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FINAL CALL

Annual Scientific Meeting 2013

Obesity Related Disorders: An Emerging Epidemic

Date : 23 June 2013 (SUN) Time : 9:30AM - 4:30PM

Venue : Ballroom, 3rd Floor, Sheraton Hotel, 20 Nathan Road, Tsim Sha Tsui, KLN

Exercise, Nutrition and Obesity Issues
- **The evolutionary origins of obesity - any hint for public health actions?**
  Mr. Gordon CHEUNG
- **A comprehensive dietary approach to obesity management**
  Ms. Sally POON
- **Exercise and Obesity**
  Ms. Jenny NG
- **Dangers of self-medication to treat obesity**
  Dr. Vanessa NG

Adult Obesity
- **Obesity - a common cause of hypertension**
  Prof. Bernard CHEUNG
- **Risk factors and updated management of GERD**
  Dr. Benjamin WONG
- **Obesity and Type II Diabetes: Cause and Effect**
  Prof. Annie KUNG

Obesity, Sleep and CNS
- **Obesity - Does sleep play a role?**
  Prof. Yun-kok WING
- **Obesity and obstructive sleep apnea**
  Dr. Jamie LAM

Epilepsy and Obesity
Dr. Mario CHAK

Paediatric Obesity
- **Obesity and diabetes in the youth- what you should know?**
  Prof. Alice KONG
- **Fatty liver in children**
  Dr. Chung-mo CHOW
- **Adolescent eating disorder: updates on diagnosis and management**
  Dr. Phyllis KL CHAN

Surgical Treatment of Obesity
- **Bariatric surgery for Obesity**
  Prof. Kwok-wai NG
- **Post-bariatric Truncoplasty**
  Dr. Peter PANG
- **Vaser Liposuction and Abdominoplasty for Abdominal Obesity**
  Dr. Chun-on MOK

Obesity and Dyslipidaemia
- **Obesity and dyslipidaemia: can medication outweigh lifestyle?**
  Prof. Brian TOMLINSON

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The Cover Shot

This is a male Plumbeous Redstart, which is a very beautiful small bird that lives near rivers and streams. It feeds on worms and insects found along the river and between rocks. It makes short fly-catching flights and snatches insects from the water surface. The photo is taken with a Canon 1DX+ 500mm F4 at Tsuen Wan, New Territories.

Dr. Simon KU
MBBS(HK), MRCP(UK), FHKAM(Medicine), FHKCP
Dermatologist in Private Practice

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Editorial

Dr. Kingsley Hau-ngai CHAN

MBBS (HK), MRCP (UK), Dip Derm (Glasg), FHKCP, FHKAM (Medicine), FRCP (Glasg)

Honorary Consultant Dermatologist, Hospital Authority, Speciatist in Dermatology & Venereology

Editor

Dr. Kingsley Hau-ngai CHAN

First Do No Harm

The DR Medical Centre incident has raised concerns over the efficacy and safety of various medical-cosmetic treatments and procedures available in the market. While the incident reflected the general public's great appetite for new treatments to address their dermatological issues, it has, more importantly, highlighted the importance for all medical treatments offered to patients to be firmly supported by solid scientific evidence such that we can live up to our Hippocratic oath "to first do no harm to the patient".

In this issue, we have received a collection of submissions on recent advances in the treatment of various common dermatological problems: Dr. NM LUK, a highly-regarded dermatologist and the Director of Dermatology Research Centre at the Chinese University of Hong Kong discusses recent advancements in the management of alopecia areata; Dr. KK LO, an eminent dermatologist and the former consultant-in-charge of the Social Hygiene Service reviews the management of atopic dermatitis in adults; Dr. Mimi CHANG shares her experience and local data in managing and treating bullous pemphigoid; Prof. Henry CHAN, a renowned dermatologist with a keen research interest in the cosmetic dermatology field, gives an update in the recent advances in the treatment of pigmentary disorders. In this issue, we are extremely honoured to feature a guest contributor, Prof. BOEHNCKE, Chairman of the Dermatology Unit at the Geneva University in Switzerland, who shares his experience in the management of psoriasis.

Last but not least, I would like to thank Dr. Simon KU, a talented photographer for contributing to our cover photo. Thanks are also due to Dr. Chi-keung KWAN, our honorary social secretary in the Social Hygiene Service for sharing his tips on hiking routes around Hong Kong.

I hope that like me, you will find the articles in this issue comprehensive, interesting and helpful. Enjoy!

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- Stiefel, a GSK company, is committed to improving the quality of life for the approximately 390 million people[1] around the world who suffer from atopic eczema/atopic dermatitis.
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Alopecia Areata – a Brief Review and a Report of a Local Study Using Diphenylcyclopropenone for Treatment of Alopecia Areata

Dr. Nai-ming LUK

MB ChB (CUHK), FHKAM (Med), FHKCP, FRCP (Glasgow)

Dermatologist

Epidemiology

Alopecia areata (AA) is a non-scarring autoimmune disease. The life time prevalence is estimated to be 1.7 - 2%[1,2] with equal sex incidence. However, more than 60% of patients develop the disease in their childhood and only 20% are older than 40 years old.

