicians India 1990; 38(9):657-9 [Abstract]
7. Ramanujam K. Tuberculoid leprosy or sarcoidosis? A diagnostic dilemma. Lepr India 1982; 54(2):318-23 [Abstract]
8. Kaur S, Dhar S, Bambery P, Kanwar AJ, Khajuria A. Cutaneous sarcoidosis masquerading as relapsed borderline tuberculoid leprosy? Int J Lepr Mycobact Dis 1993; 61(3):455-458 [Abstract]
9. Thaipisuttikul Y, Kateruttanakul, P. Sarcoidosis mimics lepromatous leprosy: a case report. J Med Assoc Thai 2007; 90:171-174
10. Rathinam SR, Namperumalsamy P. Global variation and pattern changes in epidemiology of uveitis. Indian J Ophthalmol 2007; 55:173-183
11. Lannuzi, MC, Rybicki, BA, Teirstein AS. Sarcoidosis. N Engl J Med 2007; 357(21):2153-2165
12. Liam CK, Menon A. Sarcoidosis: A review of cases seen at the University Hospital, Kuala Lumpur. Sing Med J 1993; 34:153-156

ANNOUNCEMENT - Administrative Update

Monitoring doctors' performance in skin biopsy using CUSUM technique

Chang CC, *AdvMDerm (UKM)*, Azizan NZ, *AdvMDerm (UKM)*, Roshidah B, *FRCP*

Assessment of doctors' performance is an integral part of quality improvement in health care. Traditionally, evaluation of competency in performing procedures involves informal peer review and formal credentialing. However, these methods are often subjective and are without explicit reference to universally accepted standards of practice[1,2]. Recently, statistical process control techniques which have long been used in manufacturing industry, have gained popularity in quality improvement in health care[3]. One of the techniques used in the objective monitoring of doctors' performance is the Cumulative Sum (CUSUM) method. First described by E.S. Page in 1954, CUSUM is based on sequential monitoring of a cumulative performance measure over time[2]. A graphical representation of CUSUM in a line chart is designed to detect any early change in performance associated with an unacceptable rate of adverse outcome[2]. Early warning of poor performance ensures patient safety with minimum morbidity, and timely corrective actions can be taken to improve the doctor's performance.

Skin biopsy is an important diagnostic and therapeutic procedure in dermatology. Although it is a minor surgical procedure, its outcome depends greatly on the surgeon's skill and competency. A good outcome of a skin biopsy can be defined by the absence of post-biopsy wound infection, presence of cosmetically acceptable scar, and the tissue sample collected being representative as well as adequate for histopathological interpretation. A Skin Biopsy Registry has been established by a group of dermatologists and it has the following objectives:

1) To monitor the performance and competency of doctors in performing skin biopsies
2) To determine factors affecting the outcome of skin biopsies
3) To assess the concordance rates between pre-biopsy clinical diagnosis and histopathological diagnosis from skin biopsy

Individual doctors are required to collect data on the skin biopsies he or she performs. Data on each biopsy is collected prospectively at three separate occasions.

Firstly, patient demographic and clinical data as well as details of biopsy and post-biopsy care are documented immediately after biopsy. Next, during post-biopsy review when patient returns for suture removal, any complications such as wound dehiscence or infection are recorded.

Lastly, during histopathological review, the biopsy tissue sample is evaluated by the pathologist whether it is adequate and representative for histopathological interpretation.

For the purpose of CUSUM charting, two outcome parameters of skin biopsy are measured:

1) Wound infection rate within 14 days post-biopsy
2) Rate of biopsy tissue sample being representative of skin lesion and adequate for histopathological interpretation

As there are no universally accepted standards for these outcome measures in the published literature, the acceptable and unacceptable failure rates can be determined by performing a retrospective audit of available data or a consensus decision of dermatologists[4]. Following implementation of CUSUM charting, these rates may be revised and adjusted based on the average performance of the trainees and dermatologists.

