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香 港 醫 訊

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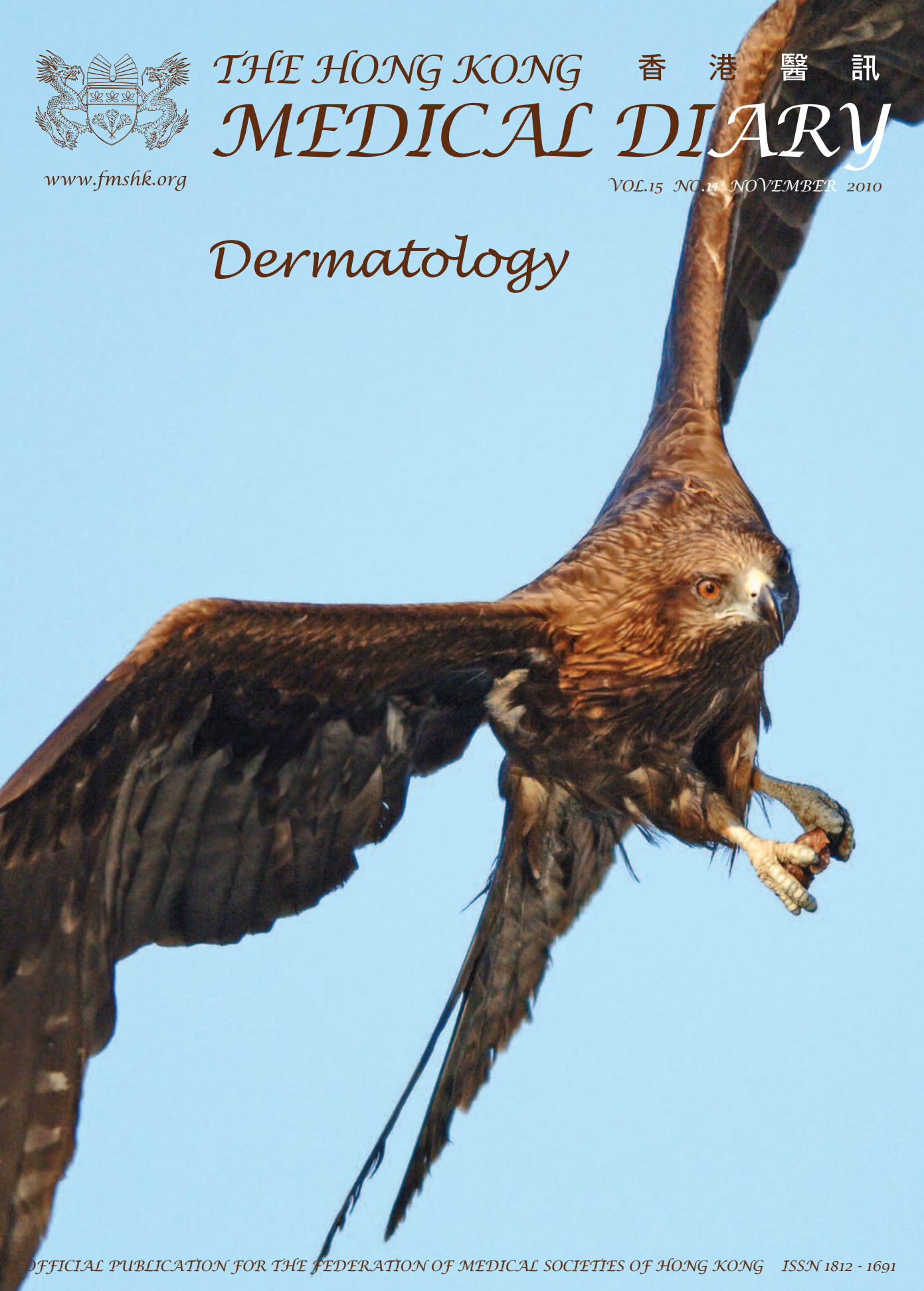
Dermatology



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Contents

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Skin Diseases – More than Skin Deep

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Specialist in Dermatology & Venereology

Editor

Dr. Kingsley HN CHAN

Although the skin is our body's largest organ, skin diseases rarely receive a proportional degree of attention from the general public, and sometimes even by doctors. Skin diseases are often dismissed as superficial afflictions or something that will go away on its own in time. However, skin diseases are far from being just skin-deep and can have widespread and sometimes devastating impact on one's well-being.

While it is true that skin diseases are rarely life threatening, they can nevertheless have a pronounced impact on one's psychological and social well-being. Simply think back to your teenage years when acne on your face can make you extremely self-conscious or even shy away from having social contacts. The link between skin diseases and mental health has been well established: Studies have shown that psychiatric disorders including depression, social phobia and anxiety disorders can develop secondary to acne vulgaris[1-5]. Acne patients also report greater levels of anxiety and depression than patients with serious medical illnesses, such as cancer[5]. By providing acne patients with effective treatment, not only are they physically treated, patients also report significant improvements in self-esteem, affection, obsessive-compulsiveness, shame, embarrassment, body image, social assertiveness and self-confidence[6].

Another reason why dermatological problems deserve more attention than they currently receive is their prevalence - Skin diseases are frequent encounters by family physicians and general practitioners[7-9]. According to a study published in the *2008 Annuals of Family Physician*, skin diseases account for more than one eighth of all diseases seen by family physicians in the Netherlands[10]. Another study which analysed statistics from 2002-2005 in the US, found skin conditions to account for 8% of all visits to family physicians and the common skin disorders they diagnosed were dermatitis, pyoderma, tinea, benign neoplasms and candida[11].

In view of the deep psychological impact skin diseases can have and their widespread prevalence in the general population, I trust that the present issue of the Hong Kong Medical Diary will be of great interest to you all. In this issue, we are privileged to have received inputs on the treatment of some of the most commonly encountered skin diseases from a number of very experienced and well-respected dermatologists in town, to whom I would like to express my utmost gratitude: **Dr. KK Lo**, a highly-skilled dermatologist and the former consultant-in-charge of the Social Hygiene Service shares his experience in managing and treating eczema, a very common skin condition amongst the local population; **Dr. LY Chong**, former consultant at the Social Hygiene Service currently in private practice, discusses the complex systemic involvement of psoriasis; **Dr. William Tang**, another eminent dermatologist in private practice shares his experience in managing nail disorders; **Dr. KM Ho**, the consultant-in-charge at the Social Hygiene Service and **Dr. TS Cheng**, an experienced dermatologist provide us with a review of superficial fungal infections. I also provide a review of the medical treatments

for acne in this issue. In addition to the aforementioned medical dermatological diseases, we are also privileged to have **Dr. Henry Chan**, a renowned dermatologist with keen research interests in the field, gives us an update in the recent advancements in cosmetic dermatology. I would also like to thank **Dr. Simon Ku**, a very artistic dermatologist, for providing the cover shot of this issue and **Dr. CK Kwan**, a keen hiker for sharing some of his favourite hiking pathways in Hong Kong.

I hope these articles will convince you that dermatological issues are indeed more than just skin-deep and encourage everyone to give skin diseases their due attention and care. May I also take this opportunity to wish you all a joyful Christmas and all the best in 2011.

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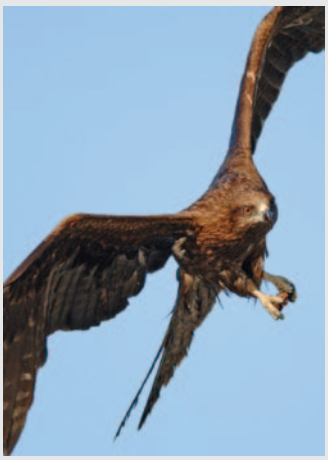
The Cover Shot



Birds Photography

Birds photography is probably one of the most exciting and challenging endeavours of the photographic world. Birds, both at-rest and in-flight, are elegant and beautiful. Capturing wild birds in their natural habitats often requires big telescopic lenses like 500, 600 or even 800mm. This photo is a black kite, the commonest raptor found in HK. It was captured in one late afternoon, when the sun was shining from the side, illuminating her face and the food inside her claws. It was taken at almost eye-level with the bird flying directly towards the camera, giving it an intrusive and vivid composition.

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Practical Approach for "Eczema"

Dr. KK LO

Dr. KK LO

FRCP, FHKCP, FHKAM (Med)
Specialist in Dermatology & Venereology

This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded one CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 30 November 2010.

Eczema is the commonest diagnosis in the clinical practice for both specialists and family doctors when encountering patients complaining of skin problems. Eczema is prevalent in a local students study.

It appears as an apparent simple and easy condition to deal with but indeed it is one of the conditions that our patients are often dissatisfied about the outcome of the treatments. In this brief review, I describe my approach to let family doctors having a more systemic and logical approach to the management of this common skin problem.

My approach to the management of eczema follows to the convention of Western medicine. We shall be able to make the accurate diagnosis by standard clinical methods, namely: clinical history, physical examination and investigations if needed. Having an accurate diagnosis is a half-way success and the subsequent management is divided into general and specific treatments. General treatment is actually more important though very often its emphasis is skipped by both the clinician and our patients.

The terms eczema and dermatitis are regarded as synonyms in clinical practice though dermatitis is used more often for exogenous causes. Eczema is derived from Greek, "ek" meaning out and "zein" to boil. Eczema defines the superficial inflammatory itchy skin problem though eczema may mean different things to different people. There are different views on eczema even among specialists and it is no wonder how confusing our clients and colleagues do in our daily practice. My approach to the diagnosis of eczema is by exclusion. The difference between a specialist and a family doctor may be just in the different length of exclusion list of differential diagnoses (see table 1) in making the diagnosis.

Eczematoid rash is characterised by an erythematous scaly rash with an ill-defined border in different phases, acute, subacute and chronic. Furthermore, it is a dynamic rash in that it will gradually change its configuration, size, shape and colour with time in terms of days, weeks and months. It can remit and reappear with a turnover time from days to months. However, if a persistent (never remitted and stayed in the same location) erythematous scaly rash presents for years, a diagnosis of eczema is unlikely. Skin biopsy or referral to a specialist to exclude other serious conditions of papulosquamous dermatoses (Table 1) must be considered.

Table 1: Differential diagnoses of eczema

Common cutaneous conditions considered for exclusion by family doctors	Additional cutaneous conditions considered for exclusion by specialists
Various forms of Superficial fungal infections	Neoplastic conditions e.g. Mycosis fungoides, Bowen disease, Langerhans cell histiocytosis
Various forms of psoriasis and psoriasiform eruptions	Chronic infections e.g. Hansen disease, secondary syphilis
Lichen planus and lichenoid eruptions	Autoimmune diseases e.g. dermatomyositis, subacute lupus erythematosus, pemphigus foliaceous
Various forms of cutaneous drug eruptions including photodermatitis	Nutritional diseases e.g. zinc deficiency, essential fatty acid deficiency, Pellagra (niacin deficiency)
	Others: pityriasis lichenoides, pityriasis rosea, lichen striatus, glucagonoma syndrome, congenital immunodeficiency e.g. Wiskott Aldrich syndrome & DiGeorge syndrome

Just making an accurate diagnosis of eczema may not be adequate professionally. We should have a very simple classification of eczema so that we may communicate with our clients more confidently though the treatment and outcome may vary little. The classification of eczema will vary from one specialist to another. Mine is a simple one (Table 2). I divide eczema into two main groups: exogenous (external factors may play a more important role) and endogenous (constitutional factor of the person is more important). The prototype examples of exogenous eczema are irritant contact eczema and allergic contact eczema. Usually, contact eczema starts in some local contact sites of our skin and a meticulous examination of the location may give the clues of allergen or irritants. Irritant contact eczema is more common and the different layman names of irritant contact eczema of hands have indicated their common occurrence in the community. A detailed history on occupation, daily hobbies and interests is essential. Allergic contact eczema is a type IV hypersensitivity condition and can be confirmed by appropriate patch tests with specific contact allergens though it may not be easily available in common clinical practice. With good substitution for avoidance of future contact with the allergens or proper protection from the irritants, the prognosis of exogenous eczema is good. However, we should remind our clients not to forget the importance of compliance to general advice and care of the skin

(Table 3) which is the key for a successful cure.

Table 2: Simple classification of eczema	
Exogenous eczema	Endogenous eczema
Irritant Contact eczema e.g. housewife dermatitis	Atopic eczema
Allergic contact eczema e.g. Nickel contact dermatitis	Seborrhoeic eczema
	Stasis eczema
	Discoid/Nummular eczema
	Asteatotic eczema
	Vesicular hands & feet eczema (dyshydrotic eczema or pompholyx)
	Unclassified papular eczema, lichen simplex chronicus

Table 3: General management of eczema	
	Remarks
Environmental modification	Avoid excessive bathing. Avoid all types of contact irritants including common soap, detergent, shampoo and cleansing agents. May need to change or modify job nature or posting. Use appropriate protective measures including gloves, barrier creams
Use soap substitutes and liberal application of emollients	Many types of fragrance free emollients and moisturisers will help skin protection especially in low humidity season. Common brand creams include aqueous cream, emulsifying ointment. Individual preference may affect choice. Proper use of moisturisers would improve the skin barrier function of eczema patients
Diet	The role of food avoidance, food allergy, supplements with probiotics and breast feeding becomes less clear for prevention of atopic eczema in recent studies. Defer introduction of solid food to infants may help to delay onset of atopic eczema but cannot prevent it. Normally, a balanced diet is recommended. Elimination diet for severe atopic eczema is rarely needed and should be conducted with advice of specialists; patients' self initiated food restrictions may not be helpful to improve eczema though psychologically useful. A properly kept food diary would be a useful objective tool to find out the real problematic food
Emotion stability	Occasional extreme anxiety may need psychological treatment
Awareness of complications of eczema e.g. secondary cutaneous bacterial infection	An empirical course of Cloxacillin would be useful for occasional exacerbations of atopic eczema. Severe eczema herpeticum should be referred to specialists and commence a course of acyclovir without delay

Endogenous eczema has a relatively poorer prognosis in that we cannot change our genetic make-up to prevent the relapse of eczema. Common forms of endogenous eczema encountered in our clinical practice include atopic eczema (infantile, childhood, adolescent and adult), seborrhoeic eczema, stasis eczema, vesicular hand eczema (pompholyx), nummular or discoid eczema, asteatotic or xerotic eczema and unclassified popular eczema. Personal and family history of atopy diseases (e.g. asthma, allergic rhinitis) offer hints for the diagnosis of atopic eczema. Specific areas of distribution and involvement in the body e.g. eczema involving scalp, nasolabial fold, V-of chest and flexural area will favour more on the diagnosis of seborrhoeic eczema; eczema involving lower limbs with sign of venous hypertension and insufficiency will guide you to the diagnosis of stasis eczema, coin-like nummular patches in the body and often remit and recur in the same area may point to the diagnosis of nummular eczema; generalised dry skin with fine scaling and superficial cracks on the dry skin especially in the elderly will be an easy diagnosis for asteatotic eczema. However, some of these features may be mixed and not so clearly defined. Clinical experience and closer follow-ups for the course of the eczema will help to give the diagnosis of the very common type of endogenous eczema: unclassified popular eczema. Again, this type of eczema is diagnosed by exclusion of all other possible definite types of eczema before we label it as such. It may transform to a more definite form of eczema later on follow up visits. Lichen simplex chronicus or chronic neurodermatitis commonly appears on some typical sites such as the nape and side of neck, scrotum, vulva, eyelid and ankles. Habitual scratching of these areas results in lichenified hyperpigmented plaques with accentuation of surface markings. Potent topical steroids with occlusion may sometimes be needed to break this itch scratch vicious cycle of lichen simplex chronicus.

The specific treatment for eczema is summarised in Table 4. Perhaps the most difficult part in the management of eczema is to allocate adequate time to explain the disease nature and to understand the genuine concerns of our clients in the consultation. Having a good rapport with our patients, better understanding of general treatment e.g. diet advice, environmental modification, use of emollients and the proper use of topical steroids and immunosuppressive medications, the management of eczema can be optimised with better client satisfaction. Only a few types of extensive or resistant form of eczema (e.g. erythroderma in adult atopic eczema or seborrhoeic eczema) should require the long term care from specialists.