Clinical features

AA is usually asymptomatic, though some patients may have pruritus, burning sensation or pain[3]. Usually it is noted incidentally by the patient, family members or hair dressers. Typical lesions of AA are oval or round skin-coloured bald patches with hair follicles preserved. The exclamation mark hairs, which taper proximally and widen distally, are said to be characteristic and are commonly found at the peripheral active lesions. The hair pull test may be positive. Besides the scalp, which is the commonest affected area, any hair bearing area such as the axilla and pubic area could be affected. On recovery, usually hypo-pigmented hairs appear which eventually resume its pigmentation.

AA could be classified according to the extent of hair loss[2]. Patchy AA, in which there is partial loss of scalp hair; Alopecia totalis (AT), in which all scalp hair is lost; Alopecia universalis (AU), which all the scalp, axillary and pubic hairs are lost. Patchy AA is the most common pattern seen. There is also an ophiasis pattern where hair loss is mainly concentrated as bands over the posterior occipital area. This pattern is associated with a poor prognosis.

Nail abnomalities have been reported in 7 to 66% of patients with AA[4]. Nail pitting, trachyonychia, beau lines, onychorhexis, onychomadesis, koilonychia, punctuate or transverse leukonychia have been reported.

Dermoscopic features include yellow dots, black dots, broken hairs, short vellus and tapered hairs. These features however are non-specific but can be helpful in doubtful cases of AA[5].

Associated abnormalities

Autoimmune thyroid diseases are the most common associated disease with AA and are found between 8 to 28%[6]. However, the titre of the anti-thyroid does not correlate with the severity of AA. Vitiligo is found between 3 to 8% compared with 1% in the general population. Also atopy is almost doubled in AA patients compared with the general population[7].

Other diseases that have been reported to be associated with AA include Down's syndrome, Addison disease, Autosomal Recessive Autoimmune Polyglandular Syndrome, Pernicious anaemia, Lupus, Sjogren syndrome. The less common autoimmune diseases are usually associated with AT/ AU[8].

AA patients could have anxiety and mood disturbance[9]. Besides, eye complications such as asymptomatic lens opacities and fundus change has been reported in 40 to 50% of AA patients[10].

Pathogenesis

It is widely believed that AA is an autoimmune disease as supported by i) presence of inflammatory cells around the hair follicles, ii) presence of hair follicle-specific auto-antibodies; iii) response to treatment with immunosuppressive medicaments and iv) the association with other autoimmune diseases such as autoimmune thyroid disease[11]. Other aetiologic factors that have been suggested to cause AA include stress, infectious agents, vaccination, hormonal factors and genetics. In the recent genomewide association study, at least 8 regions in the genome were found to be associated with AA[12].

Differential diagnosis

In children, AA should be differentiated from trichotillomania where patients may be stressful or suffer from behavioural disorders. Tinea capitis is another condition that may present with patchy hair loss. Institutional outbreaks could be the scenario and Wood's lamp screening to be followed by microscopy and culture of plucked hairs could differentiate between the two. Congenital patches of hair loss affecting the frontal and temporal hair line could prompt at the diagnosis of temporal triangular alopecia and frontal fibrosing alopecia in the case of black American ethnicity should be considered. For diffuse AA, differentiation should be made with telogen effluvium, lupus and sometimes syphilis in the right settings.

Clinical course and Prognosis

The clinical course of AA is unpredictable, spontaneous recovery could occur in more than 50% of patients within 1 year[2]. However, more than 80% of patients have relapse or recurrent disease[1]. Patients with AT / AU have less than 10% chance of full recovery.

Poor prognosticators include extensive hair loss, long

duration of disease, ophiasis pattern, positive history of atopy, positive family history, the presence of other autoimmune diseases, nail involvement and early age of onset[13].

Investigations

For patients with classical presentation of patchy hair loss, normal scalp and presence of characteristic exclamation mark hairs, no further investigations are required. To differentiate from other causes of hair loss, Wood's lamp examination, skin scrapping for fungal element, hair clipping for fungal culture, blood tests for autoimmune immune diseases such as Lupus or thyroid diseases, Syphilic serology, and complete blood picture and biochemical screening may be required. Last but least, a skin biopsy may be justified for difficult cases.

Histopathology

In acute AA, aggregates of lymphocytes can be found around the anagen follicles giving the "Swarm of bees" appearance[14]. The T cells are mainly of CD4+ and CD8+ cells. Other cells present include Langerhans cells, eosinophils, mast cells and plasma cells. Around the hair follicles, micro-vesiculation, apoptosis, necrosis, macrophages and foreign body giant cells could sometimes be found. In chronic disease, significant hair miniaturisation with little or no inflammation could be the only finding[15].