The CUSUM chart is a plot of a cumulative score versus the index number of a series of consecutive procedures. The CUSUM score is determined by the following formula when the outcome of each consecutive procedure is known2:

$$C_n = \max (0, C_{n-1} + X_n - k)$$

Correspondence
Dr Chang Choong Chor, *AdvMDerm*
Department of Dermatology, Hospital Kuala Lumpur
Jalan Pahang, 50586 Kuala Lumpur, Malaysia
E-mail : ccchor@gmail.com

X_n is the outcome measure for the nth procedure.

X_n is 0 for success and 1 for failure.

k is the reference value determined by pre-defined standards of performance in terms of acceptable and unacceptable failure rates.

At the start, CUSUM $C_0 = 0$.

When an individual performs at an acceptable level of performance, the CUSUM curve runs at a horizontal trend. However, when the failure rates reach an unacceptable level, the curve slopes upward and crosses a horizontal line called decision interval, *h*, signaling unacceptable performance. When this occurs, the individual is required to determine and correct the cause of poor performance.

Figure 1 shows an example of typical CUSUM curves of a trainee learning to perform a procedure and a consultant who is an expert on the procedure. The consultant's CUSUM curve is flat, indicating that he has been performing within the specified standards. In contrast, the trainee's CUSUM curve rises initially crossing two horizontal lines (decision intervals). This indicates poor performance requiring close supervision and re-training. Nevertheless, this trainee seems to have acquired the competence after a certain period, as shown by his CUSUM curve which later plateaus to a horizontal trend.

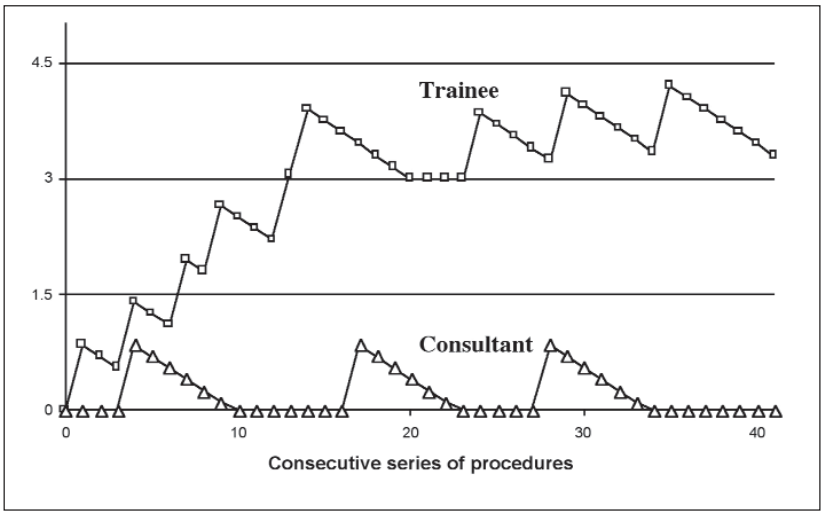
With the financial and technical support received from the Ministry of Health through the Clinical Research Centre, the Skin Biopsy Registry is currently being developed with an online electronic database. This is then linked to the eCUSUM web application (http://app.acrm.org.my/eCUSUM) which will obviate complex manual calculations and tedious charting on paper graphs. Individual doctor can evaluate his or her own performance by logging in to the eCUSUM application to view the CUSUM charts. Supervisors can have access to their trainees' CUSUM charts for periodic monitoring.

A 3-month pilot study is currently being conducted in the Department of Dermatology, Hospital Kuala Lumpur. CUSUM for skin biopsy will be used in the public hospitals in the very near future. It is our fervent hope that CUSUM will encompass other diagnostic and therapeutic procedures in dermatology and will interest dermatologists in private practice as well.