Table 4: Specific treatment of eczema	
	Remarks
Topical medications	Different strengths of topical steroids under doctors' guidance and monitor (must clearly explain to patients for their proper application and their long term adverse effects) are still the mainstay of treatment; potent steroids will only be useful for short term use for exacerbations). Topical Calcineurin inhibitors (expensive but lack skin atrophy on long term use and would be good choice for applying on face or eyelids) are getting popular. Tar smells and its appearance is less acceptable to patients
Oral antihistamines	For symptomatic treatment only. Oral antihistamines suppress pruritus, allay anxiety and allow sleep with less scratch; used as adjunctive treatment
Systemic medications	All are with various degrees of adverse effects and should be used with caution especially on long term use. They should be closely monitored for their efficacy as well as adverse effects e.g. azathioprine, cyclosporine A. Other immunosuppressive therapies e.g. Mycophenolate mofetil, Methotrexate have been tried but they are not considered standard therapy. Long term administration of systemic steroids plays no role in the management of eczema though short term uses can alleviate exacerbations. Once commenced, it is difficult to wean off without flare ups of eczema
Phototherapy	Both narrow band UVB phototherapy and systemic or local PUVA are useful but the long term effects of photoageing and carcinogenic effects limit their application
Others	Wet-wrap is a damp layer of cotton dressing used in combination with emollients or diluted topical steroids wrapping the affected areas overnight with an outer coat of dry gauze wrapping. Its efficacy is good for small children though it is tedious for parents. Traditional Chinese herbal medicine (TCM) has been found to be useful but standardisation of TCM is problematic and it has been reported with hepatotoxicity. No clinical benefits were found in more recent studies with use of evening primrose oil. Reports of homeopathy, mind-body therapies and topical honey to treat eczema are known but evidences are weak

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Practical Approach for "Eczema" " by Dr. KK LO and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 1 CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 30 November 2010. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. **Eczema is derived from Latin with "zein" meaning to cook.**
2. **Eczematoid rash is characterised by a persistent non-scaling erythematous rash on the skin.**
3. **General advice and care of the skin is as important as specific treatment for the management of recalcitrant eczema.**
4. **Stasis eczema is one form of endogenous eczema.**
5. **Lichen simplex chronicus commonly affects the genitalia .**
6. **Viral infection is one of the causes for exacerbations of eczema in patients with atopic eczema.**
7. **An empirical course of Cloxacillin is not recommended for occasional flares of atopic ezema.**
8. **Topical Calcineurin inhibitors are always better than topical steroid treatment for management of eczema.**
9. **Long term phototherapy for treatment of severe atopic eczema is useful and safe.**
10. **Traditional Chinese herbal medicine is not useful in management of eczema.**

ANSWER SHEET FOR NOVEMBER 2010

Please return the completed answer sheet to the Federation Secretariat on or before 30 November 2010 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Practical Approach for "Eczema"

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Answers to October 2010 Issue

Thyroid Eye Disease: a Comprehensive Review

1. F 2. F 3. T 4. T 5. F 6. F 7. F 8. T 9. T 10. F

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Psoriasis is a well known chronic, non-contagious skin disorder since ancient times. The word "Psora" is a Greek word meaning "To itch". It is notorious for its chronicity in its natural course and difficulties in the management. Although great efforts have been tried in medical researches for decades, it is still not curable and its exact aetiology remains unknown.

Prevalence in Chinese

In Western literature, it is well reported that 1-3% of the Caucasian population have psoriasis. Though it is a common skin disease, the prevalence reported in Chinese is lower than that of Caucasians. From the limited resources available, it is estimated that psoriasis occurs in about 0.1-0.3% of the Chinese population.[1]

In 1984, a nation-wide screening of psoriasis had been conducted in 24 regions (53 centres) of China, involving a coverage of 6,617,917 population. About 11,393 cases of psoriasis (0.123% among the population studied; range from <0.1% to >0.3%) had been reported. Interestingly, the prevalence is highest in the Northeast provinces and lowest in the Southern ones where there is more sunshine. It is also higher in cities than the villages.

In Hong Kong, the prevalence is estimated to be approximately 0.3% of the total population.[2] It is the 5th leading new cases of skin disorders in public dermatology clinics, with more than 600 new cases annually.

Immuno-pathogenesis of Psoriasis

Despite its unknown aetiology, there have been breakthroughs in the understanding of the immuno-pathogenesis of psoriasis in recent years. It is now almost certain that psoriasis is a T-lymphocyte mediated inflammatory dermatosis with hyper-proliferation of keratinocytes in genetically predisposed subjects (Diagram 1). It is regarded as one form of immune-mediated inflammatory diseases (IMID), which is a term designated for organ-specific diseases in which cells and cytokines of the adaptive immune system cause tissue inflammation or destruction.

Until recent years, it was believed that IMID was either mediated by Th1 T-cells (which is stimulated by IL-12) or TH2 T-cells. The former subset includes psoriasis, rheumatoid arthritis, multiple sclerosis and inflammatory bowel diseases, while the latter subset includes atopic dermatitis and asthma. A new pathogenic concept in IMID however is developed upon the discovery of a new T-cell lineage in 2005. This new cell lineage is called Th17 T-cell, which is defined by the production of IL-17, and stimulated by IL-23. This subset likely includes psoriasis, rheumatoid arthritis, multiple sclerosis and inflammatory bowel diseases. IL-12 and IL-23 are structurally related with a common 40kD subunit (p40), which leads to the development of a new group of biologics called anti-P40 (anti-IL12/23) that blocks both the Th1 and Th17 pathways.

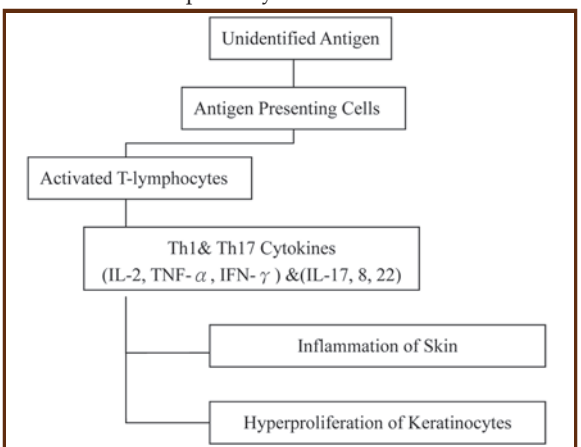

Diagram 1. Postulated Immuno-pathogenesis of Psoriasis

Co-morbidities of Psoriasis

The traditional belief about psoriasis is that it is a cutaneous disease without visceral involvements, albeit 10-30% of the patients have joint involvements. This concept is challenged in recent few years when more and more systemic co-morbidities had been reported. When the term "psoriasis and co-morbidities" is searched in Medline, more than 200 articles can be retrieved over the past two decades. It is one of the hottest research topics in dermatology in the past 5 years, as shown by the numerous publications from different countries and different indexed journals. The possible co-morbidities of psoriasis reported in literatures are summarised in Table 1. Three important areas which may have impacts on medical health are worth mentioning here.

- Cardiovascular diseases (hypertension, myocardial infarction), cerebrovascular and peripheral vascular diseases
- Metabolic syndrome (obesity, diabetes mellitus)
- Non-alcoholic fatty liver
- Autoimmune diseases (Crohn's disease, ulcerative colitis, multiple sclerosis)
- Lymphoma, melanoma, non-melanoma skin cancers
- Depression, suicide
- Smoking, alcoholism
- Osteoporosis

Table 1. Possible co-morbidities of psoriasis reported in literatures

1.Cardiovascular Diseases

Gelfand JM, et al. had published an article on *JAMA* in 2006 concerning the risks of myocardial infarction in patients with psoriasis.[3] They had conducted a population-based cohort study using data collected by general practitioners participating in the General Practice Research Database in the United Kingdom from 1987-2002. A total of 556,995 control patients and patients with mild (n = 127,139) and severe psoriasis (n = 3,837) were studied, and controlled for traditional cardiovascular risk factors (diabetes mellitus, history of myocardial infarction (MI), hypertension, hyperlipidaemia, smoking). They found that the adjusted relative risks of MI are 1.54 (1.24–1.91) and 7.08 (3.06-16.36) respectively in mild and severe psoriasis as compared with controls.

Xiao J, et al. had published another article in *J Eur Acad Dermatol Venereol*. in 2009 about the prevalence of myocardial infarction in patients with psoriasis in central China. Data were collected from the medical records section of five hospitals in the Mainland between 1999 and 2007.[4] After adjusting for systemic therapies and other known cardiovascular risk factors in addition to age and sex, they found that the odds ratio (OR) of having an MI were 1.72 (95% CI, 1.29-2.30) and 2.01 (95% CI, 1.45-2.79) respectively in mild and severe psoriasis.

Related to the cardiovascular co-morbidities, Ludwig RJ, et al. had published an article in *Br J Dermatol*. in 2007 concerning psoriasis as a possible risk factor for the development of coronary artery calcification (CAC). [5] They found a significantly increased prevalence (59.4% vs. 28.1%, P = 0.015) and severity (CAC score according to Agatston 3.7 vs. 0.0, P = 0.019) of CAC in patients with psoriasis vs. controls. Multiple linear regression calculations identified psoriasis as a likely independent risk factor for CAC.

2.Metabolic Syndrome (diabetes mellitus, hypertension, hyperlipidaemia & obesity)

Sommer DM, et al. had published an article about the prevalence of the metabolic syndrome in patients with moderate to severe psoriasis in *Arch Dermatol Res*. in 2006.[6] They had investigated a total of 581 adult patients hospitalised for plaque type psoriasis as compared to 1,044 hospital-based controls. A distinct pattern of chronic disorders was found to be significantly associated with psoriasis, including type II diabetes mellitus [odds ratio (OR)=2.48], arterial hypertension (OR = 3.27), hyperlipidaemia (OR = 2.09), and coronary heart disease (OR = 1.95). The combined presence of these conditions together with obesity, known as the metabolic syndrome, was clearly more prevalent in psoriasis patients (OR = 5.29).

In the cross-sectional study on association between psoriasis and the metabolic syndrome by Cohen AD, et al., published in J. Dermatology in 2008,[7] it had demonstrated that psoriasis was associated with the metabolic syndrome (OR = 1.3, 95% CI = 1.1-1.4), ischaemic heart disease (OR = 1.1, 95% CI = 1.0-1.2), diabetes mellitus (OR = 1.2, 95% CI = 1.0-1.3), hypertension (OR = 1.3, 95% CI = 1.2-1.5) and obesity (OR = 1.7, 95% CI = 1.5-1.9). This study included 16,851 patients with psoriasis and 48,681 controls.

3. Lympho-proliferative Diseases

Gelfand JM, et al. had studied on the risks of lymphomas in psoriasis and published in *J Invest Dermatol*. in 2006.[8] Their study used large population-based cohort data collected from the General Practice Research Database in the United Kingdom (1988-2002), involving 153,197 psoriasis patients and 765,950 control patients without psoriasis. The adjusted relative risks in Non-Hodgkin's lymphoma, Hodgkin's lymphoma, T-cell lymphoma and all lymphomas in severe psoriasis were 0.73, 3.18, 10.75 and 1.59, indicating that there is higher risks of lymphomas in patients with psoriasis.

Possible Mechanistic Links Between Psoriasis and its Co-morbidities

The link between chronic inflammation and metabolic and vascular disorders is now increasingly recognised. It is postulated that proinflammatory cytokines (such as tumour necrosis factor-alpha) involved in the immune-mediated or inflammatory pathway may contribute to atherogenesis, peripheral insulin resistance, and hypertension. Macrophages and adipocytes also share common features such as expression of cytokines, FABPs, and nuclear hormone receptors, which may contribute to obesity.[9]

Many studies had reported that various IMIDs, including psoriasis, are at higher risks of developing systemic co-morbidities. IMIDs may cause these co-morbidities through shared genetic risks, common environmental factors, or common inflammatory pathways that are co-expressed in IMIDs and target organs.[10]

As mentioned in previous paragraphs, psoriasis is now classified as an IMID of the skin with T-cell mediated pathogenetic pathways and involvement of various inflammatory mediators. This may similarly predispose to the increasingly reported associated co-morbidities. The potentially systemic nature of the inflammatory processes in the pathogenesis of psoriasis has thus led to the postulation that it may be considered as a systemic disease, rather than a pure cutaneous disease.

Is Psoriasis Really an Independent Risk Factor for These Co-morbidities?

Despite increasing reports from difficult countries supporting the association of these co-morbidities of psoriasis, there were skeptical views about their true causal relationship. Nijsten T and Wakkee M. had written an excellent and critical commentary in *J Inves Dermatol*. 2009 Jul issue about the complexity of the association between psoriasis and its co-morbidities.[11]

Although these studies did involve very large data base, their designs were not without shortcomings and pitfalls. Most of them are observational studies which were not primarily designed for the detection of these co-morbidities. Diagnostic bias and detection bias were unavoidable.

Moreover, many confounding factors may be involved in these co-morbidities, as illustrated in Diagram 2. For example, psoriasis itself may lead to impaired health-

related quality of life (HRQOL) such as depression, anxiety and stress, which may result in an unhealthy life style leading to alcoholism and chronic smoking. The presence of arthropathy may lead to lack of exercise. The well known side-effects of some systemic antipsoriatic drugs may also count, such as acitretin may cause hyperlipidaemia and cyclosporine may cause hypertension. All these confounding factors may contribute to the cardiovascular diseases and metabolic syndrome. Conversely, obesity and smoking may increase the risk of developing psoriasis as reported, while drugs like lithium in treating manic-depressive illnesses and certain beta-blockers in treating cardiovascular diseases may also induce psoriasis.

Despite many studies that had supported the association of these co-morbidities, there are also inconsistencies in the findings in other studies.[12]

Finally, although in the supporting papers, most of the reported associations had reached statistical significance after statistical analysis, as they had involved very large data base, it is essential to know that **statistical associations do not equate to causal relationships, and do not always have clinical relevancy.**

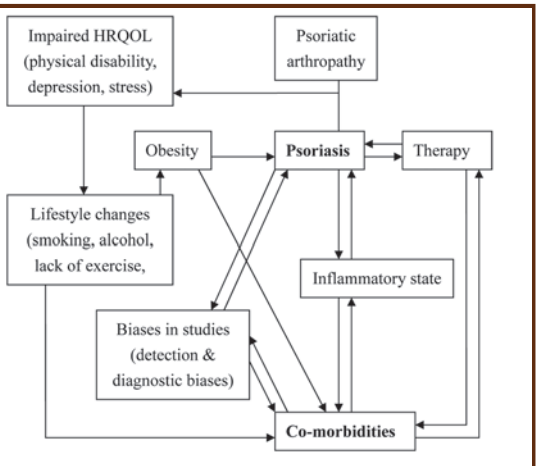

Diagram 2. Schematic overview of possible confounding factors influencing the association between psoriasis and its co-morbidities (Modifeid from Nijsten T and Wakkee M)[11]

Conclusion

Although there are increasing reports that psoriasis may have significant systemic co-morbidities, judging from the present evidences, causality has not yet been proven. Whether there is true causal relationship between psoriasis and these co-morbidities is still uncertain and open to debate. Upgrading psoriasis to an systemic disease obviously will have significant impacts on the management of this chronic disease, such as more aggressive treatments, routine screening of the co-morbidities, and possibly healthcare resources reallocation. Therefore, until more well proven evidences are available, it is still more appropriate to regard psoriasis as a cutaneous disease at the moment. Nonetheless, psoriasis is definitely more than skin depth in its impact on the affected patients, in view of its physical and psychosocial impairments. It also reminds dermatologists the need to manage patients holistically as a whole, rather than just focusing on their skin.

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ELIDEL® breaks the eczema cycle

- Reduces major flares[1]
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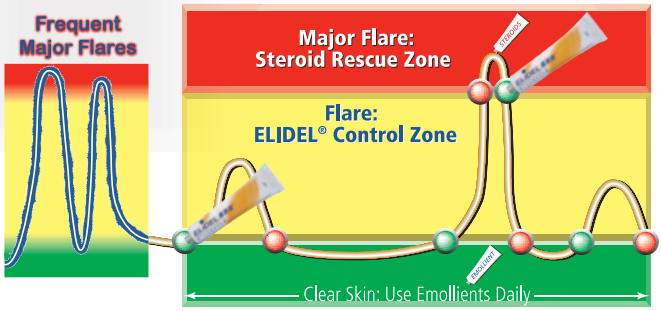
- Reserves mid- to high-potency steroids for rescue therapy[1]



Scenario shown above may not be representative of all patients; individual response may vary.

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‡Treatment should be discontinued upon resolution of disease. Patients should be re-evaluated if symptoms persist beyond 6 weeks or worsen at any time.

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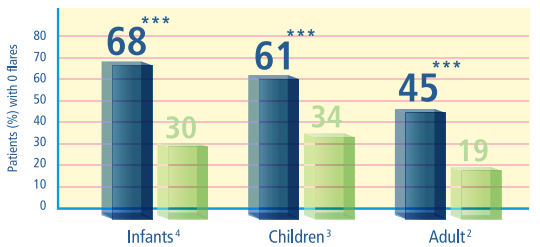
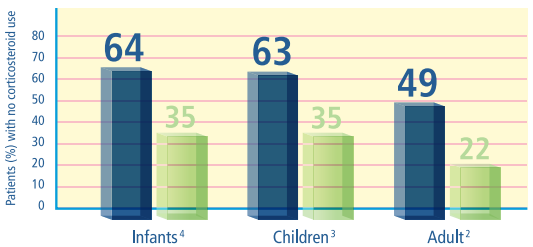
Patients completing 6 months of study with 0 flares

***All ranked flares, p < 0.001: Van Elteren test



• Eliminate the need for corticosteroid[2,3,4]

Patients completing 6 months of study



Study 2: 192 adults patients, with a clinical diagnosis of AD were double-blinded and randomized 1:1 to receive treatment over 24 weeks with either pimecrolimus cream or vehicle cream.
Study 3: 713 children patients (2-17 years), with clinical diagnosis of AD were double-blinded and randomized 2:1 to receive treatment for 1 year with either pimecrolimus cream or vehicle cream.
Study 4: 251 infants patients (3-23 months), with clinical diagnosis of AD were double-blinded and randomized 4:1 to receive treatment for 1 year with either pimecrolimus cream or vehicle cream.