Treatment

For localised disease, intralesional and topical corticosteroid, topical minoxidil[16], topical anthralin[17] and topical immunotherapy[18], prostaglandin analogs[19], or phototherapy[20] could be tried. For generalised AA, systemic corticosteroid, cyclosporine, sulfasalazine, methotrexate and azathioprine could be used[21].

Other supports

Though AA is not a lethal disease, it could have a significant psychosocial impact on patients[9]. Attending physicians should explore the patients' psychology and provide psychological support if needed. For AT / AU, a false wig could improve patients' outward appearance and morale. In cases of psychiatric co-morbidities, psychiatric referral could be considered. Patients' family support and local support groups could also be of help and The National Alopecia Areata Foundation (www.naaf.org) in the US can provide further information on this condition and a pen pal programme for patients whom want to share their feeling of seeking advise from patients with AA.

Local study: Topical immunotherapy diphenylcyclopropenone (DPCP) treatment on Chinese patients with extensive and steroid resistant AA[18]

Recently topical immunotherapy has been introduced to treat patients with extensive AA. However, no study has been performed to evaluate this modality of treatment in Hong Kong. We set out to study the safety and efficacy of DPCP in Chinese patients with extensive AA who failed steroid treatment.

Between June 2009 and May 2010, we had treated 31 patients in total with age between 12 and 48 years old. Those treated were AA patients with 30% or more hair loss and failed topical or intralesional steroid for at least 6 month.

Classification of disease severity and outcome measurement

The extent of hair loss was divided into five classes with S1 disease (no hair loss); S2 disease (26-50% hair loss); S3 disease (51-75% hair loss); S4 disease (76-99% hair loss) and S5 disease (100% hair loss). Clinical response was measured as: CR (complete response): >90% re-growth; PR (partial response): >50-90% re-growth; MR (minimal response):>10 - 50% re-growth; NR (no response): <10% re-growth of scalp hair. Relapse was defined as >25% hair loss after hair regrowth.

The treatment regime

During the first visit, patients were sensitised with 2% DPCP (FLUKA, Sigma-Aldrich) for an area of 2 x 2 cm^2 on the scalp and were asked to return two weeks later for appraisal. For those without untoward effects, DPCP with escalating concentrations (0.001%, 0.01%, 0.05%, 0.1%, 0.5%, 1.0% and 2.0%) were applied to the whole scalp at weekly intervals. Patients were instructed to avoid direct sun exposure and to wash off the DPCP 48 hours after application. The target concentration was to maintain the reaction of itchiness and erythema of the treated areas for 48 hours.

Results

Thirty-one (16 male, 15 female) Chinese patients with steroid resistant and extensive AA were treated with DPCP during the study period. Their mean age was 28.9 years (SE 10.4). The mean age of onset was 17.8 years (SE 8.8) with average disease duration of 11.2 years (SE 7.7). Ten patients had history of atopy and 4 had history of thyroid disease. Nail changes (pitting and trachyonychia) were found in 14 patients and family history of AA in 2 patients. Thirteen patients (41.9%) had S5 disease (100% hair loss), 9 patients (29.0%) had S4 disease (76-99% hair loss), 4 patients (12.9%) had S3 disease (51-75% hair loss) and 5 patients (16.1%) had S2 disease (26-50% hair loss).

Treatment outcome

Two patients abandoned the treatment due to severe side effects. Of the remaining 29 patients, 4 (13.8%), 7 (24.1%), 5 (17.2%) and13 (44.8%) patients achieved >90% (CR) (Figure 1), >50-90% (PR), >10-50% (MR) and <10% (NR) hair regrowth respectively within the treatment period of 6 to 18 months. And, out of the 4 patients with CR, 4 (100%) had relapse (>25% hair loss) and 5 out of 9 patients with PR had relapse after 18 months follow up. The overall relapse rate in our patients with PR and CR was 69.23%.

Adverse events

Among the adverse reactions, all patients complained of itchiness and noted erythema over the treated sites. These were regarded as desired reactions. More than half of the patients had eczematous reactions with scaling (55%), vesiculation and localised bullous eruptions (76%). Cervical lymphadenopathy was noted in 15 (52%) of patients while post-inflammatory hyperpigmentation developed in 13 (45%) of patients. Vitiligo developed in one patient over the treated area.

Prognostic factors

Based on the 29 patients who had completed at least 6 months of DPCP treatment, chi square tests were used to correlate the good treatment response (>50% regrowth) to sex, childhood onset of disease (< 18 years), chronic duration of disease (> 10 years), personal history of atopy, family history of AA, extent of alopecia (totalis vs areata) and presence of nail changes. It was found that childhood onset of disease (p=0.015) was significantly correlated with a poor response. This was also confirmed by the binomial logistic regression model as an independent risk factor for poor treatment response (p=0.047, OR=0.160).