References

1. Williams SM, Parry BR, Schlup MMT. Quality control: an application of the CUSUM. Br Med J 1992; 304: 1359-1361
2. Lim TO, Soraya A, Ding LM, Morad Z. Assessing doctors' competence: application of CUSUM technique in monitoring doctors' performance. Int J Quality Health Care 2002; 14(3):251-258
3. Lim TO. Statistical process control tools for monitoring clinical performance. Int J Quality Health Care 2002; 15(1):1-2
4. Kestin IG. A statistical approach to measuring the competence of anaesthetic trainees at practical procedures. Br J Anaesth 1995; 75:805-809

Figure 1 CUSUM chart for a procedure performed by a trainee and a consultant



ANNOUNCEMENT

Continuous Professional Development

22nd World Congress of Dermatology

Organizers	:	International Leagues of Dermatological Societies (ILDS) with the support of the Korean Dermatological Association (KDA)
Theme	:	Connecting the World Through Innovative Dermatology
Venue	:	Seoul, Korea
Date	:	24 - 29 May 2011
Program	:	website: www.wcd2011.org

Abstract submission deadlines for Free papers: 31 October 2010

Quiz

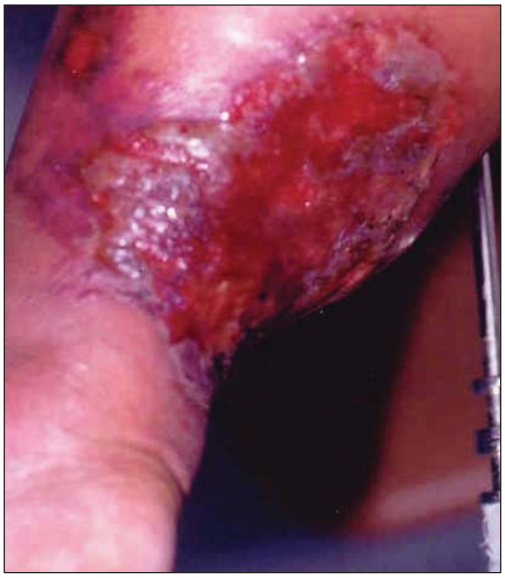
Dermatology Care Plan Quiz for nurses

State the nursing care plan required by each patient.

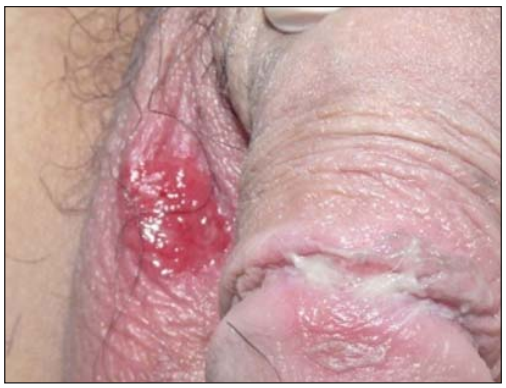
e.g.

Nursing problem	Nursing intervention	Expected Outcome	Comment
Weeping lesion	Wet wrap with astringent solution regularly	Dry scaly lesion	Stop using astringent when lesions are dry

Slide A Patient is admitted for management of erysipelas at his left leg



Slide B Patient has fixed drug eruptions on his genital



Answer is given on page 60

Answers to Clinical Diagnositic Skill Test

Slide A

0	ADR
0	tumour
1	appendageal disorders
1	bacterial infection
0	autoimmune disorders
0	psedolymphoma
0	lupus ertyhematosus
0	lymphoma
0	leprosy
2	rosacea

This patient has roasacea as evidence by the acneiform-like papules in the absence of comedone and the slightly large and reddish nose which can result in rhinophyma. It is important to recognise this condition because of the risk of associate keratitis. Rosacea patient may respond to antbiotic, topical metronidazole gel or retin A cream.