• No steroid side effects such as skin atrophy[6] • Reserves topical corticosteroids for rescue medication[5]

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Important note: *Before prescribing, consult full prescribing information.* ***Presentation:*** *Elidel is available in aluminium tubes with an epoxy protective inner lacquer and polypropylene screw cap. Tubes of 15, 30 and 100 grams.* ***Indications:*** *Elidel 1% cream is indicated for patients 3 months of age and older with atopic dermatitis (eczema) for the short term treatment of signs and symptoms and intermittent long-term treatment of emerging and resolving lesions in atopic dermatitis where the use of topical corticosteroid is not yet warranted, no longer needed, or is inadvisable (according to the usage restrictions in the respective topical corticosteroid Product Information).* ***Dosage and administration:*** *Apply a thin layer of Elidel 1% cream to the affected skin twice daily and rub in gently and completely. Elidel 1% cream may be used on all skin areas, including the head and face, neck and intertriginous areas. Elidel cream should only be applied to areas of eczema (see "Precautions"). The amount of cream to apply can be limited by using the fingertip dosing unit : a fingertip unit is the amount of cream expressed from a tube and applied from the distal skin crease to the tip of the palmar aspect of an adult index finger. A fingertip unit of cream is sufficient to cover the surface corresponding to two hand areas of eczema. Carers should wash their hands after application of Elidel to their children. Emollients can be applied after using Elidel 1% cream. Elidel 1% cream should be initiated at the the first sign of itching (Pruritus), such as persistent scratching or rubbing, and/or persistent redness (erythema), and/or thickening of the skin (Infiltration), to prevent progression to flares of atopic dermatitis. Treatment should be discontinued when there is no longer evidence of the disease apart from dry skin. Treatment should be resumed at the first signs of recurrence. In short-term clinical trials, Elidel cream was used twice daily for up to 6 weeks, or until resolution of signs and symptoms, if this occurred before 6 weeks. In long-term clinical trials Elidel was used intermittently for up to 12 months in infants and children and 6 months in adults (see Clinical Trials section for more information on the extent of drug exposure during the long-term trials). Elidel treatment was initiated at the first signs of itching, redness and/or skin thickening. Treatment with a moderately potent topical corticosteroid was initiated if the disease progressed to flares (severe erythema, papulation/infiltration, with or without oozing/crusting). When topical corticosteroid therapy was initiated for the treatment of flares, Elidel therapy was discontinued. Elidel therapy was recommenced to treat residual disease or if topical corticosteroid therapy was no longer appropriate or inadvisable because of potential risks. In general, the duration of treatment with Elidel cream for each eczema episode is up to 6 weeks (see also"Use in Infants" below). If no improvement in the signs and symptoms occurs after 6 weeks, or in case the condition worsens, Elidel should be stopped and the patient should be re-evaluated. Long-term continuous usage of Elidel cream is not recommended. Therapy should be intermittent, in conjunction with other therapies (see "Indications").* ***Contraindications:*** *Hypersensitivity to pimecrolimus or to any of the excipients.* ***Precautions:*** *Long-term safety of Elidel 1% cream has not been established. Although a causal relationship has not been established, rare cases of malignancy (e.g. skin and lymphoma) have been reported in patients treated with topical calcineurin inhibitors, including Elidel 1% cream. Elidel cream should not be applied to areas affected by cutaneous premalignant changes (e.g. actinic keratoses) as caused, for example, by excessive sun exposure or phototherapy, or to areas where skin cancers have been removed. The safety of Elidel 1% cream has not been established in patients with Netherton's syndrome and generalised erythroderma. Elidel 1% cream is not recommended in patients with Netherton's syndrome or severely inflamed or damaged skin (e.g. erythroderma) where there is a potential for increased absorption. The safety and efficacy of Elidel 1% cream in immunocompromised patients have been studied. The use in immunocompromised patients is therefore not recommended. In clinical studies, 14 cases of lymphadenopathy (0.9%) were reported while using Elidel cream. These cases were usually related to infections and were noted to resolve upon appropriate antibiotic therapy. Of these 14 cases, the majority had either a clear aetiology or were known to resolve. Patients who receive Elidel cream and who develop lymphadenopathy should have the aetiology of their lymphadenopathy investigated. In the absence of a clear aetiology or in the presence of infectious mononucleosis, discontinuation of Elidel cream should be considered. Patients who develop lymphadenopathy should be monitored to ensure it resolves. Elidel 1% cream should not be applied to areas affected by acute cutaneous viral infection. In the presence of a dermatological bacterial or fungal infection, the use of an appropriate antimicrobial agent should be instituted. If resolution of the infection does not occur, Elidel 1% cream should be discontinued until the infection has been adequately controlled. Use of Elidel 1% cream may cause mild and transient reactions at the site of application, such as a feeling of warmth and/or burning sensation. Patients should see a physician if an application site reaction is severe. Patients with atopic dermatitis are predisposed to superficial skin infections including eczema herpeticum (Kaposi's varicelliform eruption). Treatment with Elidel cream may be associated with an increased risk of eczema herpeticum. In the presence of this skin infection, the balance of risks and benefits associated with Elidel cream use should be evaluated. Pimecrolimus per se was neither phototoxic nor photocarcinogenic in animal studies, but the cream base was found to slightly enhance the development of skin tumours induced by UV radiation in hairless mice. Care should be taken to avoid exposure to the sun of skin areas treated with Elidel cream (see "Adverse Reactions" - Post-marketing data). Patients should be advised to wear protective clothing, hats, low irritant sunscreens when Elidel is used. Elidel is to applied first. Elidel should not used in patients who are receiving phototherapy. Care should be taken to avoid contact with eyes and mucous membranes. If accidentally applied to these areas, the cream should be thoroughly wiped off and rinsed off with water.* ***Use in Pregnancy (Category B3):*** *There are no adequate data from the use of Elidel 1% cream in pregnant women. The effects of pimecrolimus on embryofoetal development have not been adequately assessed in animals following dermal administration. Following oral administration, pimecrolimus and at least some of its metabolites crossed the placenta of rats and rabbits. Oral administration of pimecrolimus to rats and rabbits produced no evidence of teratogenicity at respective doses up to 45 and 20 mg/kg/day (associated with blood AUC of 27 and 3 times maximum value observed in adult patients during clinical trials, respectively). However, evidence of embryofoetal toxicity (increased post-implantation loss, reduced live litter size, decreased placental weight, decreased fetal body weight and increased fetal retardation) was observed in rats at oral doses of >10mg/kg/day. No maternal or embryofoetal toxicity was observed in rats dosed at 2 mg/kg/day (corresponding to blood AUC below the maximum value observed in adult patients during clinical trials) or in rabbit doses at 20 mg/kg/day(corresponding to an AUC of 3 times the maximum value observed in adult patients during clinical trials). Elidel cream should not used in pregnant women.* ***Breast-feeding:*** *Caution in nursing women. Nursing mothers should not apply Elidel 1% cream to the breast.* ***Interactions with Other Drugs:*** *Potential interactions between Elidel 1% cream and other drugs have not been systematically evaluated. Based on its minimal extent of absorption, interaction of Elidel 1% cream with systemically administered drugs are unlikely to occur. A vaccination response survey was conduction in 76 children aged 3-23 months who were treated with pimecrolimus 1% cream for up to 2 years. These children had moderate to severe atopic dermatitis with an average of 27.6% total body surface affected. The results showed that the proportions of children who had protective antibodies titres were in accordance with the seropositivity rates of age-matched children reported in literature. Application of Elidel 1% cream to vaccination sites, as long as local reactions persist, was not studied and is therefore not recommended.* ***Adverse reactions: Very common:*** *application site burning ♦* ***Common:*** *application site reactions (irritation, pruritus and erythema), skin infections (folliculitis). ♦* ***Uncommon:*** *impetigo, condition aggravated, herpes simplex, herpes simplex dermatitis (eczema herpeticum), molluscum contagiosum, application site disorders such as rash, pain, paraesthesia, desquamation, dryness, oedema, skin papilloma, furuncle ♦* ***Rare:*** *alcohol intolerance, allergic reactions (e.g. rash, urticaria, angioedema), skin discoloration (e.g. hypopigmentation, hyperpigmentation), malignancies (including lymphoma, skin cancers) without an established causality ♦* ***Very rare:*** *anaphylactic reactions.*

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Nail and Nail Disorders

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The nail unit is a dynamic complex which forms an important part of the integument. This complex consists of the nail matrix (NM), nail bed (NB), hyponychium, nail fold (NF) and the nail plate (NP). Cells of the nail matrix, under the protection of the proximal nail fold mature and keratinised to form the NP. The NM contributes to the most portion of the NP while about 10-15% is produced by the NB. As the NP grows distally, the continued addition of keratinised material makes the NP increases in thickness while it lengthens distally. There are variations in the rate of growth between different nails such that the middle finger (longest digit) grows the fastest. Finger nails grow faster than toe nails. A finger nail grows about 1 cm in 3 months while a toe nail only grows about 1 cm in 9 months. Nail growth can be affected by many factors (Table 1).

Table 1 : Some acquired factors affecting nail growth

Faster	Slower
Daytime	Night
Summer	Winter
Men	Women
Young	Old
Right hand (dominant)	Left hand/non-dominant
Minor trauma/nail biting	Denervation
Fingers	Toes
Psoriasis	Finger immobilisation
Thyrotoxicosis	Yellow nail syndrome
Pregnancy	Fever

Clinical Examination of Nail

When examining the nails, one should examine all 20 nails with the digits relaxed. Nail polish and lacquer should be removed. For a new consultation, it is better to advise the client in advance to avoid applying topical nail medicaments or cosmetics and to keep nail growing for sometime till slightly longer so that an accurate nail examination can be performed. The rest of the skin and mucous membranes and other systems should be examined for evidence of disease. Close examination of pigmented lesions and vasculature can be facilitated by the use of dermoscopy.

Common investigations for nail diseases like swab for culture and sensitivity test, nail scraping and nail clipping are simple and easy to perform. A microscopic examination of the nail scraping can be done after the nail specimen has been treated with 30% potassium hydroxide. Clipped nail specimens should also be sent for fungal culture. However, when the specimens harvested are minute in amount, it is better to send all for fungal culture as mycologic yield for onychomycosis is low. [2] A higher amount of nail specimen could be obtained by curettage. Drilling the proximal border of the diseased nail to obtain nail samples where live fungi could be more abundant has been reported to give a higher yield. [3]

Considering the special anatomical structure of a nail in contrast to skin, investigations like plain X-rays for bone/joint abnormalities, ultrasound and MRI for soft tissue lesions should be made for accurate evaluation and confirmation of the clinical suspicion as deemed appropriate in collaboration with radiologists. The hard keratinous NP forms a natural physical barrier from a thorough clinical examination. In addition, it may also hinder delivery of topical therapy. Therefore, physicians would need reasonably good exploration skill in order to tackle the varied features seen in different nail disorders.

Normal Variants, Minor Ailments and Common Nail Disorders (Table 2, Table 3)

The normal nail appearance varies among individuals. Common features include length and width variation. The size of the lunula also differs among different individuals. A variety of pathological abnormalities can affect the nails but sometimes they do occur in a much milder form in otherwise normal persons. When pits affect a normal person, they are much fewer in number and usually affect only one or two nails; punctate leukonychia occurs as white spots at one or two sites of the NP, possibly attributed to minor trauma and is not significant other than cosmetic nuisance. It is noted that striae leukonychia can sometimes be hereditary and a positive family history gives the clue. Small grooves can occur on the thumb nail due to a habit-tic. One or two splinter haemorrhages under the distal NP can also be trauma-related and very often not due to systemic diseases. Old people have fine longitudinal ridges producing mild NP roughness which is aged-related.

Table 2 : A simple classification of nail abnormalities

Surface	Configuration	Consistency	Soft tissue	Colour	Others
Pits	Koilonychia	Brittle	Paronychia	Leukonychia	Myxoid cyst
Grooves	Clubbing	Hard	Ragged cuticle	Brown	Subungual exostosis
Ridges	Transverse overcurvature	Soft	Splinter haemorrhage	Black	Tumours
Lines		Atrophic	Periungual warts	Yellow	Onycholysis
		Hypertrophic	Pterygium	Blue-grey	IGN*
			Hypertrophic proximal NF	Red	

*IGN = Ingrowing nail

Table 3 : Common nail diseases seen in dermatological clinic						
Idiopathic	**Infection**	**Irritant**	**Inflammatory**	**Trauma**	**Pigmentary**	**Tumour**
Trachyonychia	Onychomycosis	Chronic paronychia	Psoriatic onychopathy	Subungual haematoma	Longitudinal melanonychia	Myxoid cyst
Onycholysis	Periungual wart		Lichen planus	IGTN*		

*IGTN = Ingrowing toe nail

Onychomycosis

Defined as a fungal infection of the nail. Represents up to 30% of diagnosed superficial fungal infections. Fungi include Trichophyton, Microsporum and Epidermophyton species. Yeasts and nondermatophytic moulds are also responsible for a minority of cases.

There are four clinical types of onychomycosis: 1. Distal lateral subungual onychomycosis (DLSO): primarily involves the distal NB and the hyponychium, secondary involvement of the underside of the NP. Usually caused by T. rubrum. 2. Superficial white onychomycosis (SWO): is an invasion of NP on its surface. T. Mentagrophytes is a common pathogen. 3. Proximal subungual onychomycosis (PSO): involves the NP mainly from the proximal NF, caused by T. rubrum and more frequently affecting HIV-positive patients. 4. Candida onychomycosis: commonly affecting nails of hands. If seen in chronic mucocutaneous candidiasis, it may produce massive NB hyperkeratosis and destroys the nail. Whichever the clinical types, untreated onychomycosis rarely may deteriorate to total destruction of the nail plate (total dystrophic onychomycosis).

Moulds account for a minority (<10%) of onychomycosis. Scopulariopsis brevicaulis infections begin at the lateral nail edge and burrow beneath the NP, producing large quantities of cheesy debris. Scytalidium hyalinum produces onychomycosis plus a moccasin-type tinea pedis. Other features may include paronychia, and transverse fractures of the proximal NP. Infections by these organisms occur more commonly when local pathology is present, e.g., chronic paronychia, Raynaud's phenomenon, peripheral vascular disease etc. Cultures of these pathogens should be done using a medium not containing cycloheximide. Treatment efficacy of mould infections using standard oral antifungal treatment is low.

The type of causative pathogens causing onychomycosis varies. In US, most tinea pedis and onychomycosis are due to T. rubrum. In a Mexico school where most people wore leather sandals, Trichosporon cutaneum, Candida spp and Trichophyton mentagrophyte accounted for most infections, whereas T. rubrum was not isolated in them. Cutaneous Scytalidium infections are common in patients from the tropics, especially the West Indies and Africa but not in Hong Kong. Onychomycosis may affect diseased nails such as psoriatic nails and the prevalence varies. It is roughly 22% compared to 13% for those with other diseases. Onychomycosis occurs more frequently in men than in women.

Diagnosis of onychomycosis is confirmed when fungal hyphae are seen by microscopy or by culture. Nail specimens should be obtained from the most proximal part of the diseased area where the disease is active and fungi are still viable. Any dystrophic subungual debris should also be included. Immediate examination after soaking with 30% KOH is possible. Histopathologic examination with PAS stain enhances visualisation of fungal hyphae. Culture is done using Sabouraud agar with chloramphenicol and cycloheximide. Sensitivity of culture varies among different studies and is in general about 30% to 70%. Culture is needed for identification of genus and species. Differential diagnoses for a dystrophic nail include psoriasis, lichen planus, periungual contact dermatitis. Sometimes, the features in these conditions may not be distinguishable. A search for lesions in mucous membranes, scalp and other skin areas may give possible clues.

Although onychomycosis may be asymptomatic, complications arising from non-treated cases do occur. Patients with DM or peripheral neuropathy may be at higher risks from complications. Treatment and its mode depends on many factors such as age, pregnancy, general debilitation, co-existing systemic diseases and concurrent medications, disease severity, treatment cost, and adverse drug reactions. Most traditional topical agents such as castellani's paint, clotrimazole lotion have low efficacy especially for toe nail diseases and the clinical response could only be seen after a long period of treatment. Modern topical drugs like amorolfine and ciclopirox nail lacquers are modestly effective at moderate cost. Systemic treatment using 'modern' antifungals is the main stay of the treatment of onychomycosis. For finger nail diseases, terbinafine is given in doses of 250 mg/day for 6-8 weeks. For toe nails 12-16 weeks, Itraconazole is given in pulses. Each pulse is 200 mg bd for 1 week of each month, for 2 consecutive months for finger nail treatment and 3 to 4 months for toe nails. Fluconazole 150-300 mg once a week for 6 to 12 months appears to be effective. A relapse of onychomycosis after an apparent successful treatment is not uncommon. A recent Korean study on 207 patients reported 36% of relapse. [4]

Table 4: Diagnosis of onychomycosis caused by dermatophytes		
Clinical		**Laboratory**
Primary criteria	*Secondary criteria**	
White/yellow or orange/brown patches or streaks	Onycholysis	Positive microscopy
	Subungual hyperkeratosis/debris	Positive culture for dermatophyte
	NP thickening	

* Tinea pedis often occurs concomitantly with pedal onychomycosis, and T. manuum with infected finger nails

Systemic antifungals are difficult to eradicate moulds such as Scopulariopsis brevicaulis and Fusarium spp. A combination of systemic antifungals and nail lacquers may give a better success rate. Nail avulsion is another option. Candida infection of nails needs topical and systemic treatments. Both itraconazole and terbinafine have been associated with adverse effects such as deranged liver functions. Liver failure is rare and may occur secondary to prolonged high dose therapy in patients with concurrent liver problem. Itraconazole may be associated with a small risk of congestive heart failure and may interact with drugs that are metabolised

by the cytochrome P450 enzyme system. Interactions with terbinafine and tricyclic antidepressants have been reported. Monitoring of LFT is needed for systemic treatment especially for those with a history of hepatic disease or abnormal baseline LFT.

Trachyonychia

Trachyonychia is a condition characterised by a roughness of the nail surface, longitudinal riding, loss of lustre and grey-white discoloration affecting the nails, which become brittle and split at the free edge. Histopathologic examinations reveal features of eczema/dermatitis, lichen planus or psoriasis. It is usually a self-limiting condition. The condition is not exactly the same as Twenty-nail dystrophy which describes any disorder that causes dystrophy of all 20 nails.