Discussion

Our study confirmed the efficacy of DPCP in Chinese patients with steroid resistant and extensive AA. Thirteen (44.8%) of our patients achieved either a PR or CR which was slightly less than a recent review of 142 patients[22]. This might be due to the inferior clinical profiles of our patients such as longer duration of diseases (10.61 years vs 6 years), large number of AT/AU patients (44.8% vs 17.7%) and the short duration of treatment (mean 9 months vs 20 months). Two patients had ophiasis on presentation, despite achieving CR and PR in other scalp areas, the ophiasis showed only slight regrowth. Also, out of the 4 patients with CR, 4 (100%) had relapse (>25% hair loss) and 5 out of 9 patients with PR had relapse after 18 months follow up. In our patients, the overall relapse rate for PR and CR was 69.23%.

Adverse effects due to DPCP were usually tolerable. In our study, two patients abandoned the treatment due to severe bullous and urticarial eruptions. The former, however had a full regrowth of her hair. It was not known whether it was due to the 4 treatments she had received or due to the natural course of the disease. Also one patient developed progressive vitiligo over the treated area and subsequently gave up the treatment. The vitiligo did not improve even 1 year after stopping treatment. On further enquiry, the patient volunteered that she actually had a small depigmented lesion at her left axilla before treatment which was stable over the years. This raised the question whether patients with vitiligo should be excluded from DPCP treatment as the risk of vitiligo complicating the treatment may be higher. Otherwise all the other adverse effects were endurable to our patients with symptomatic treatment of topical steroid or oral antihistamine. One point to note is that the climate is Hong Kong is hot and humid and almost all our patients complained of burning sensations after DPCP application aggravated by occlusion and sweating (due to their wig or hats), so patients in hot climatic areas should be forewarned. Furthermore two of our patients experienced short periods of chills on the day of treatment which subsided the following day without treatment. Compared with the conventional topical and intralesional steroid therapy, topical immunotherapy has to be applied weekly and hence patients have to attend the clinic more frequently. These are the practical considerations that patients should consider before embarking on this modality of treatment.

In the literature, poor treatment outcomes of DPCP included severity of alopeica at the time of presentation[23], presence of nail changes and duration of disease before treatment[24], age of onset, duration of disease and personal history of atopy[25]. Taking all these studies together, the most consistent prognosticator would be the extent of AA before treatment. In our study, using chi square tests to correlate treatment outcomes and clinical parameters showed that childhood onset of disease (<18 years) was a poor prognosticator which was further confirmed in logistic regression models. There was no correlation between treatment responses with extent of disease on presentation, personal history of atopy, duration of disease longer than 10 years, family history of Alopecia Areata. Due to the small sample size in our study, these correlations need further verification by larger studies.

Limitation of this study

Our study was a retrospective study with only a short follow up period. The sample size was small (n=29) and the highest DPCP concentration used was only 2%. In addition, most of the patients had only just 6 months of treatment.

In conclusion, our retrospective study showed that DPCP was effective and tolerable to Chinese patients with steroid resistant and extensive alopecia. DPCP could be considered in case patients failed the conventional therapy or those who do not want to be subjected to systemic treatment or phototherapy.

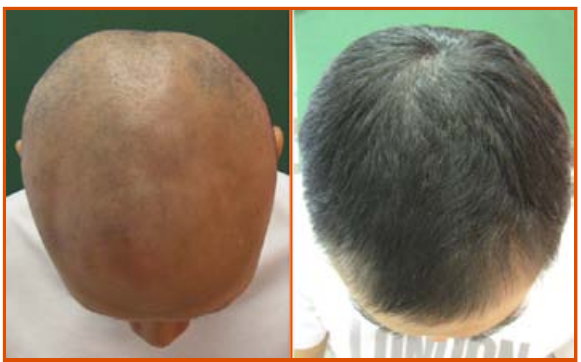

Figure 1. Complete remission after 6 months of DPCP treatment

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Reference 1. Dahlof B, Sever PS, Poulter NR, et al. Prevention of cardiovascular events with an antihypertensive regimen of amlodipine adding perindopril as required versus atenolol adding bendroflumethiazide as required, in the Anglo-Scandinavian Cardiac Outcomes Trial-Blood Pressure Lowering Arm (ASCOT-BPLA): a multicentre randomized controlled trial. Lancet. 2005;366:895-906.

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Comorbidities Associated with Psoriatic Arthritis

Prof. Wolf-Henning BOEHNCKE

Dept. of Dermatology, Geneva University Hospital, Geneva, Switzerland

Prof. Wolf-Henning BOEHNCKE

Psoriatic arthritis (PsA) is usually associated with psoriasis (of the skin - PsO). Although there are arguments suggesting that PsA might be an entity in its own right, it seems reasonable in daily practice to consider both PsA and PsO to be manifestations of one "psoriatic disease". This pragmatic approach is particularly feasible, as it becomes increasingly clear, that this "psoriatic disease" goes beyond skin and joints. Subsequently, the most relevant clinical comorbidities often associated with this "psoriatic disease" will be discussed. Whenever possible, publications analysing the contribution of PsA to these comorbidities will be cited. However, a bias due to associated PsO cannot completely be excluded.