Slide B

0	ADR
1	tumour
0	appendageal disorders
1	bacterial infection
1	autoimmune disorders
1	psedolymphoma
1	lupus ertyhematosus
1	lymphoma
2	leprosy
-3	rosacea

Differential diagnosis of facial plaques include leprosy, lupus erythematosus, granuloma fasciale, polymorphic light reaction, benigh lymphocytoma cutis, lymphoma. By suspecting this condition will patient be investigated fully with regular follow-up.

Slide C

-2	psoriasis
1	keratoderma
1	acanthosis
0	erythroderma
2	scabies
0	lichen planus
-2	contact dermatitis
-2	fungal infection
0	secondary syphilis
-2	keratoderma blenorrhagica

Opaque scaling at finger webs that may extend to palms is a sign of Norwegian scabies. If mistaken for dermatitis, psoriasis or fungal infection will result in prolonged morbidity and spread of scabies to others including healthcare providers.

Slide D

-3	pomphylx
-3	keratoderma
-3	acanthosis
-3	erythroderma
-3	scabies
-3	lichen planus
-3	contact dermatitis
-3	vasculitis
2	bullous disease
-3	scalding

This autoimmune bullous disease is easily mistaken for common chronic inflammatory disease. The round erosions and crusting on these erosions should prompt the diagnosis of bullous disease.

Slide E

0	ADR
0	non-infective inflammation
0	fungal infection
0	tumour
0	dermatitis
1	post-inflammatory hyperpigmentation
0	congenital naevus
0	acnathosis
2	arsenic poisoning
0	mycosis fungoides

The skin lesions on patient's back resemble water droplets on a dirty windscreen. This should prompt the diagnosis of arsenic poisoning. The dyspigmentation represent sun damage skin with formation of pigmented macules and actinic keratoses.

Slide F

-1	dermatitis
-1	non-infective inflammation
1	fungal infection
0	viral infection
0	appendageal disorders
2	pityriasis versicolor
-1	psoriasis
0	herpes viral infection
-1	discoid dermatitis
-1	pityriasis rosacea

Pityriasis versicolor can appear as hyperpigmented macules in fair skin patient whereas in dark skin patient they appear as hypopigmented macules. The round and wrinkled macules which can become confluent but still retain the curved edge prompt the diagnosis of Pityriasis versicolor.

Sum up the number of score 2, 1, 0, -1, -2 and -3 that you have collected.

SCORE	TOTAL NUMBER COLLECTED	IMPLICATION
-3		Delayed diagnosis may result in irreversible outcome / deformity
-2		Delayed diagnosis may result in prolonged morbidity
-1		Therapy for wrong diagnosis may worsen the primary skin lesion
0		No effect on outcome of primary skin lesion but cost wastage of medication, investigation kit and money
1		Therapy for this diagnosis can have good outcome
2		Correct diagnosis enables appropriate investigations, thearpy and even long term follow-up

How did you performed? You should aim for correct diagnosis and minimize delayed diagnosis.

Answers to Dermatology Care Plan Quiz

Slide A Erysipelas

Nursing problem	Nursing intervention	Expected Outcome	Comment
Pain in the leg	analgesics	Improve pain score	Stop analgesic when pain has subside
Swollen leg	Rest & elevate leg	Reduced calf circumference	Do not elevate the leg.
Blistering & weeping lesions	Wrap lesion with diluted KMNO4 solutions Wet	Dry wrinkled scaly skin	Stop wet wraps once swelling and blisters have resolved
Dry scaly skin	Use moisturising soap	Moist non-scaly skin	Stop moisturizing soap when skin normalise

Slide B Fixed drug eruptions on the genitals

Nursing problem	Nursing intervention	Expected Outcome	Comment
Weeping lesion	Wash genitals with astringent solution (normal saline or diluted KMNO4 solutions)	Dry scaly skin	Stop astringent solution once skin dries up
Adverse drug eruptions (ADR)	Educate patient to avoid drug by giving allergy card	Prevent recurrent ADR	Inform patient the possibility of cross reactions with sulphur drugs and black dye if patient has ADR to bactrim