Table 7: Causes and association of trachyonychia

Common	Uncommon/rare
Idiopathic (20 nail dystrophy)	Ichthyosis vulgaris
Alopecia areata	Ectodermal dysplasia
Eczematous histology	Selective IgA deficiency
Psoriasis	Knuckle pads and dark lunulae
Lichen planus	Systemic amyloidosis
Chemicals	Pemphigus vulgaris
	Sarcoidosis

Idiopathic trachyonychia of childhood is a benign condition that usually returns to normal within a period of time. Trachyonychia may affect about 12% of children and 3.3% of adults with alopecia areata. It occurs more frequently in alopecia totalis or alopecia universalis. Trachyonychia is mostly symptomless and patients only complain of cosmetic nuisance and sometimes brittle nails. It will not produce subsequent nail scarring. Treatment is not usually required. A mild topical steroid and moisture may be prescribed but there is no evidence to support that they hasten recovery.

Onycholysis

Refers to the detachment of NP from the NB. Most often the detachment occurs at the distal free margin, less commonly it occurs laterally or proximally. When the detachment extends proximally and reaches the matrix, the process is complete. The free edge may rise up like a hood, or coils upon itself like a roll of paper.

The greyish white colour that appears in the onycholytic part of a nail is due to subungual air collection but the colour may vary from yellow to brown, depending on the aetiology. Onycholysis creates subungual spaces that gather dirt and keratinous debris. This area may become malodorous.

In psoriasis, there is a yellowish-brown margin visible between the pink, normal nail and the white, separated area. In the oil spot variety, the NP-NB separation starts in the middle of the nail and sometimes is surrounded by a yellow margin due to accumulation of serum-like exudates containing glycoprotein, in and under the affected nails. Glycoprotein deposition is common in inflammatory and eczematous diseases affecting the NB.

Onycholysis can be asymptomatic, producing cosmetic nuisance, but it can be painful at times. Transillumination of the terminal phalanx gives a good view of the affected areas. The longer the duration of onycholysis the more difficult the NP would reattach onto the NB.

Table 9: Some causes of onycholysis

Cause	Examples	Comment
Idiopathic		? irritant, cosmetic, Water
Systemic	Hyper- hypo-thyroidism Fe deficiency Lupus erythematosus Pregnancy	
Cutaneous	Psoriasis Blistering diseases Lichen planus Contact dermatitis NB tumour	In psoriasis, the separation may start in the middle of the nail. Oil-spot sign+.
Drugs	Bleomycin, retinoids, captopril	
Photo-onycholysis	Chlorpromazine, chloramphenicol, tetracyclines, Psoralen-UVA, thiazide, diuretics.	Photo-onycholysis may be sudden. Examine the skin.
Trauma	Accidental, occupational, sculpture	
Chemicals	Nail cosmetics Hydrofluoric acid.	Contains formaldehyde
Infection	Onychomycosis Candida onycholysis Subungual wart	Candida onycholysis exclusively affect finger nails.
Congenital	Malalignment of the hallux nails	Pseudomonas pyocyanea produces greenish-yellow discoloration

Patients with multiple nail onycholysis should be scrutinised for a systemic cause like thyrotoxicosis or iron deficiency. Bacterial swabs for culture taken from onycholytic NB and clipped nail for fungal culture for suspected superimposed bacterial or fungal infection. Treatment of onycholysis depends on the underlying cause. For chronic idiopathic cases, the prognosis is guarded. Patients should refrain from contact with irritants, chemicals and avoid local trauma. Hand protection is needed when doing wet work. Proper drying of fingers after hand washing is needed. The area can be made dry by using a hair dryer. The detached NP should be clipped away. If it is complicated by a nail infection (e.g., Pseudomonas, aspergillus, an antibacterial solution should be applied to the exposed nail bed 1-2 times a day. 2% acetic acid may also be used for Pseudomonas infections.

Chronic Paronychia

An inflammatory reaction of the proximal NB to irritants or allergens. It affects the finger nails that are continually exposed to a wet environment and to multiple microtrauma, favouring cuticle damage. It is more common in DM, F>M. Majority affects 30-60 year olds but sometimes seen in children who suck or bite fingers. Risk groups: bar-tenders, canteen workers, chefs and fishmongers. Often the index and middle fingers are affected for they are more likely subject to minor trauma.

The condition begins as a slight swelling at PNF of one or more nails. The PNF becomes tender but less than in acute paronychia. Often the cuticle has vanished and sometimes pus may form below the nail fold. Secondary colonisation with C. albicans and/or bacteria occurs in many cases. Darkening and irregularity of the nail edges are frequent. (Darkening can be due to pigments from Pseudomonas infections).

Pathogenesis

Although candida and bacteria may be isolated, the condition is primarily not an infection. Some postulated that foreign materials which could be derived from non-viable C. albicans had been introduced into a relatively sterile nail fold. This sets up an inflammatory reaction accounting for the chronicity of paronychia. Acute exacerbations may occur and are due to secondary bacterial infections. Cultures show various organisms such as S. aureus or S. albus, Proteus spp, Escherichia coli, and Pseudomonas pyocyanea.

Treatment

Chronic paronychia usually gives good response to potent topical steroids. Broad spectrum topical antifungal and antibacterial solutions can be given during exacerbations secondary to superimposed fungal/bacterial infections. Patients must be educated to keep nails dry. Wet work must be avoided or else use a pair of cotton gloves under rubber gloves. Any fiddling with the nails is detrimental to healing. The affected finger nails can be temporarily covered with a porous surgical tape for protection, but prolonged occlusion aggravates. For recalcitrant paronychia, surgical excision of the NF may help.[5] Removal of nails is not necessary except if extensive Candida infection of the NP occurs.

Ingrowing Nails (IGN)

The condition refers to nail growing into the surrounding soft tissue. Often the toe nails especially the big toe is affected and often the lateral and medial margins of the NP grow into the sulcus and may even form a spur like projection, resulting in pain, inflammation and secondary infections. Both the finger and toe nails can be affected and it can also be bilateral.

The types of IGN occur in neonates, infants and adults appear different in their predisposing causes. In the commonest types that affect adolescents and adults, a broad rigid NP, a transverse overcurvature, tight footwear and cutting the NP following the contour of the NP instead of cutting it square are said to predispose to ingrowing. Four major types are described in adults: distal nail embedding, pincer nail, juvenile IGTN and hypertrophy of the lateral lip. Distal nail embedding occurs when a distal soft tissue wall develops after nail shedding following subungual haemorrhage or nail avulsion.

Management

Prevention: treat any predisposing factors (onychomycosis, hallux vulgas). Trim the NP square, rather then curving them to follow the contour of the toe.

If an incipient ingrowing nail is recognised, a wisp of cotton inserted under the nail plate will often permit it to ride up and out of the sulcus. In established cases, excision of the granulation tissue, antibiotics, dressings and removal of a portion of the NP and the NM (wedge excision) are indicated in order to achieve healing and preventing recurrences. The use of a flexible plastic tube split longitudinally inserted along the NP edge as a splint allows recovery of mild forms of IGTN. This treatment principle was first advocated by Wallace et al.[6]

Longitudinal Melanonychia (LM)

Describes the presence of single or multiple longitudinal pigmented streaks within the nail.

Causes

1. Focally active NM melanocytes but normal melanocyte number.
2. NM melanocyte hyperplasia but without nest formation.
3. NM naevus: architecture is similar to that of skin naevus with nests arranged at dermo-epidermal junction.
4. Melanoma of the nail soft tissue: affects 2-3% of melanomas in Caucasians but 15-20% in Blacks. No sex predominance. Mean age 55-60 years. Mostly found in the thumbs or great toes.

Features of nail melanoma

1. A spot in the NB/NM.
2. LM running through the whole visible nail.
3. Hutchinson's sign: brownish black pigmentation from LM on the proximal and lateral NFs. But not exact for Bowen's disease of nail unit, hyperpigmentation only reflects rough 'transparent' NFs (pseudo-Hutchinson's sign).
4. Others: progressive darkening of pigmentation, nail deformity, ulceration, bleeding or tumour mass breaking through the nail.

Diagnostic confirmation is histologic. About 25% of melanomas are amelanotic and may mimic pyogenic granuloma, granulation tissue or IGN. Other simulators: chronic paronychia, mycobacterial infection, mycotic onychodystrophy, and subungual haematoma.

Nail unit melanoma has a poor prognosis, with up to 50% of patients dying within 5 years of the diagnosis. The 5 year's survival rates were reported from 35-50%.

The ABCDEF alert for subungual melanoma[7]

A: Age (peak incidence being in the 5th to 7th decades of life)

B: Band (pigment from brown to black, breadth of 3 mm or more, irregular and blurred borders)

C: Change in the nail band (rapid increase in size, failure of nail dystrophy to improve with adequate treatment)

D: Digit most commonly involved (thumb>hallux>index finger, single digit >multiple digits, dominant hand)

E: Extension of the pigment to soft tissue (Hutchinson's sign)

F: Family or personal history of dysplastic naevus or melanoma

Conclusion

The nail apparatus is a complex integumental structure. The nail can be affected by many diseases be they primary or secondary. Treatments for some of the nail disorders are difficult. A good understanding of the nail physiology and surgical anatomy enables clinicians to deliver the best possible treatment.

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Approach to Acne Management – A Review of the Different Medical Treatments

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Introduction

A recent study has shown that the prevalence of acne among Hong Kong adolescents was at a staggering 91.3%[1] and a study in Australia also found acne to be one of the most common dermatological diseases encountered by the family physicians[2]. Although acne is never life-threatening, it can nonetheless have profound psychological impacts on patients if left untreated. Psychiatric disorders including depression and anxiety disorders can develop secondary to acne[3-7] and in some cases, acne patients actually experience more severe anxiety and depression than patients with serious medical illnesses including cancer[7]. Through providing effective treatment to acne patients, not only were they physically treated, they also report significant improvements in self-esteem, affection, obsessive-compulsiveness, shame, embarrassment, body image, social assertiveness and self-confidence[8].

Pathophysiology of Acne

In order to effectively manage acne, it is important to understand the pathophysiological causes of acne – (1) Increased sebum production, (2) Abnormal desquamation of follicular epithelium, (3) P. acnes proliferation and colonisation and (4) Inflammation and immune response. Treatments for acne can be classified into topical and systemic medications. Different treatments should be given to patients according to their clinical status and background health history. Table 1 summarises the actions of the common acne medications on these pathophysiological factors.

Actions of different treatments

Suppression of:

	Sebum production	Comedones	*P. acnes*	Inflammation
Benzoyl peroxide	—	↓	↓	↓
Topical antibiotics	—	↓	↓	↓
Topical retinoids	—	↓↓	—	↓
Oral isotretinoin	↓↓	↓	↓↓	↓↓
Oral antibiotics	—	↓	↓↓	↓↓
Oral contracetpives	↓	—	↓	↓

Table 1

Treatment of Acne

Topical Medications

1.Topical retinoids
Topical retinoids help to reduce obstruction within the follicles and are effective in the management of both non-inflammatory and inflammatory acne[9]. Different concentrations of topical retinoids are available for acne of different severity. When prescribing a topical retinoid, it is important to warn the patients that it may sometimes cause skin irritation[10].

2.Topical benzoyl peroxide
Benzoyl peroxide is a bactericidal agent that has been proven effective in acne treatment. It can help to prevent or decrease the development of P acne resistance[11].
As a result, it is often used in combination with oral or topical antibiotics.

3.Topical antibiotics
Topical antibiotics have been shown to be effective in managing mild acne and are well tolerated[12]. However, the increase in resistance of P. acnes to topical antibiotics has limited the use of these medications as a single therapeutic agent[13].

In summary, topical mediations are effective in treating mild acne. Combinations of topical treatments are often used as studies have shown that combination therapies are better than monotherapy: Combining topical retinoids and topical antibiotics is more effective than either agent used alone[14] Combining topical antibiotics with benzoyl peroxide reduces bacterial resistance, enhances efficacy and is more effective than using either of the agents alone[15].

Systemic Medications

1.Systemic antibiotics
Systemic antibiotics are commonly used to treat moderate and severe acne. They help to reduce the P acnes population and decrease inflammation. The most commonly used antibiotics are tetracycline, doxycycline, and minocycline[16]. It is important not to use these medications in pregnant women as it may lead to certain birth defects and discolouration in the babies' teeth. Patients should also be warned about the possible adverse side-effects of systemic antibiotics, such as gastric upset. In addition, doxycycline may be associated with photosensitivity and minocycline may be associated with pigment deposition in the skin, mucous membranes and teeth.

2.Oral contraceptives

Oral contraceptives have been shown to be effective in treating acne[17]. It is particularly good for patients who also need contraception. However, the clinical response is usually slow. Patients must also be warned of the possible side-effects, including breast tenderness, weight gain, melasma, etc.

3.Oral isotretinoin

Oral isotretinoin targets all the pathophysiological factors of acne[18]. The indications for the use of this medication include (1) severe nodular acne, (2) acne resistant to oral antibiotics and (3) acne that produces physical scarring or psychological impacts. The approved dosage is 0.5 to 2.0 mg/kg/day and a course usually lasts a few months. There is a greater chance of remission if patients receive 120 – 150mg/kg over the treatment course. According to the literature, 39% of patients treated with oral isotretinoin experienced no relapses after stopping the medication for 3 years. Since isotretinoin is a vitamin A derivative, it interacts with many of the biological systems of the body, and may cause side-effects including those of the mucocutaneous, musculoskeletal, ophthalmic and liver systems. Elevation of blood cholesterol as well as headaches may also occur. Changes in mood, and even suicidal tendencies have also been reported in patients taking the medication. Most of the adverse effects are temporary and resolve after the drug is discontinued. It is therefore important to watch out for these adverse effects in each consultation.

In short, systemic medications are effective in treating moderate to severe acne. However, patients should always be warned about the potential side-effects before prescribing these medications.

Summary

As acne is one of the most commonly encountered skin diseases amongst doctors and that it is a disease that can be physically and psychologically distressing to patients, it is hoped that this review will help practitioners to identify the appropriate treatment for acne of various severity. Table 2 summarises the acne treatment algorithm suggested by the American Academy of Dermatology[20]. It can be seen that a topical retinoid alone is already effective for mild acne, whereas oral plus topical medications are needed to treat moderate to severe acne.

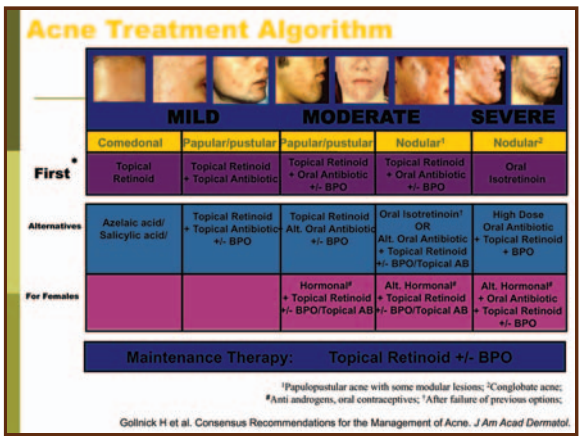

Table 2

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Erratum

1. With reference to the article 'Thyroid Eye Disease: a Comprehensive Review' in the Oct 2010 Issue, the legends of Figure 3 and Figure 4 on page 6 should be:

   Figure 3 (A) Conjunctival injection seen around insertion of horizontal (in this case, lateral) recti muscles. (B) Exposure keratopathy and early microbial keratitis. (C) Severe conjunctival chemosis and injection during active TED
   Figure 4 (A) Diffuse, symmetric, tendon-sparing extraocular muscles enlargement in axial CT scan of the orbits. (B) Symmetric severe proptosis, straightening of optic nerves with mainly enlargement of fat compartment and minimal muscle involvement often seen in young female patients with TED who are typically non-smokers.