Osteoporosis

Contrary to rheumatoid arthritis, there are conflicting data on PsA and osteoporosis: On one hand, a study analysing bone mineralisation through ultrasound densitometry in 186 patients and 100 controls showed demineralisation in two thirds of the patients[12], and another documented peri-articular bone loss particularly in early PsA[16]. On the other hand, Borman et al. did not detect differences regarding mineralisation in PsO patients with or without PsA[7]. It might well be, that PsO may result per se in osteoporosis[2].

Malignomas

While PsO is known to be associated with an increased risk for lymphomas and other haematopoietic malignancies as well as skin cancer, similar data are lacking for PsA. In a large cohort study in Toronto, no evidence for an increased risk for malignancies has been observed[21].

Infections

When compared to rheumatoid arthritis, patients with psoriasis and/or PsA seem to have a lower risk for severe infections. Clinical studies with etanercept and adalimumab indicate 3.75, 1.62 and 1.24 severe infections per 100 patient years in rheumatoid arthritis, PsA, and PsO, while the respective numbers are 4.65, 281, and 1.32 in the case of adalimumab[8]. Still, namely infections of the upper respiratory tract are important comorbidities in patients treated with biologics[22].

Metabolic syndrome and cardiovascular diseases

Numerous studies document an association of PsO with the so-called metabolic syndrome (table 1)[14]. Similar results have also been published in PsA[11]. As the increased mortality of PsO patients is primarily due to an increased rate in cardiovascular diseases, the question of whether PsO is an independent cardiovascular risk factor in its own right is heatedly discussed[1]).

Table 1 : WHO definition of the metabolic syndrome.

Main criteria (all must be met)	Diabetes mellitus Pathological glucose tolerance Elevated fasting blood glucose or Insulin resistance
Additional criteria (2 or more must be met)	Arterial hypertension (>140/90 mm Hg) Dyslipidaemia (Triglycerides > 1,695 mmol/l and HDL ≤ 0,9 mmol/l in men or ≤ 1,0 mmol/l in women) Central obesity (hip-to-waist ratio >0,9 (men) or 0,85 (women)

On one hand, the metabolic syndrome basically represents a cumulation of "traditional" cardiovascular risk factors (see Table 1). Moreover, obesity – as a known risk factor for PsO – is an essential criterion for diagnosing the metabolic syndrome. And finally, dyslipidaemia as another facette of the metabolic syndrome is often present already at the time of onset of PsO[20]. These points argue against a role of PsO as an independent cardiovascular risk factor.

On the other hand, there is a "dose effect" in as much as severe but not mild PsO is associated with an increased cardiovascular mortality[19]. In a case-controlled study, we showed that PsO is associated with coronary artery calcification, indicating coronary artery disease[18]. PsA patients show an increased thickness of the wall of the carotid artery when compared to healthy controls. This finding, too, points towards increased atherosclerosis[10]. In addition, a recently published systematic review concludes that most studies point towards an increased cardiovascular risk among PsA patients[17].

The concept of the « psoriatic march »

The role of PsO as an independent cardiovascular risk factor is thus supported by epidemiologic studies. However, a pathogenetic link is also very likely, as pointed out in the concept of the « psoriatic march » [5]: Atherosclerosis is an inflammatory condition, driven by other systemic inflammatory conditions[15]. PsO is one such systemic inflammatory conditions, as biomarkers of inflammation can readily be detected e.g. in patients´ sera. These biomarkers include adipokines, mediators secreted by adipocytes.Some of these exhibit anti-

insulinic effects and therefore cause insulin resistance[4]. At the level of the endothelial cells (which also express the insulin receptor), vasodilating signals are no longer effective, resulting in endothelial dysfunction and vascular stiffness. This provides the basis for atherosclerotic plaque formation and subsequently arterial hypertension, myocardial infarction, or stroke. In recent years, this concept has been supported by publications from numerous groups[6].

Clinical consequences

Comorbidities directly influence therapeutic decisions. In patients with PsO and PsA it is advantageous to select a drug that is effective to control both. This became increasingly easier with the availability of tumour necrosis factor alpha blocking biologics. But other comorbidities have to be considered as well, as they might represent contraindications for the use of "conventional" drugs to treat PsO and/or PsA. Some "conventional" drugs might increase the patients´ cardiovascular risk; this is true for retinoids (dyslipidaemia!) and cyclosporine A (hypertension!).

Comorbidity results in comedication[13]. Thus, potential drug-drug interactions also need to be taken into account when opting for a systemic anti-psoriatic therapy. This risk is relatively high for cyclosporine A and methotrexate, when compared to biologics.

Suggestions for patient monitoring

Recently, Spanish dermatologists have published an algorithm to comprehensively monitor PsO patients for comorbidities[9]. Given the complexity of this algorithm, it is questionable, if such a concept can be implemented in the daily practice of dermatologists in private practice (or family doctors). Still, PsO patients should not only be screened for signs and symptoms of PsA, but also for key cardiovascular risk factors. As of now, the latter can be restricted to patients with moderate to severe PsO. Parameters often suggested to be monitored comprise pulse and blood pressure (hypertension), Body Mass Index or waist circumference (obesity), fasting blood lipids (dyslipidaemia), as well as fasting or occasional blood glucose (diabetes mellitus). This would be a feasible approach for daily practice (Table 2) [3].

Table 2: suggested monitoring of the most relevant cardiovascular risk factors in patients with moderate to severe PsO (modified from[3]).*

Risk factor	Parameter	Zielwert
Arterial hypertension	Blood pressure	If no more than 2 risk factors: >140/90 mm Hg If >2 risk factors, end organ damage (e.g. kidneys), diabetes mellitus, or metabolic syndrome: >130/80 mm Hg
Obesity	Waist circumference	Men: <102 cm Women: < 88 cm
Dyslipidaemia	Fasting blood lipids	1 Risk factor: LDL-Cholesterin <160 mg/dl >1 risk factor: LDL-Cholesterin <130 mg/dl Metabolic syndrome. LDL-Cholesterin <100 mg/dl High-risk patients: LDL-Cholesterin < 70 mg/dl
Diabetes mellitus	Fasting blood glucose Or Occasional blood glucose	Fasting blood glucose <100 mg/dl

** >10% of body surface area affected*

As moderate to severe PsO is regarded as an independent cardiovascular risk factor, results of such a screening need to be communicated to the patients´ family doctors (if the family doctor has not performed such a screening himself). This information is essential to define individual treatment goals for the respective comorbidities. From a dermatologist´s perspective, it is mandatory that family doctors should consider PsO and PsA when treating hypertension and dyslipidaemia.

Summary for daily practice:

- **PsA is usually associated with PsO. The most important comorbidities are identical.**
- **These comprise osteoporosis, infections, metabolic syndrome, and cardiovascular diseases.**
- **PsO/PsA, as a chronic systemic inflammatory disorder, represents an independent cardiovascular risk factor.**
- **Patients with moderate to severe PsO (>10% of the body surface area affected) and/or PsA should be monitored for the most relevant "traditional" cardiovascular risk factors.**
- **Presence and the number of cardiovascular risk factors need to be taken into account when defining treatment goals for cardiovascular comorbidities, namely hypertension and dyslipidaemia.**
- **Comorbidites and comedication influence treatment decisions.**

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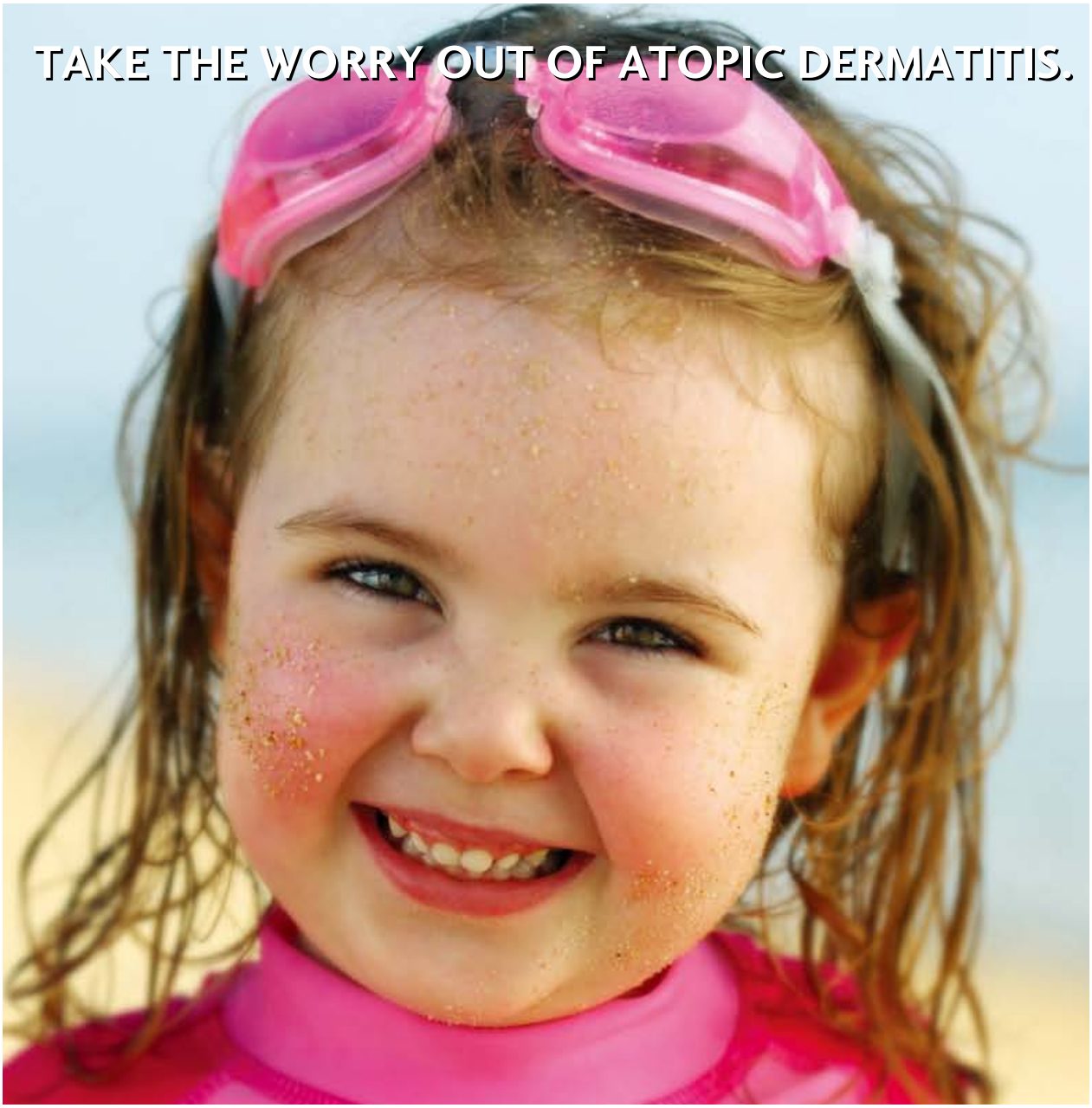