2. With reference to the article "The Roles of Dental Professionals in the Management of Obstructive Sleep Apnoea" in the Mar 2010 Issue, The title of the author, Dr Hannah Daile CHUA, on page 4 should be:

   Dr. Hannah Daile CHUA
   DMD, MA, MDS, MOSRCS, PhD
   Post-doctoral Fellow, OMFS, Faculty of Dentistry, The University of Hong Kong

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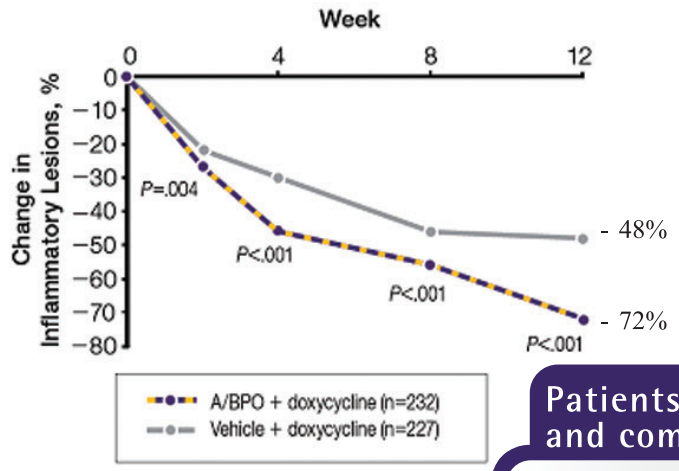
Median reduction in inflammatory lesions over 12 weeks (N=459)



At week 4

- 46% median reduction in inflammatory lesions at week 4 (P< .001)[1]

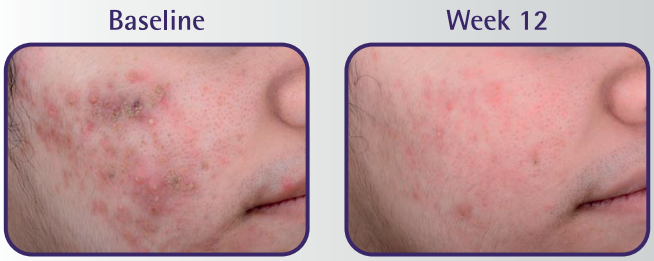
At week 12

- Patients treated with Epiduo® Gel + 100 mg doxycycline experienced a 64% median reduction in total lesions at week 12 (P< .001)[1]
  - 72% median reduction in inflammatory lesions at week 12 (P< .001)[1]
  - 61% median reduction in noninflamma tory lesions at week 12 (P< .001)[1]

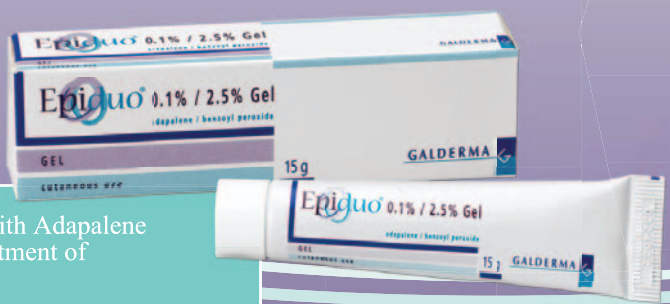
Cutaneous tolerability results

- Mean tolerability scores for both arms at each visit were all less than mild (<1)
- Few local cutaneous dermatologic adverse events were reported in the Epiduo® Gel + 100 mg doxycycline arm (1.7%)
- Most reported adverse events were GI disorders, which were primarily associated with doxycycline use (9.6%)

Patients with inflammatory and comedonal lesions[1]



Patient 9076 (Caucasian female)



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atients with severe acne[1]

Effective and Safe Combination Therapy for Severe Acne Vulgaris: A Randomized, Vehicle-Controlled, Double-blind Study of Adapalene 0.1%–Benzoyl Peroxide 2.5% Fixed-Dose Combination Gel With Doxycycline Hyclate 100 mg

...old, MD; Alma Cruz, MD; Lawrence Eichenfield, MD; Jerry Tan, MD; Jose... ...MSc; Jean-Charles Dhuin, MSc

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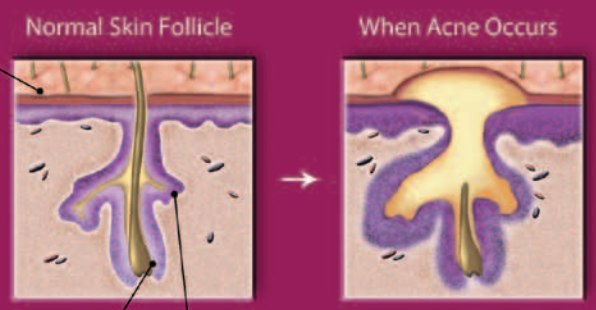

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Common Superficial Fungal Infections – a Short Review

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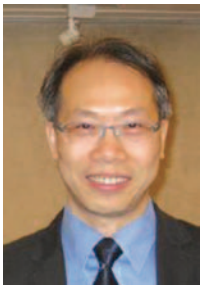


Dr. King-man HO Dr. Tin-sik CHENG

Introduction

Superficial fungal infections of the skin are among the most common diseases seen in our daily practice. These infections affect the outer layers of the skin, the nails and hair. In contrary to many of the other infections affecting the other organ systems in humans, the fungi may cause dermatological conditions that do not involve tissue invasion. On the other hand, the skin surface is the habitat of some of these fungi and is liable to environmental contamination. Therefore, mere isolation of these organisms from clinical specimens taken from the skin surface is not a sine qua non of their role in the disease causation. The main groups of fungi causing superficial fungal infections are dermatophytes, yeasts and moulds.

The dermatophytes that usually cause only superficial infections of the skin are grouped into three genera: *Microsporum, Trichophyton, and Epidermophyton.* They can be classified into three groups according to their normal habitats: 1) humans: anthropophilic species 2) animals: zoophilic species 3) soil: geophilic species. Dermatophytes grow on keratin and therefore cause diseases in body sites wherein keratin is present. These sites include the skin surface, hair and nail. Presence of hyperkeratosis such as palmoplantar keratoderma predisposes to dermatophyte infections. *Trichophyton rubrum* is the most common cause worldwide for superficial dermatophytosis.

Yeasts are not inherently pathogenic, but when the host's cellular defences, skin function, or normal flora are altered, colonisation, infection, and disease can occur. *Candida* is a normal inhabitant of the oropharynx, gastrointestinal tract and vagina in some people. Moist, wet conditions favour *Candida* overgrowth and can lead to superficial infections of the skin. *Candida albicans* is the most virulent of these organisms, and may cause diseases of the skin, nails, mucous membranes and viscera.

The yeast *Malassezia furfur*, a skin commensal, can cause pityriasis versicolor and pityrosporum folliculitis. The fungus is also related to seborrhoeic dermatitis though true infection is not present and its presence on the skin is not a sufficient condition for disease expression. The presence of oil facilitates the growth of this organism.

Moulds that are also referred as nondermatophyte filamentous fungi are ubiquitous in the environment but are not commonly pathogenic in normal hosts. They are however not uncommonly isolated from clinical specimens for fungal culture. Most of the time, they can be regarded as innocent bystanders or contaminants. These organisms sometimes however may have roles in disease causation.

Diseases Caused by Dermatophytes

Species from the genera *Epidermophyton, Microsporum* and *Trichophyton* are most commonly involved. Species from the genera *Epidermophyton* species affect nails and skin, *Microsporum* species affect hair and skin while *Trichophyton* species affect hair, nails and skin. Dermatophyte infections are subclassified in Latin names according to the sites of skin involved, e.g. tinea faciei: face; tinea manuum: hands; tinea corporis: glabrous skin, tinea cruris: crural folds; tinea pedis: feet; tinea capitis: scalp; tinea unguium: nails. Infections involving more than one site of the integument is not uncommon. It is an axiom to look for infections of other sites when dermatophytosis is found in any one of these sites.

Tinea Corporis and Variants

Tinea corporis, the classic 'ringworm', is a dermatophyte infection of the glabrous skin of the trunk and extremities. Common causes are *T. rubrum* and *T. mentagrophytes.* The typical lesions are pink-to-red annular or arciform patches and plaques with scaly or vesicular borders that expand peripherally with a tendency for central clearing. Inflammatory follicular papules may be present at the active border. Sometimes, when the follicular epithelium is grossly involved resulting in folliculitis, it is known as Majocchi's granuloma.

Topical steroids are often prescribed for skin rash. Sometimes, they are wrongly prescribed for tinea. The presentations of the fungal infections are changed as the inflammatory response is decreased leading to the condition known as tinea incognito. The well defined margins and scaling may be absent while diffuse erythema and scales, papules and pustules may be found.

When the face is affected, it is called tinea faciei. The typical annular lesions with peripheral scaling are frequently absent. The clinical clue is the presence of red borders which are faintly demarcated, serpiginous and involving only one side of the face.

Tinea Pedis

Tinea pedis occurs in four main patterns: interdigital, moccasin, ulcerative and vesiculobullous. In the interdigital type, erythema, scaling and maceration with

fissures are found in the web spaces, in particular, the web space between the 4th and 5th toes. This type is usually associated with *T. rubrum* or *T. mentagrophytes*.

Diffuse scaling on the soles extending to the sides of the feet is found in the moccasin type, usually caused by *T. rubrum*. Genetic predisposition is proposed to explain the strong family history and recalcitrant nature of this type of tinea pedis.

The ulcerative type, usually caused by *T. mentagrophytes* var. *interdigitale*, typically begins in the two lateral interdigital spaces and extends to the lateral dorsum and the plantar surface of the arch. The lesions of the toe webs are usually macerated and have scaling borders. Secondary bacterial infection is not uncommon which may be referred to as mixed toe web infection.

In the vesiculobullous type, usually caused by *T. mentagrophytes* var. *interdigitale*, vesicular eruptions on the arch or side of the feet are found. This type may give rise to the dermatophytid reaction which is an inflammatory reaction at sites distant from the site of the associated dermatophyte infection. Pompholyx like lesions on the hands are the classic dermatophytid reaction.

Tinea Manuum

Tinea manuum may present as diffuse hyperkeratosis with predilection to the palmar creases of the palms and digits. White powdery scales along the palmar creases are typically seen. It may also present as annular lesions like the typical tinea corporis but on the dorsum of the hands or as pompholyx like lesions on the palmar aspect. Infection of only one hand is common and usually occurs in a patient with concomitant tinea pedis. The term 'two feet and one hand syndrome' is coined to describe this interesting condition. Tinea unguium of the involved hand might be observed. The typical fungi responsible for tinea manuum are the same as those for tinea pedis and tinea cruris.

Tinea Cruris

Flexural tinea usually only occurs in the groins and does not involve the axillae or submammary folds. The infection occurs more in males. It begins in the crural folds and may extend to the thighs, buttocks and gluteal cleft area. Scrotal infection alone is rare. Infection is nearly always from the patient's own feet and is caused by the same organisms as those causing tinea pedis.

Tinea Unguium

Both dermatophytes and non-dermatophytes can cause onychomycosis. Less than 10% of cases of onychomycosis are due to yeasts or non-dermatophyte moulds, while dermatophytes account for approximately 90% of cases. Toenail infections are more common than fingernail infections and are usually found along with tinea pedis. The main causative dermatophytes are *T. rubrum, T. mentagrophytes* and *E. floccosum*. The clinical presentations of onychomycosis are as follows: 1) distal and lateral subungual onychomycosis (DLSO): it is the most common type, usually caused by *T. rubrum*. Discolouration, subungual hyperkeratosis and distal onycholysis start at the hyponychium spreading proximally. 2) proximal subungual onychomycosis (PSO): the dermatophytes invade the nail unit under the proximal nail fold and spread distally. It is usually caused by *T. rubrum* and is usually associated with immunosuppressed conditions, e.g. HIV infection. 3) superficial white onychomycosis (SWO): the fungi, mainly *T. mentagrophytes*, directly invade the superficial layers of the nail plate but do not penetrate it leading to a white, crumbly nail surface. 4) total dystrophic onychomycosis.

Tinea Capitis

Tinea capitis usually occurs predominantly in prepubertal children. It can be acquired from infected puppies and kittens and by close contact with infected children. The three most common dermatophytes causing tinea capitis are *Trichophyton tonsurans*, *Microsporum canis* and *Microsporum audouinii*. The causative agent varies in different geographical areas. In the USA and in some cities in the UK, *T. tonsurans* is the most common cause. In Hong Kong, tinea capitis is usually caused by *M. canis*. Pet exposure is associated with infections caused by *M. canis*.

The dermatophytes can invade hair in three patterns: ectothrix, endothrix and favus. Arthroconidia are found around the hair shaft in ectothrix infections and within the hair shaft in endothrix infections. Hyphae and air spaces are found within the hair shafts in favus. Many fungi producing a small spore ectothrix pattern and *T. schoenleinii*, which causes endothrix infection, will show fluorescence under Wood's light because of the presence of pteridine.

Tinea capitis can present in the following patterns: seborrhoeic pattern, black-dot pattern, kerion and favus. In the seborrhoeic pattern, dandruff-like scaling is found on the scalp. Prepubertal children presenting with suspected seborrhoeic dermatitis on the scalp should be presumed to have tinea capitis until proven otherwise. In the black dot pattern, patchy alopecia with black stumps of broken hair shaft due to breakage of hair near the scalp are found. In kerion, boggy masses covered with pustular folliculitis are found and scarring may ensue afterwards. In favus, most frequently caused by *T. schoenleinii*, yellow saucer-shaped adherent crusts made up of hyphae and spores occur around the hairs.

Fungal culture and species identification provide additional information for patient management. Zoophilic dermatophytes, *M. canis*, may have an animal source and therefore the pets should be examined by veterinary surgeons for the presence of similar infections. Anthropophilic dermatophytes, *T. tonsurans*, should prompt the attending physician to look for infections of the household or institutional contacts or even institutional outbreaks. Mild infections and asymptomatic carriers with positive fungal culture but no clinical signs can be found in tinea capitis, especially in *T. tonsurans* infections. The carriers are considered infectious as they shed the fungus. Institutional outbreaks are however very uncommon in Hong Kong.

Diseases Caused by Yeasts

Candidiasis

Candida species are capable of producing skin and mucous membrane infections. However, cutaneous candidosis is less common than dermatophytosis. There are about two hundred species in the genus *Candida* and about twenty of them are associated with human or animal infections, e.g. *C. albicans, C. tropicalis, C. glabrata, C. parapsilosis, C. krusei, C. guilliermondii, with C. albicans* accounting for most of the infections.[1] A decrease in the prevalence of *C. albicans* as a cause of infection and an increase in non-albicans *Candida* (NAC) such as *C. glabrata, C. krusei,* and *C. parapsilosis* was found in the last decade. It could possibly be due to the extensive use of azole drugs, such as fluconazole, which led to the emergence of those non-albicans species such as *C. glabrata* or *C. krusei*, with inherent lower sensitivity to azoles.

The organisms can exist as commensal flora but become pathogenic in various predisposed conditions e.g. infancy, pregnancy, moist and occluded sites, diabetes, Cushing's syndrome, immunosuppression, imbalance in the normal microbial flora, etc. The yeast infects only the outer layers of skin and mucous membrane in mucocutaneous candidosis.

Skin and Intertriginous Infections

In the skin, pustules are formed which dissect under the stratum corneum peeling it away resulting in a red, denuded, glistening surface with a long, cigarette paper-like, scaling and advancing border. Pustules rupture to form a superficial collarette of scales. The infection is usually found in the intertriginous skin folds and other moist, occluded sites, e.g. webspaces, genital area and area covered by diaper. Intertriginal candidal infections affect all flexures, e.g. the groins, axillae, finger and toe webs. The rash is red, macerated and well demarcated and surrounded by satellite papules and pustules. A fringe of moist scale might be found at the border. The rash may be sore rather than itchy. In diaper dermatitis caused by *Candida*, bright red plaques in the inguinal and gluteal folds and satellite pustules may be found. Candidal infection is a frequent cause of chronic paronychia, manifesting as painful periungual erythema and swellings associated with secondary nail thickening, ridging and discolouration.

Infections of the Mucosae and Mucocutaneous Junction

Oropharyngeal candidiasis causes white plaques and pustules on the oral mucosal surface that leave a raw, bleeding base when removed mechanically. Median rhomboid glossitis is associated with *Candida* infection. *Candida* sp may cause angular cheilitis which is predisposed in drooling and edentulous elderly patients. Candidal balanitis is more commonly found in the uncircumcised and usually presents with red patches, swelling and tiny pustules. In candidal vulvovaginitis, usually causing itchiness and soreness, a curd-like discharge, pustules, erythema and oedema of the vagina and vulva are found. Pruritus ani and macerations are usually found in perianal candidiasis.

Chronic Mucocutaneous Candidiasis

Chronic mucocutaneous candidiasis is a clinical entity associated with a heterogeneous group of autoimmune, immunologic and endocrinologic diseases which is characterised by recurrent or persistent superficial candidal infections due to an impaired cell-mediated immunity against *Candida* species. This is a clinical situation wherein true candidal infection of the nail is present.

Malassezia Infections

The genus *Malassezia* comprises a group of lipophilic yeasts that have their natural habitat on the skin of humans and different warm-blooded animals. The geographic distribution of *Malassezia* species is worldwide. Since the taxonomic revision in 1996, the genus *Malassezia* was enlarged to comprise seven different species. They are part of the normal flora of human skin and *M. sympodialis* is the predominant species. Pityriasis versicolor is the only human disease in which the causative role of the lipophilic yeast *Malassezia* is fully established. *Malassezia* has been implicated in several other skin diseases, including seborrhoeic dermatitis, atopic dermatitis and folliculitis. However, the role of these yeasts in these entities is controversial.

Pityriasis Versicolor

Pityriasis versicolor occurs most frequently in hot and humid tropical climates. However, it is also prevalent in temperate climates. The fact that *Malassezia* has an oil requirement for growth explains the increased incidence in adolescents and the predilection for sebum-rich areas of the skin. Pityriasis versicolor occurs when the budding yeast form transforms to the mycelial form. Various factors have been implicated, including hot and humid environment, oily skin and excessive sweating. Because this yeast is lipophilic, use of bath oils and skin lubricants may enhance disease development.

The lesions consist of multiple white, pink to brown, oval to round coalescing macules and patches with mild and fine scaling mainly found on the seborrhoeic areas, in particular, the upper trunk and shoulders. Lesions can also be found on the face, scalp, antecubital fossae, submammary regions and groins. The lesions often become confluent and quite extensive. When pityriasis versicolor involves the flexural areas, it is sometimes referred to as 'inverse' pityriasis versicolor. Demonstration of the associated scale may require scratching of the skin surface. The fungus produces dicarboxylic acids, notably azelaic acid, that interfere with melanin synthesis leading to decreased pigmentation. Decreased tanning, due to the ability of the fungus to filter sunlight and the screening effect of tryptophan-dependent metabolites are other factors that explain the absence of pigmentation in exposed areas.

Malassezia (Pityrosporum) Folliculitis

This is characterised by inflammatory follicular papules localised predominantly on the back, chest and upper arms. These inflammatory papules are not uncommonly quite monomorphic and frank pustule formation is not common. Pruritus and the absence of comedones and facial lesions distinguish it from acne. This condition is more frequent in tropical countries and in summer in temperate regions and has been associated with

antibiotic treatment e.g. tetracyclines, corticosteroids and immunosuppression associated with organ transplantation. Scrapings or biopsy specimens show abundant *Malassezia* yeasts occluding the opening of the infected follicles. However, as the colonisation of hair follicles by *Malassezia* is not abnormal, the diagnosis of Malassezia folliculitis has to be confirmed by the response to antifungal therapy: topical treatment is effective in most cases, whereas others need systemic therapy with azole or triazole.