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A Brief Review on Treatment of Atopic Dermatitis (AD) in Adults

Dr. Kuen-kong LO

MB.,BS (HKU), MRCP, FRCP, FHKCP, FHKAM(Med)
Specialist in Dermatology & Venereology

Dr. Kuen-kong LO

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 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 30 June 2013.

Atopic dermatitis (AD) is a common skin disease that affects 10% to 30% of children but only 1 to 3% in adults. The prevalence of AD is of an increasing trend in the past 3 decades based on some epidemiologic studies. Though AD is less commonly found in adults, most adult AD patients have nearly lifelong disease. The chronic relapsing and sometimes severe itchiness in the disease course of AD has a significant impact on patients' quality of life. The treatment of frequent flare-ups in some sub-groups of recalcitrant AD is challenging.

Removal of food allergens from AD patients' diet may lead to improvement in childhood AD but its role in adults seems to play a less significant role. Furthermore the diagnosis of an IgE-mediated trigger (food and environmental allergy) is typically best made from a detailed clinical history rather than via RAST or skin prick testing because over 80% of atopics will be found to have elevated IgE levels to specific allergens (i.e. the positive predictive value of these tests is low). As the skin barrier defect is found to play a more important role in the pathogenesis of AD in children, the recommendation of liberal use of emollients works for adult AD as well. Excessive prolonged bathing and use of barrier damaging strong soap are to be discouraged. In the choice of emollients and moisturisers, care should be taken to avoid those products with fragrances, sensitising preservatives, damaging surfactants and stabilisers. In general, ointments are preferred over creams and lotions especially in low humidity seasons. In recent few years, newer moisturisers with contents incorporating substance with skin barrier repairing component, anti-itch natural products and nano-technology for better texture sensations are emerging in the market. They are more expensive but would be particularly beneficial for the subgroup of AD patients with filaggrin gene mutation.

The first-line medical treatment for acutely inflamed skin in AD adult patients is still topical steroid preparations. While avoiding triggering factors and liberal use of appropriate moisturisers are useful for mild AD in the stable non-flaring up phase, topical steroid preparations will be needed for acute flare-ups. In general, the choice of moderately potent to a very potent topical steroid is needed for rapid suppression of disease in adults. Prolonged usage is not recommended. The issues of skin atrophy, striae, telangiectasia, hypopigmentation and perioral dermatitis are well known. Prolonged use of topical steroids around the eyes has been reported to induce open-angle glaucoma and cataracts. The risk of systemic side effects from prolonged use of topical steroids has not been studied and in general, it is considered low. I usually recommend adult AD patients to stop the use of topical steroids when the acute dermatitis is controlled or not continued for more than 2 weeks in the same site. It is required to give the same period of rest (1-2 weeks) before re-treatment to avoid tachyphylaxis and cutaneous side effects of topical steroids. Intermittent topical steroid (e.g. Twice-weekly to frequently relapsing sites) application following recovery from an acute flare-up can result in a prolonged disease-free period and aborts frequent relapses.

However, in some specific areas of the body e.g. face and flexural area, side effects from the use of topical steroids is more susceptible. A very short duration of topical steroid usage (e.g. for 2-3 days) with close monitoring is acceptable. It is usually safer to choose the mildest formulation (1% hydrocortisone acetate) or to choose topical calcineurin inhibitors for these areas.