Mould Infections

Mould is ubiquitous in the environment, but pure mould infection of the skin is very uncommon. Superficial skin infection may mimic moccasin tinea pedis, tinea manuum, and tinea unguium.

Principles of Laboratory Diagnosis of Superficial Fungal Infections

Clinical diagnosis is usually good enough for the routine management of patients. If laboratory confirmation is deemed required, the following principles should be borne in mind. As cutaneous diseases associated with these fungi may or may not have genuine tissue invasion, and skin surface is the habitat of some of these fungi and the skin surface is liable to environmental contamination, mere isolation of some of these fungi from clinical specimens taken from the skin surface is not a *sine qua non* of their role in disease causation. The laboratory approach to these cutaneous conditions may involve answering the following 3 questions: 1) what is the purpose of performing the laboratory tests under consideration? 2) which is the most appropriate laboratory test? 3) how to interpret the laboratory results in the concerned clinical context? To better inform the laboratory microbiologists, it is prudent to provide the essential clinical information and specify the organisms of interest on the request form. The laboratory diagnostic approach will involve 1) wet mount KOH examination that can be performed rapidly at the "bed-side" with or without staining (e.g. by Parker's blue black ink, chlorazole black), 2) culture for proper species identification.

The scales from active lesions produced by skin scraping can be collected and wrapped in colour paper (and put in a properly sealed container) and sent to the supporting laboratory by mail. Cleansing of the site may be required in those grossly contaminated sites such as from a "dirty" foot before performing skin scraping.

Diseased hairs should be plucked (not cut) in those cases of suspected tinea capitis. Scraping of diseased scalp skin for fungal study is the recommended approach of the British Association of Dermatologists.[2] Specimen collection by cytobrush or toothbrush are alternative methods of sample collection especially in the context of outbreak investigation.[2, 3] As aforementioned, species identification in tinea capitis is preferred.

Subungual hyperkeratotic material should be collected with a curette in those cases of suspected onychomycosis. Sampling should also be collected as proximal as possible in those cases of clinical distal lateral subungual onychomycosis. Simple nail clipping of the distal diseased nails may not give the maximum yield. Repeated sampling is sometimes required to isolate the causative fungi. Two types of media, one with and the other without cycloheximide, are ideally used to culture nail samples.[4] The one with cycloheximide suppresses the growth of the non-dermatophyte filamentous fungi and hence enhances the growth of the dermatophytes. Whereas the one without allows the growth of the non-dermatophyte filamentous fungi, some of which may sometimes cause skin diseases. Therefore a positive culture for non-dermatophyte filamentous fungi should be interpreted with care and correlated with the clinical features of the patient.

Laboratory diagnosis of *Candida* infection can also be made with the help of Gram staining of relevant clinical samples. Demonstration of yeasts with pseudohyphae formation is regarded as pathognomic of tissue invasion and hence genuine clinical disease. Fungal cultures can be performed but results have to be interpreted in clinical context.

As *Malassezia* species can be detected in many asymptomatic individuals and only cause disease when these yeasts are in certain phases of growth or metabolism, the best way to establish the diagnosis is by clinical examination (which can be assisted by Wood's light). Diagnosis can also be made with the help of KOH wet mount examination of the scales. The classic spaghetti and meat ball pattern can be demonstrated by microscopic examination of the wet mount. Fungal culture is not indicated for the diagnosis of superficial infections caused by this yeast.

Commercial kits with colour indication for positivity have been developed recently to make fungal cultures possible in the physician's office. Molecular diagnostics are also developed to increase the sensitivity of laboratory tests for tinea unguium.[5] These technological advances will certainly facilitate the clinical management of cutaneous fungal infections in the community based clinical settings.

Treatment

Pharmacological treatments for superficial fungal infections can be grouped into topical and systemic. Generally speaking, imidazoles (isoconazole, tioconazole, clotrimazole) and the triazoles (itraconazole, fluconazole) are active against yeast and dermatophytes, topical allylamines (terbinafine, naftifine) and amorolfine may be fungicidal, polyenes (nystatin, amphotericin B) are active against *Candida* sp but not dermatophytes; systemic terbinafine albeit thought to be fungicidal to dermatophytes does not work for yeast infections. With the exception of tinea captitis and tinea unguium which (except for those cases with only mild distal disease) requires systemic treatment, topical treatment may be tried in most other superficial fungal infections. Systemic treatment may also be considered in those cases with extensive disease or significant hyperkeratosis. The treatment course for topical treatment spans from as short as 1 week to 4-6 weeks. In the real life situation, a longer course is not uncommonly required. Systemic regimes are summarised in the following table.

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Summary of systemic treatment of superficial fungal infections

Disease	Systemic		Duration	Remarks
Tinea capitis	Griseofulvin	500-1000 mg once daily or in divided doses (adults) 15-20 mg/kg once daily or in divided doses (children, under 50 kg)	as long as necessary, usually 3 to 4 months in ectothrix and 2 months in endothrix infections	Only drug approved by FDA for children To take with food
	Terbinafine	250 mg daily (adults) 62.5 mg daily (10-20 kg, over 1 year) 125 mg daily (20-40 kg) 250 mg daily (>40 kg)	2–4 weeks	Superior to griseofulvin in *T. tonsurans*, similar efficacy against *T. violaceum*, less efficacious than griseofulvin for *M. canis*
	Itraconazole	200 mg daily 5 mg/kg daily (children)	4–8 weeks	Potential drug interactions, related to heart failure
	Fluconazole	200 mg daily 6 mg/kg daily (children)	3–4 weeks 2 weeks	Use limited by side effects
	Ketoconazole	200 – 400 mg daily 5 mg/kg daily	4-6 week	Use limited by hepatotoxicity
T. corporis	Griseofulvin	500 – 1000 mg daily	2-4 weeks	
	Terbinafine	250 mg daily	1-2 weeks	
	Itraconazole	100 mg daily 200 mg daily	2 weeks 1 week	
T. cruris	Griseofulvin	500 -1000 mg daily	2-4 weeks	
	Terbinafine	250 mg daily	2 weeks	
	Itraconazole	100 mg daily 200 mg daily	2 weeks 1 week	
T. manuum	Terbinafine	250 mg daily	2 weeks	
	Itraconazole	100 mg daily 200 mg bd 200 mg daily	30 days 1 week 1 week	
T. pedis	Griseofulvin	750-1000 mg daily	4 - 8 weeks	
	Terbinafine	250 mg daily	2 weeks	
	Itraconazole	200 mg daily 200 mg bd	2 weeks 1 week	
T. unguium	Griseofulvin	750-1000 mg daily	6 – 12 months (fingernail) 12 – 18 months (toenail)	FDA approved; cure rate: ~30%
	Terbinafine	250 mg daily	6 weeks (fingernail) 12 – 16 weeks (toenail)	FDA approved; cure rate: ~80%; most effective in dermatophyte infections; serious side effects in less than 1% of patients; monitor LFT at 4 to 6 weeks
	Itraconazole	200 mg daily 200 mg bd (pulse dosing)	6 weeks (fingernails) 12 weeks (toenails) 1 week, to repeat after 21 day interval, finger nails: 2 courses; toenails: 3 courses	FDA approved; effective in infections caused by dermatophytes, yeasts and moulds; side effects diminished when taken as pulsed doses; monitor LFT if used more than 1 month
	Fluconazole	100 – 200 mg daily 150 – 400 mg/week		Less effective in dermatophytes than in yeasts
	Ketoconazole	200 – 400 mg daily	≥6-12 months	More effective for *Candida* than dermatophytes; highest incidence of LFT abnormalities
Candidiasis	Fluconazole	200 mg once followed by 100 mg daily	2-3 weeks	
	Itraconazole	100 mg daily or twice daily	2 weeks	
	Ketoconazole	200 mg daily or twice daily	1-2 weeks	
Pityriasis versisolor	Itraconazole	400 mg stat 200 mg daily	7 days	
	Fluconazole	400 mg stat		
	Ketoconazole	400 mg stat 200 mg daily	10 days	
Malassezia folliculitis	Itraconazole	200 mg daily; increase to 400 mg daily if clinically indicated Paediatric <2 years: Not established >2 years: 3.3-6.6 mg/kg/d once	discontinue when lesions resolve	relapse almost always occurs when treatment is withdrawn, topical ketoconazole is indefinitely continued after successful initial treatment with oral medication

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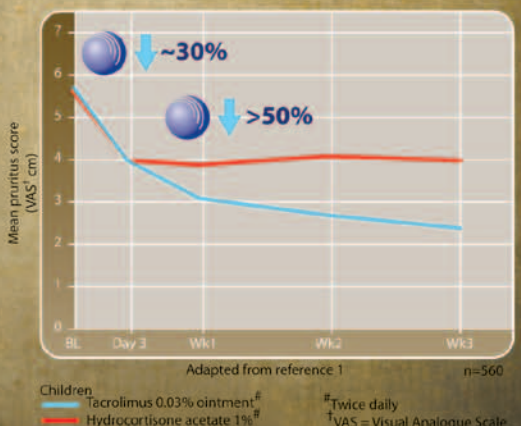

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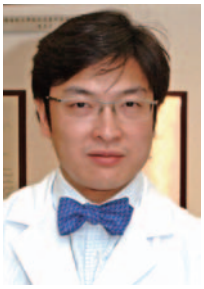
Recent Advances in Cosmetic Dermatology in Asians



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Abstract

Asians differ from Caucasians as photoageing tends to present with pigmentary changes as the main issue. Furthermore, with a higher epidermal melanin context, there is a greater risk of complications especially post-inflammatory hyperpigmentation and such high epidermal melanin context also acts as a competing chromophobe for the underlying target. In this review article, recent advances in the area of skin rejuvenation in Asians as well as new technologies for skin tightening and body contouring will be discussed.

Introduction

The global financial crisis in 2008 did have an impact to the development of medical devices in cosmetic dermatology. Many companies merged to reduce cost and some unfortunately went into bankruptcy (such as Rhytec-the company that developed plasma skin rejuvenation technology). Furthermore, most companies delayed the introduction of new technologies in such time of uncertainty which also implies that there are relatively fewer recent advances in cosmetic dermatology. Nonetheless, there are several new developments and this article will discuss such development in the treatment of acquired pigmentary disorders; fractional resurfacing; non-invasive skin tightening and body contouring.

Treatment of Acquired Pigmentary Disorders

Freckles and Lentigo

Many lasers and light sources can be used for the treatment of freckles and lentigines. Q-switched (QS) lasers employ quality switching, a technology that involves the use of an electromagnetic switch to abruptly stop the laser from passing through the cavity and when the blockage is then suddenly removed, laser pulses with extreme short durations (in the nanosecond range) and high energy (1,000,000 W/cm^2) are produced. QS lasers can be most effective for the treatment of freckles and lentigines especially in light skin patients. However, previous studies indicated that post-inflammatory hyperpigmentation (PIH) can occur among Asians as QS lasers produce excessive tissue response due to their high energy and therefore resulted in greater degree of inflammation which in Asians is translated to a higher risk of PIH[1,2]. To reduce such risk, long pulsed pigment laser and intense pulsed light source have been used and lower risk of PIH can be obtained. Recently, the technique of compression window to empty the blood vessels and in doing so, reduce the risk of purpura has been advocated[3]. The purpose is to reduce haemosiderin deposition that can arise as a result of purpura post-laser treatment. Haemosiderin deposition can also contribute to the appearance of post-inflammatory hyperpigmentation in Asians. More recently, the concept of contrast between lesional and non-lesional skin in the treatment of lentigines among skin of colour is introduced. Unlike, light-skin patients whereby such contrast is great, complication is uncommon. However, for Asians or skin of colour when such contrast is low then issues occur especially when one uses a large spot size device. To avoid complications with a large spot size device, the operator has to reduce the fluence and often clinical efficacy is compromised. If the operator wishes to obtain better efficacy and push up the fluence, above threshold injury can take place leading to rather undesirable effects. Such hypothesis was validated in a recent retrospective study looking at forty Chinese subjects with lentigines treated with four different devices[4]. The long pulsed pigment laser with a compression window and small spot size achieved the greatest degree of improvement and least complication (Figure 1).

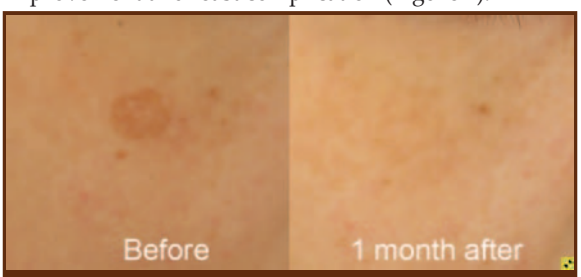

Figure 1: 1month after 2 monthly treatments with long pulsed KTP 532nm laser (14J/cm2;2mm;2ms)

Melasma

Treatment of melasma remains the main challenge and while low fluence large spot size QS 1064nm Nd:YAG laser, otherwise known as laser toning or laser facial, had been advocated to be effective in the treatment of melasma, it is not without adverse effects. A recent article that has been accepted to be published reported 14 Hong Kong women that developed punctate depigmentation as a result of frequent treatments with this laser[5]. Looking at two recent published articles from Thailand and Korea, the risk of such complication is about 10% (5 out of 47 patients) after about 6 treatment sessions when one combined the

results of both studies [6,7]. Clearly, laser toning should be considered as a second line therapy and only if patients are properly informed regarding potential complications.

Fractional resurfacing has also been used for the treatment of melasma and the new 1927nm thulium laser is being explored as a better option for the treatment of melasma. Our own experience, however, failed to confirm such observation and further studies to determine the optimal parameters are necessary before 1927nm thulium laser can be considered to be the treatment of choice for melasma.

Non-invasive Skin Tightening

Non-invasive skin tightening remains a popular cosmetic procedure given the lack of down time and complications associated with such procedures. Non-invasive skin tightening involves the delivery of energy source deep into 2-4mm of the dermis inducing collagen damage. The subsequent healing response can lead to skin tightening. There are several new developments in recent years that are worthwhile to mention.

Mono-polar radiofrequency has been used for non-invasive skin tightening since the last decade and is considered to be the gold standard for non-invasive skin tightening. Mono-polar radiofrequency involves the use of cooling to protect the epidermis and delivery of radiofrequency energy deep into the skin by attaching one electrode to the handpiece and the other to the trunk. The main drawback with this procedure is the pain associated which was considered to be severe among 5% of the treated subjects[8]. The third generation mono-polar radiofrequency device was designed with the intention to reduce such pain. By incorporating a vibration handpiece into the system, it reduces pain based upon the gate theory. By creating vibrations, the neurological system is confused as both the pain and vibration senses are transmitted through the same neural fibre. Another new development is to divide the radiofrequency pulses into five with cooling pulses in between and once again the intention was to fool the neurological system. To achieve greater efficacy, the radiofrequency delivery was changed to allow more even distribution. We have been using this new model for the last 12 months and in our experience, this device is more superior than the previous model and that patients are experiencing less pain without any compromise in clinical efficacy.

Focused ultrasound is another new technology that obtained United States Food and Drug Administration (US FDA) approval last September for eyebrow elevation. This device utilised a see-and-treat approach whereby the probe allows imaging of the tissue before application of the focused ultrasound, the energy of which is to be translated into thermal energy, deep into the target zone[9]. In an Institutional Review Board (IRB) approved study (Western IRB, Seattle, Washington, USA), we conducted a company sponsored trial (Ulthera, Arizona, USA supported and provided indemnity) and examined thirty one Chinese patients for skin tightening. Efficacy and complications were assessed by two blinded observers as well as subjective structured questionnaires. Our data indicated significant improvement in the lower face with transient adverse effects including occasional bruises, mild oedema and transient dermal papules[10]. While such results are promising, optimal parameters to achieve consistent clinical outcome and long term data are lacking. As a result, it cannot yet replace mono-polar radiofrequency as the gold standard for non-invasive skin tightening.

Fractionated Technologies

The concept of fractional photothermolysis has revolutionised the development of cosmetic technologies and many new devices are still being developed based upon this concept of inducing microscopic areas of injury with healthy tissue in between and thereby, allow rapid healing to take place.

The role of 1927nm thulium laser in the treatment of melasma has already been discussed. The advantage of this laser is that it has a water absorption coefficient that is 10 times that of 1550nm allowing more superficial thermal injury with up to 70% of the skin being removed during a single session. In our experience, in combination with 1550nm Erbium laser for skin rejuvenation, the dual approach can lead to more rapid results in skin rejuvenation and therefore better patient satisfaction (Figure 2).

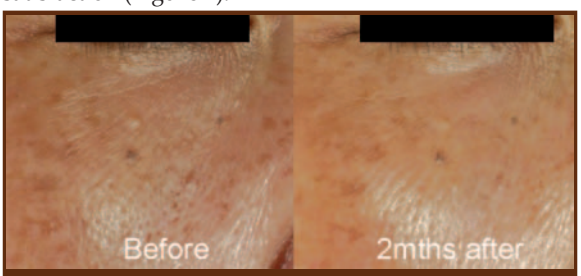

Figure 2. Skin rejuvenation after one treatment session with dual wavelength 1550nm and 1927nm lasers

In fractionated bipolar radiofrequency, multiple electrodes are mounted on the tip of the handpiece. Passage of radiofrequency creates epidermal and dermal injuries with epidermal injury occurring first and then dermal damage as a subsequent event. By alternating the parameters of the radiofrequency, sublative effects can be induced whereby there is more dermal damage with minimal epidermal disruption[11]. The advantage of sublative rejuvenation is the lower risk of post-inflammatory hyperpigmentation especially in Asians. In our study that looked at 12 acne scar subjects treated monthly with this device, our preliminary data indicated significant improvement of acne scar with PIH rate of 1.9%, which is lower than previously reported PIH rate among patients treated with non-ablative fractional resurfacing.

Minimally invasive radiofrequency needles involve the insertion of radiofrequency needles into the skin and in doing so, induce significant focal thermal injury deep into the skin. Early results suggested it can cause skin tightening comparable to 37% of that obtained in a surgical facelift[12]. This is indeed impressive and the commercial device should be available in Hong Kong in October 2010. The main issue is pain and post-

inflammatory hyperpigmentation and further study in our local population is necessary to determine its efficacy and risks.

Body Contouring

In August 2010, US FDA made history by approving two devices for non-invasive body contouring. Zeltiq (Zeltiq, Pleasanton, CA, USA) was approved for non-invasive fat layer reduction and Zerona (The Erchonia1 LipoLaser, manufactured by Erchonia Medical, Inc. McKinney, TX, USA), for reduction of the circumference of various body parts.

Cryolipolysis (Zeltiq, Pleasanton, CA, USA) is a novel technology that utilises heat extraction to remove energy from the skin and fat layer, cooling the tissue gradually and lowering the temperature at the subcutaneous layer to 4-8 C[13]. This changes the triglyceride from liquid to solid state (triglyceride at body temperature is in a liquid state) and by maintaining such change for an hour, upon subsequent removal of the device, the triglyceride returns to the liquid state. Such alternation has been hypothesised to cause the adipocytes to go into apotosis which occurs gradually over a 2 to 6 months period. A recent study indicated a reduction of 20% of the fat layer after 2 months and 25% after 6 months[14]. Adverse effects are mild and transient including bruises due to the suction applicator, mild numbness that lasted for an average of 3 weeks and slight abdominal discomfort. Safety studies looking at the liver function and fasting lipid indicated no significant changes after the procedure and since its commercial launch in mid 2009 initially as an off label use (it was approved by US FDA for treatment of cellulite before current approval for fat reduction was obtained), cryolipolysis had been shown to be effective in localised fat reduction[15]. Our own data indicated after a single treatment, 70% of the treated subjects found the results to be satisfied to very satisfied with 81% reported noticeable difference in the treated area (Figure 3).

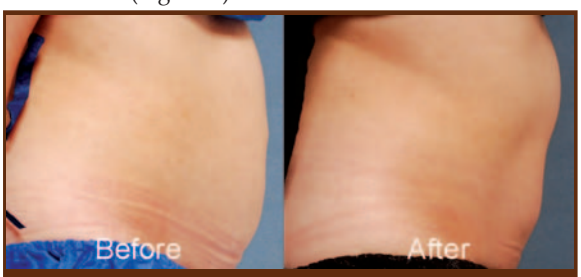

Figure 3: 2 months after treatment with Zeltiq around the abdomen and waist

Low energy laser (Zerona, Erchonia Medical, Inc. McKinney, TX, USA) was granted US FDA approval for non-invasive body contouring and was approved for reduction of the circumference of various body parts. The concept derived from the fact that in an in vitro setting upon exposure to laser at 635 nm with an output of 10mW, adipocytes release 90% of triglyceride after 6 minutes of exposure[16]. The commercial device consisted of five 635nm diode lasers, 17mW output, each with a scanner. Patients then underwent a 40 minutes treatment session with the laser scan to the body at a randomised manner. In a double blinded controlled placebo clinical trial that looked at 67 subjects who were randomised to receive either treatment with the device or sham treatment (same lasers but with output of only 2.5mW), after 6 treatment sessions within a two week period, there was significant reduction of total circumferences measured across the waist, hip and bilateral thighs[17]. It was such kind of data that led to its FDA approval. However, in the author's opinion there are several issues that need to be considered before readers rush out to purchase this device. First of all, tape measurement can be very inconsistent and to achieve repeatability, is very difficult. Second of all, an in vitro experiment differs significantly from an in vivo one. For the low energy laser to penetrate through the epidermis and the dermis and then affect the subcutaneous tissue is by no means easily obtainable especially among skin of colour given the high epidermal melanin context. Unlike light skin individuals whereby an optical window exists between 600 nm to 1000nm (and light source can penetrate deeper), melanin has a much wider absorption spectrum and for skin of colour, such epidermal melanin context will prevent any such kind of optical windows to exist[18]. Finally, unlike in the US whereby the power output of a red laser pointer (635nm) is limited to 5mW, in other countries including Hong Kong, one can get high output laser pointers at a very reasonable cost. In fact, for 100mW 635nm diode laser pointers (almost 5 time the output of this device), the cost is about US$ 70. As a result, if such low energy laser is effective in non-invasive body contouring, then one can just give a powerful laser pointer to the patient to do it at home! Those particularly interested in this technology should also read the acknowledgement section (one of the most lengthy piece the author has ever come across) of the paper and draw their own conclusions regarding the study's findings.

Conclusion

In conclusion, there are many new cosmetic procedures for the treatment of pigmentation, skin rejuvenation and body contouring. With the appropriate parameters, safe and effective treatments can be obtained in Asians. Careful assessment of the clinical data especially in their application to skin of colour is necessary before physicians purchase such device in their practice for clinical use.

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Upcoming Certificate Courses of the Federation of Medical Societies of Hong Kong

Date	Course No	Course Name	Target Participants	CME/CNE
12 Nov 2010 - 3 Dec 2010	C169	Certificate Course on Assessing and Managing Violent Patients/People in the General Health Care Settings	Healthcare Professionals	10 CNE Points / CME Accreditation in application
15 Nov 2010 - 20 Dec 2010	C167	Certificate Course on Sports Medicine and Emergencies	Medical and Health Professionals	9 CNE Points / CME Accreditation in application
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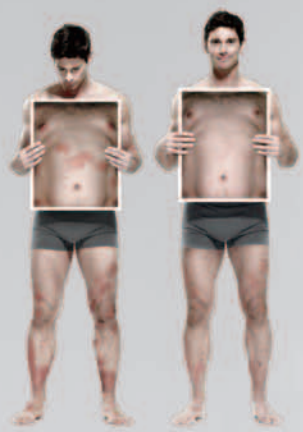

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HUMIRA® is indicated for the treatment of moderate to severe chronic plaque psoriasis in adult patients who failed to respond to or who have a contraindication to, or are intolerant to other systemic therapy including cyclosporine, methotrexate or PUVA.[2]

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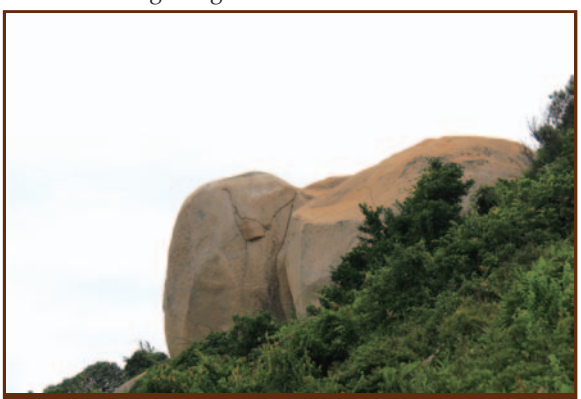
What Do They Look Like?

Dr. Chi-keung KWAN

MBBS(HK), MRCP(UK), FHKCP, FHKAM(Medicine)
Specialist in Dermatology & Venereology

Dr. Chi-keung KWAN

Time really flies. If you have a good memory, you should remember that I introduced to you three hiking pathways two years ago in the Medical Diary. One was the Hong Kong Trail (港島徑) from Shek O Road (石澳道) to Big Wave Bay (大浪灣). One was the MacLehose Trail (麥理浩徑) from Long Ke Wan (浪茄灣) to Pak Tam Au (北潭坳) and the last one was the Lantau Trail (鳳凰徑) from Ngong Ping (昂坪) to Lantau Peak (鳳凰山). Have you ever tried any one of them? This time, I bring you to see the distinctive and interesting rocks on the outlying islands of Hong Kong.

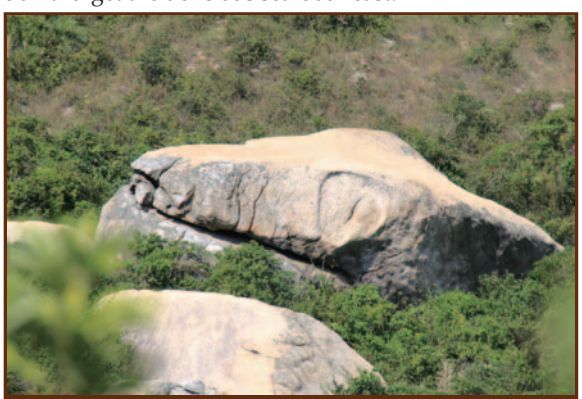


What do they look like? - Rocks from Cheung Chau

Cheung Chau Island (長洲)

Let's start with the easiest pathway first. Cheung Chau is a very famous dumb-bell island. It is in the Southwest of Hong Kong. I am sure that most of us has been there for leisure and enjoying the delicious seafood. Besides the beautiful beaches and one Olympic champion, there are wonderful scenery and peculiar rocks. Getting off the ferry, we go to the northern part first by turning left. We take the Cheung Kwai Road (長貴路) to Tai Kwai Wan (大貴灣). Afterwards, we ride up a small hill following the Cheung Pak Road (長北路). In around 20-minutes' walk, we climb a short flight of stairs on the left and will soon see the Lizard Rock (蜥蜴石) and Eagle Rock (麻鷹石). There are also other amazing rocks that we may name with imagination.

We go down to Tung Wan Tsai (東灣仔) which is a small, quiet and beautiful beach. Then, walk up to Pak Kok Tsui (北角咀), the highest pavilion. If the sky is clear, Lamma Island, Pok Fu Lam, QMH, and the Tsing Ma Bridge are all caught in our eyes. Going down we will reach Tung Wan (東 灣). It is a renowned beach in Cheung Chau. The St. John Hospital (長洲醫院) will be on the right. After passing a hotel and Kwun Yam Wan (觀音灣), we begin to climb up a small hill, around 100 metres, to a pavilion, Chi Ma Hang (芝麻坑). Not far away, we can follow the Small Great Wall (小長城) and reach the "Vase Rock" (花瓶石) and "Face Rock" (人頭石). Honestly speaking, the "Vase Rock" does not look like a vase at all to me; in contrast, the "Face Rock" is really similar to a human face. Of course, we need to look at the rock from a particular angle. We can see the eyes, eyebrows, nose and mouth clearly. We continue walking along the southern coast of Cheung Chau and can enjoy the infinite sea view. In around 45 minutes, we will see the sign to Pak Cho Wan (白鱈灣). We follow the concrete path and will reach a small rock forest. We can then walk under the huge rocks. Although the rock forest is really small and it takes only 3 minutes to walk pass, the fantastic scenery makes me feel like being in Guilin (桂林). Last but not least, the Cheung Po Tsai Cave (張保仔洞) will be at the end of the route. Most of us are very familiar with the historical background of the Cheung Po Tsai Cave. However, I do not believe Cheung Po Tsai would store the treasures and jewellery in such a small and narrow cave. Afterwards, we can walk back to the pier and enjoy the magnificent sunset. Before going back to the city, don't forget the delicious seafood feast.



What do they look like? - Rocks from Cheung Chau

Poi Toi Island (蒲台島)

Poi Toi Island is one of the most fascinating places to view peculiar rocks in Hong Kong. It is situated at the southern part of Hong Kong and is opposite Stanley. We take a Kai To (街 渡) at the Aberdeen pier. Within around one-hour journey, we arrive at the Tai Wan Pier (大灣碼頭). We walk up the hill gradually along the

Poi Toi Country Trail (蒲台郊遊徑) and then reach the demolished Mo's House (巫氏古屋). While continuing our journey, we need to pay particular attention because the coffin rock (棺 材 石) is nearby and it is very easy to be missed. The coffin rock is like a coffin floating on the trees. We then meet a large turtle (靈 龜 石) slowly climbing towards a monk(僧人石). Not far away, there is a Buddha's palm (佛 手 岩). We can see each finger clearly. We can have lunch on the Island. This pathway is easy but we need to pay attention to the Kai To schedule.

Tung Ping Chau (東平洲)

For seeing interesting and peculiar rocks, everyone must agree that Tung Ping Chau is worth to go. Tung Ping Chau is a small crescent-shaped island. It is formed mainly by sedimentary rocks. Since she is at the far eastern part of Hong Kong facing the Pacific Ocean, there are lots of distinctive sea platforms, sea stacks and sea cliffs. We can spend one day walking around the island. It takes one and a half hour to Tung Ping Chau by ferry from Ma Liu Shui (馬料水) near the University Station.

After getting off the ferry, we turn left to Kang Lau Shek (更樓石). The Kang Lau Shek (更樓石) are two unique sea stacks facing the Pacific Ocean. They are about 7 metres tall and most of us climb up to the roof of the rocks to take photos though it may have potential danger. 更樓 means the tower for security guarding. Not far away lies the Lan Kwo Shui (難過水). 難過水 means difficult to pass the water. It is one of the most typical and impressive sea wave-cut platforms in Hong Kong. It can be fully viewed during low-tide period but completely covered at high-tide. I strongly recommend you NOT to pass Lan Kwo Shui. As the name suggests, the rough sea and strong wave make it very difficult and extremely dangerous to pass. Actually, there is a pathway at the top of the cliffs of Lan Kwo Shui. We can get past by using this pathway. Walking along the pathway, Lung Lok Shui (龍落水) is in front of us. Lung Lok Shui (龍落水) means a dragon going into the sea from the sky. Continuing our trip, here comes Cham Keng Chau (斬頸洲). Cham Keng (斬頸) means chopping off the neck. The name sounds horrible. The waves cut along the weakest point of the island penetrating along the landscape. Continuous erosion of the rocks leads to the collapse of the landscape forming a channel with the cliffs on both sides. People can walk through the "chop wound" of the neck to see the miracle of nature. Afterwards, we go back to the pier to enjoy the wind breezing and the beauty of the coast while waiting for the return ferry. I am sure that it must be a very fruitful geography lesson visiting Tung Ping Chau.

This time, we see a lot of interesting, peculiar and distinctive rocks and landscapes. There are still many interesting landscapes in Hong Kong which are worth visiting. It is boring to read the texts. So, don't hesitate, let's set off now.

FMSHK Charity Concert for Bereaved Children

Hong Kong Academy for Performing Arts
Sept 19, 2010

This year marks the 45th Anniversary of the Federation and our affiliated HKFMS Foundation has set up a charity fund, towards the mission of helping the bereaved children who have lost their parents through tragedy and illnesses.

On Sept 19, 2010, our Charity Concert and Launching Ceremony for the Charity Project were successfully held. We were blessed with vey encouraging response and feedback, with kind donations from many parties with hearts of gold. The evening was made most memorable, with the magnificent violin performance by our Charity Project Ambassador, Ms. Takako Nishizaki, together with the superb piano performance by Ms. Amy Sze and Mr. Phoebus Chan. We were also privileged to have wonderful performances by renowned guest performers, Dr. David Fang and Dr. Jonathan Yuen from the medical community. Delightful performances by Mr Eric Wu and the musical students of the Hong Kong Academy for Performing Arts, added to the flavour of East meets West. Above all, we were most honoured to have Professor Gabriel Leung, Under Secretary for the Food and Health Bureau, to be our officiating guest and to deliver a special performance. The programme of course was made complete by our Master of Ceremony, Ms Annie Yip Liu On-lai. We would like to acknowledge the Academy for Performing Arts as our supporting organisation, and finally millions of thanks are due to our patrons, donors, concert audiences and organising committee too for their kind sponsorship, donation, participation and effort. The Concert would not have been a success without all your support.

Our next coming event for bereaved children is a recreational activity - Cathay City Visit on Nov 27, 2010. We are hopeful that around 40 bereaved children and their guardians will have the opportunity to enjoy this exciting tour. Special thanks to Cathay Pacific for their kind reception. We welcome your referral of bereaved children and youngsters who are interested to join this event. Please contact the FMSHK Secretariat on 2527-8898 for details. Further announcements of our project events will follow.

The Federation 45 Annual Dinner 2010

31st December, 2010 (Fri)

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The Hong Kong Academy of Medicine Jockey Club Building

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Seminar on Doping Control in Sports

Jointly organised by

The Federation of Medical Societies of Hong Kong

Sports Federation & Olympic Committee of Hong Kong, China

Hong Kong Anti-Doping Committee

Objectives:

This one-day seminar aims to provide anti-doping knowledge to local physicians and to increase their awareness of the roles and responsibilities they have in the fight against doping. After attending the course, the attendees will be equipped to help their patients who happen to be an athlete. The course will also update the participants on the latest development in the fight against doping, both in terms of international coordinated efforts and testing technology.

Time	Programme	Speaker
9:00 am	Registration	
9:30 am	Opening ceremony	
9:45 am	The international efforts in the fight against doping in sport *	Ms. Michele VERROKEN, (Former Director of Ethics and Anti-Doping at UK Sport)
10:45 am	Tea break	
11:00 am	The prohibited list and the role of physician in doping control #	Dr. Julian CHANG, MH (Hon. Medical Advisor of SF&OC)
12:30 pm	Q&A	
12:45 pm	Lunch break	
2:00 pm	Therapeutic use exemption (TUE) and doping control procedure #	Dr. James LAM (Member of TUE Panel, HKADC)
3:30 pm	Tea break	
3:45 pm	An overview on the testing technology for doping control #	Dr. Terrence WAN, PhD (Head of Racing Laboratory, Hong Kong Jockey Club)
4:45 pm	Q&A	
5:00 pm	Closing ceremony	

Date: 5 December 2010
Time: 9:30 am - 5:30 pm
Venue: Lecture Theatre, Olympic House, 1 Stadium Path, So Kon Po, Causeway Bay, Hong Kong
Language: * English; # Cantonese (Supplemented with English)
Parking Fee: HK$180 ; **Reservation Deadline:** 5 November 2010 (Optional)
Registration Fee: HK$300
Certificate: Award to participants with a minimum attendance of 70%
Enquiries: The Secretariat of The Federation of Medical Societies of Hong Kong
Tel : 2527 8898 Fax : 2865 0345 Email : info@fmshk.org

CME / CPD Accreditation in application

A total of **9 CNE** points for the whole course and the points will be awarded according to the number of hours attended.
Application form can be downloaded from websites: http://www.fmshk.org

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
	★HKMA Choir Voice Training Course 2010 **1**	★FMSHK Officers' Meeting ★HKMA Council Meeting **2**	★HKMA Orchestra Rehearsal ★HKMA Trailwalker Final Briefing Session **3**	★HKMA New Territories West Community Network – Clinical Evidence of Quadrivalent HPV Vaccine in Adult Women (>aged 26) ★MPS – Mastering Your Risk **4**	★Joint Surgical Symposium – Recurrences in Thyroid Carcinoma ★HKMA – Shatin Doctors Network – Management of Acne Vulgaris with New Fixed-Dose Combination Treatment **5**	**6**
★HKMA Sports Day ★MPS – Mastering Adverse Outcomes ★HKMA Tennis Tournament **7**	★HKMA Choir Voice Training Course 2010 **8**	★HKMA Kowloon East Community Network – Update on Rotavirus Epidemiology and Rotavirus Vaccine ★HKMA Kowloon West Community Network – "Management of Chronic Cough in Children: Case Series in a Community Clinic" **9**	★HKMA Orchestra Rehearsal ★HK Neurosurgical Society Monthly Academic Meeting – Information Technology in Neurosurgery ★HKMA Central, Western & Southern Community Network – Certificate Course on Orthopaedics (III) **10**	★HKMA CME Series on Chronic Hepatitis B and its Complications (NT West District) ★HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2010 – Common Eye Diseases of Children **11**	★HKMA – Shatin Doctors Network – A New Frontier in Hypertensive Management **12**	★Refresher Course for Health Care Providers 2010/2011 ★HKMA Kowloon East Community Network – Joint CME Course for Health Personnel 2010 on "Update on PCOS" **13**
★HKMA Certificate Course on Family Medicine 2010 ★HKMA Tennis Tournament **14**	★HKMA Choir Voice Training Course 2010 **15**	**16**	★HKMA Hong Kong East Community Network – PPI on "Hypertension" (PENDING) **17**	★HKMA CME Series on Chronic Hepatitis B and its Complications (Kowloon District) ★HKMA New Territories West Community Network – Lipid Disorder (PENDING) ★FMSHK Executive Committee and Council Meeting ★FMSHK and HKFMS Foundation Annual General Meeting **18**	★Oxfam Trailwalker 2010 **19**	★Oxfam Trailwalker 2010 **20**
★HKMA Tennis Tournament ★HKMA 90th Anniversary Football Cup ★Oxfam Trailwalker 2010 **21**	★HKMA Choir Voice Training Course 2010 **22**	★HKMA Kowloon West Community Network – Sleep Apnoea Syndrome **23**	★MPS – Mastering Adverse Outcomes ★HKMA Central, Western & Southern Community Network – Certificate Course on Orthopaedics (V) **24**	★HKMA New Territories West Community Network – Mood (PENDING) **25**	★HKMA Hong Kong East Community Network – Rotavirus Vaccination: Why Parents Should Consider It **26**	★MPS – Mastering Adverse Outcomes ★HKMA Trailwalker Reunion Party ★The 3rd Hong Kong International Burns and Wound Healing Symposium **27**
★MPS – Mastering Adverse Outcomes ★HKMA Tennis Tournament ★The 3rd Hong Kong International Burns and Wound Healing Symposium **28**	★HKMA Choir Voice Training Course 2010 **29**	★9th Asia-Pacific Conference on Human Genetics **30**				

Date / Time	Function	Enquiry / Remarks
1 MON 8:00 pm *(8,15, 22,29)*	**HKMA Choir Voice Training Course 2010** Organiser: The Hong Kong Medical Association, Venue: GP1, HKCC	Ms. Candy YUEN Tel: 2527 8285
2 TUE 8:00 pm – 10:00pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. K CHOI, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Christine WONG Tel: 2527 8285
3 WED 8:00 pm *(10)*	**HKMA Orchestra Rehearsal** Organiser: The Hong Kong Medical Association, Venue: Pui Ching Academy	Ms. Candy YUEN Tel: 2527 8285
	HKMA Trailwalker Final Briefing Session Organiser: The Hong Kong Medical Association, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Peony CHAN Tel: 2527 8285
4 TUE 1:00pm	**HKMA New Territories West Community Network – Clinical Evidence of Quadrivalent HPV Vaccine in Adult Women (>aged 26)** Organiser: HKMA New Territories West Community Network, Speaker: Dr. Nelson Shing-shun SIU, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building,10 Sai Ching Street, Yuen Long, N.T.	Miss Alice TANG Tel: 2527 8285 1.5 CME Points
6:00 pm	**MPS – Mastering Your Risk** Organiser: The Hong Kong Medical Association, Speaker: Dr. Ka-lam HAU, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Viviane LAM Tel: 2527 8452 2.5 CME Points
5 FRI 8:00 am – 9:00 am	**Joint Surgical Symposium – Recurrences in Thyroid Carcinoma** Organisers: Department of Surgery, The University of Hong Kong & Hong Kong Sanatorium & Hospital, Chairman: Dr. Angus C.W. CHAN, Speakers: Dr. Brian LANG & Dr. Kai-Pun WONG, Venue: Hong Kong Sanatorium & Hospital	Department of Surgery, Hong Kong Sanatorium & Hospital Tel: 2835 8698 Fax: 2892 7511 1 CME Point (Active)
2:00 pm	**HKMA – Shatin Doctors Network – Management of Acne Vulgaris with New Fixed-Dose Combination Treatment** Organiser: HKMA – Shatin Doctors Network, Speaker: Dr. Ka-lam HAU, Venue: Level 2, Jasmine Room, Royal Park Hotel, Shatin, N.T.	Dr. Wing-kin MAK Tel: 2649 4466 1 CME Point
7 SUN 1:30 pm	**HKMA Sports Day** Organiser: The Hong Kong Medical Association, Venue: Stanley Ho Sports Centre	Miss Peony CHAN Tel: 2527 8285
2:30 pm *(24,27,28)*	**MPS – Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association, Speakers: Various, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Viviane LAM Tel: 2527 8452 2.5 CME Points
7:30 pm *(14,21,28)*	**HKMA Tennis Tournament** Organiser: The Hong Kong Medical Association, Venue: Kowloon Tong Club	Miss Peony CHAN Tel: 2527 8285
9 TUE 1:00 pm	**HKMA Kowloon East Community Network – Update on Rotavirus Epidemiology and Rotavirus Vaccine** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. Gary Ka-kui AU, Speaker: Dr. Helene WAN, Venue: East Ocean Victoria Resturant, Shop 137, L1, Metro City Plaza III, Tseung Kwan O, Kowloon	Mr. Steve WONG Tel: 9045 5134
1:00 pm	**HKMA Kowloon West Community Network – "Management of Chronic Cough in Children: Case Series in a Community Clinic"** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. Gin-pang LEUNG (pending), Speaker: Dr. Kin-wai CHAU, Venue: Crystal Room I-III, 30/F., Panda Hotel, Tsuen Wan, N.T.	Miss Carman WONG Tel: 2527 8285
1:45 pm	**HKMA – Tai Po Community Network – Management of Pressure Sores in Elders with Aggressive Nutritional Therapy** Organiser: HKMA – Tai Po Community Network, Speakers: Dr. Kin-wah LIU & Dr. Lok-kwan DAI, Venue: Chiu Chow Garden Restaurant, Shop 001-003, 1/F, Uptown Plaza, Tai Po, NT	Mr. Sammy LEE Tel: 2688 2933 1 CME Point
10 WED 7:30 am	**HK Neurosurgical Society Monthly Academic Meeting – Information Technology in Neurosurgery** Organiser: Hong Kong Neurosurgical Society, Speaker: Dr. John KWOK, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Gilberto LEUNG Tel: 2255 3368 Fax: 2818 4350 1 CME Point
1:00 pm *(24)*	**HKMA Central, Western & Southern Community Network – Certificate Course on Orthopaedics (III) & (V)** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. Sabrina Lai-ching HO & Dr. Yim-kwai LAW, Speakers: Dr. Eric Hing-chung CHAK & Dr. Daniel Kwok-hing YIP, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Alice TANG Tel: 2527 8285 2 CME Point
11 THU 12:30 pm	**HKMA CME Series on Chronic Hepatitis B and its Complications (NT West District)** Organised by: The Hong Kong Medical Association, Chairman: Dr. Aaron Fook-kay LEE, Speaker: Dr. Vincent Wai-sun WONG, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building,10 Sai Ching Street, Yuen Long, N.T.	Miss Viviane LAM Tel: 2527 8452 1 CME Point
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2010 – Common Eye Diseases of Children** Organiser: The Hong Kong Medical Association, Speaker: Dr. Agnes T.Y.TSE, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Viviane LAM Tel: 2527 8452 1 CME Point
12 FRI 2:00 pm	**HKMA – Shatin Doctors Network – A New Frontier in Hypertensive Management** Organised by: HKMA – Shatin Doctors Network, Speaker: Dr. Bernard Bun-lap WONG, Venue: Level 2, Jasmine Room, Royal Park Hotel, Shatin, N.T.	Ms. Iris POON Tel: 2881 4236

Calendar of Events

Date / Time	Function	Enquiry / Remarks
13 SAT 1:30 pm *(17)*	**HKMA Kowloon East Community Network – Joint CME Course for Health Personnel 2010 on "Update on PCOS"** Organiser: HKMA Kowloon East Community Network; Hong Kong College of Family Physicans & United Christian Hospital, Chairman: Dr. David CHAO, Speaker: Dr. Yau-bong HO, Venue: Lecture Theatre, G/F., Block F, United Christian Hospital	Ms. Gary WONG Tel: 3513 4821
2:30 pm	**Refresher Course for Health Care Providers 2010/2011** Organiser: The Hong Kong Medical Association, Speaker: Dr. Suk-yin CHAN, Venue: OLMH	Miss Viviane LAM 2 CME Points
14 SUN 2:00 pm	**HKMA Certificate Course on Family Medicine 2010** Organiser: The Hong Kong Medical Association, Speakers: Dr. Linda Yin-fun HUI & Prof. Samuel Yeung-shan WONG, Venue: Queen Elizabeth Hospital, Kowloon	Miss Viviane LAM Tel: 2527 8452 3 CME Points
17 WED 1:30 pm	**HKMA Hong Kong East Community Network – PPI on "Hypertension" (PENDING)** Organiser: HKMA Hong Kong East Community Network; HA Hong Kong East Cluster Doctors Network, Venue: Lecture Theatre, G/F., HKEC Training Centre for Healthcare Management & Clinical Technology, Pamela Youde Nethersole Eastern Hospital, 3 Lok Man Road, Chai Wan, Hong Kong	Miss Alice TANG Tel: 2527 8285
18 THU 12:30 pm	**HKMA CME Series on Chronic Hepatitis B and its Complications (Kowloon District)** Organiser: The Hong Kong Medical Association, Chairman: Dr. Kin CHOI, Speaker: Dr. Henry Lik-yuen CHAN, Venue: Ballroom, Level 7, Langham Place Hotel, 555 Shanghai Street, Mongkok, Kowloon	Miss Viviane LAM Tel: 2527 8452 1 CME Point
1:00 pm	**HKMA New Territories West Community Network – Lipid Disorder (PENDING)** Organiser: HKMA New Territories West Community Network, Venue: Yuen Long	Miss Alice TANG Tel: 2527 8285
7:00 pm – 8:30 pm	**FMSHK Executive Committee and Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chambers, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
8:30 pm – 10:00 pm	**FMSHK and HKFMS Foundation Annual General Meeting** Organiser: The Federation of Medical Societies of Hong Kong and HKFMS Foundation Limited, Venue: Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
19 FRI *(20,21)*	**Oxfam Trailwalker 2010** Organiser: The Hong Kong Medical Association, Venue: MacLehose Trail	Miss Peony CHAN Tel: 2527 8285
21 SUN 2:00 pm	**HKMA 90th Anniversary Football Cup** Organiser: The Hong Kong Medical Association, Venue: CUHK	Miss Peony CHAN Tel: 2527 8285
23 TUE 1:00 pm	**HKMA Kowloon West Community Network – Sleep Apnoea Syndrome** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. Ngam LAM, Speaker: Dr. Bing LAM	Miss Carman WONG Tel: 2527 8285
25 THU 1:00 pm	**HKMA New Territories West Community Network – Mood (PENDING)** Organiser: HKMA New Territories West Community Network, Venue: Yuen Long	Miss Alice TANG Tel: 2527 8285
26 FRI 1:00 pm	**HKMA Hong Kong East Community Network – Rotavirus Vaccination: Why Parents Should Consider It** Organiser: HKMA Hong Kong East Community Network, Speaker: Dr. Paul Chik-wa LEUNG, Venue: Crowne Plaza Hong Kong Causeway Bay	Ms. Candy CHOI E-mail: candy.k.choi@gsk.com
27 SAT 3:00 pm	**HKMA Trailwalker Reunion Party** Organiser: The Hong Kong Medical Association, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Peony CHAN Tel: 2527 8285
(28)	**The 3rd Hong Kong International Burns and Wound Healing Symposium** Organiser: HK Society of Burns and Wounds Healing, Chairman: Prof. Andrew BURD & Prof. Shekhar KUMTA, Speakers: Various, Venue: Prince of Wales Hospital, Shatin, N.T.	Ms. Vicky CHUNG / Ms. Ruby LAM Tel: 3151 8900 Fax: 2590 0099
30 TUE	**9th Asia-Pacific Conference on Human Genetics** Organisers: Hong Kong Society of Medical Genetics & The Asia Pacific Society of Human Genetics, Chairman: Dr. Stephen LAM, Speakers: Various, Venue: Hong Kong Academy of Medicine, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Conference Secretariat Email: apchg2010@ctshk.com

Courses / Meetings

6/12/2010	**A "Hard" Scrotal Abscess** Organiser: Hong Kong Urological Association, Chairman: Dr. Yin-chak LAW, Speaker: Dr. Ringo Wing-hong CHU, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon, Enquiry: Dr. Hing-hoi HUNG / Ms. Tammy HUNG, Tel: 2958 6006 / 9609 6064, Fax: 2958 6076 / 8344 5115, CME Accreditation: 1 Point (The College of Surgeons of Hong Kong)
17-19/12/2010	**2010 Asian Chinese Quality of Life Conference** Organisers: International Society for Quality of Life Research – Asian Chinese Chapter; Family Medicine Unit, Department of Medicine, Li Ka Shing Faculty of Medicine, The University of Hong Kong & Hong Kong Society for Quality of Life, Co-Chairmen: Prof. Feng-bin LIU, Prof. Cindy LAM & Mr. Kwok-fai LEUNG, Speakers: Various, Venue: Li Ka Shing Faculty of Medicine, The University of Hong Kong, 21 Sassoon Road, Pokfulam, Hong Kong, Enquiry: Ms. Candy LAW, Tel: 6509 6582, Fax: 3528 5727, Email: candy@hksoqol.org, Website: http://www.hksoqol.org/conf2010
14-16/1/2011	**Hong Kong International Acupuncture Conference – Neurological and Mental Illness** Organiser: Hong Kong Association for Integration of Chinese-Western Medicine & Hospital Authority, Chairman: Dr. Vivian Taam Chi Woon WONG, Speakers: Various, Venue: Hong Kong Academy of Medicine Jockey Club Building, Enquiry: Ms. Jessie CHOW & Ms. Y.C. YEUNG, Tel: 2871 8787, 2871 8897 / 3119 1850, Fax: 2871 8898
22/1/2011	**Hepatobiliary & Pancreatic Surgery and Liver Transplantation** Organiser: Department of Surgery, The University of Hong Kong, Queen Mary Hospital & Hong Kong Chapter of American College of Surgeons, Venue: Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong, Enquiry: Forum Secretary, Hong Kong Surgical Forum, Tel: (852) 2855 4885 / (852) 2855 4886, Fax: (852) 2819 3416, E-mail: hksf@hku.hk, Web-site: http://www3.hku.hk/surgery/forum.php
12-14/5/2011	**18th Asian Congress of Surgery & 37th Philippine College of Surgeons Mid-year Convention** Organiser: Asian Surgical Association, Venue: Waterfront Cebu City Hotel & Casino, Lahug, Cebu City, Philippines, Enquiry: Congress Secretariat, Tel: (632) 9274973-74; (632) 9281083; (632) 9292359, Fax: (632) 9292297, E-mail: secretariat@acs2011.org, Website: www.acs2011.org

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Reference: 1. K.D. Kaufman et al. Eur J Dermatol 2002; 12:38-49. 2. Data on File (MSD, Hong Kong)

12-2011-PPC-09-HK-9023-J