The topical calcineurin inhibitors (TCI) (tacrolimus and pimecrolimus) are safe to use in adult AD. The US FDA issued a public health advisory for labelling that the potential cancer risk of these medications in March 2005. However, the action was based on studies (in mice, rats and monkeys exposed orally, and in rodents exposed topically) in which cancers developed following exposures to these calcineurin inhibitors (CI) at systemic levels more than 25 times higher than the maximum human exposure following topical application. Furthermore, the molecular size of CI (>800 daltons) is larger than that of steroids (<500 daltons). This makes CI less penetrating to intact skin. In a 1-year study of adults with moderate to severe AD treated with tacrolimus ointment, blood levels were consistently found to be low. Given the lack of significant systemic exposure following topical application of TCI, the American Academy of Dermatology states that there are no data that can prove that proper topical use of TCI is dangerous in humans. Intermittent proactive treatment with TCI (e.g. once to twice weekly) to frequently relapse sites of AD adults can be a useful regimen to render a disease-free period.

Topical coal tar products are less welcomed by adult AD patients as it is less cosmetically acceptable. They are not expensive and can reduce itching in some chronic skin

lesions. However, they should not be used in acutely inflamed and damaged skin to reduce the potential for development of allergic contact dermatitis.

Wet wraps and wet dressing treatment proven to be effective in paediatric AD patients may not be practical for adult AD patients as it is a labour intensive process and larger wraps in adults are not cost effective.

Oral sedating H1-antihistamines are commonly prescribed in adult AD to reduce pruritus especially at night time. The improvement can be attributed to enhancement of nocturnal sleep and reduction in scratching. It may be more useful for those who have associated rhino-conjunctivitis. Newer non-sedating H1-antihistamines are less useful for treatment of adult AD.

Systemic therapies are often not required for treatment of AD except in short terms of flare-ups and complications. A 5 -7 days' course of systemic antibiotics (against staphylococcus) is often prescribed to abort acute flare-ups of adult AD especially when there are signs of impetiginised eczema. Systemic steroid should be used infrequently and only to quell major flare-ups in adult AD. Other systemic therapies e.g. cyclosporine, azathioprine, mycophenolate mofetil, methotrexate, intravenous gamma-globulin have been used for acute flare-ups and in some subgroups of very recalcitrant and severe AD. Phototherapy with narrow band UVB has been found to be useful for treatment of AD in adults. Though phototherapy seems to be a safer alternative to other systemic treatments for adult AD, it has its limitations. It is time consuming, requiring multiple visits per week to the treatment centre making the costs of treatment prohibitive for most patients.

There has been little or no evidence to support other treatments such as dietary supplements with fish oil, evening primrose oil, prebiotics, probiotics, antioxidant vitamins and zinc supplements. The roles of hypnotherapy and biofeedback or other relaxation techniques may be useful for some subgroups of adult AD patients but data are lacking for proving its effectiveness. Treatment of AD with Traditional Chinese Medicine (TCM) has been studied before but it is not conclusive. The variability of TCM compositions (some may contain steroids) and its potential liver toxicity in some reports are obstacles for us to recommend it without reservation in the today's evidence based Western Medicine culture.

Further Reading

Diana Rubel, Thiru Thirumoorthy, Retno W Soebaryo et al, ; Consensus guidelines for the management of atopic dermatitis: An Asia-Pacific perspective. The Journal of Dermatology 2013; 40:1-12

MCHK CME Programme Self-assessment Questions

Please read the article entitled "A Brief Review on Treatment of Atopic Dermatitis (AD) in Adults" by Dr. Kuen-kong LO and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 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 June 2013. 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. Atopic dermatitis is a common skin disease that affects adults up to 10%.
2. Atopic dermatitis in adults has a good prognosis and can be self remitting in 10 years' time.
3. Manipulation of diet and food is an important strategy for management of atopic dermatitis in adults.
4. Food allergy tests usually yield important information for management of adult atopic dermatitis.
5. The first-line medical treatment for acute exacerbations in atopic dermatitis adult patients is topical steroid.
6. Prolonged topical steroid usage can lead to skin hypertrophy.
7. Topical coal tar preparation is one of the popular products used by adult atopic dermatitis patients.
8. Telfast (newer generation of H1 oral antihistamine) is very effective in reducing pruritus in adult atopic dermatitis at night time.
9. Wet-wrap therapy for adult atopic dermatitis patients is a cost effective measure for management of adult atopic dermatitis.
10. Narrow band UVB phototherapy is a useful and inexpensive therapy practical for all adult atopic dermatitis patients.

ANSWER SHEET FOR JUNE 2013

Please return the completed answer sheet to the Federation Secretariat on or before 30 June 2013 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

A Brief Review on Treatment of Atopic Dermatitis (AD) in Adults

Dr. Kuen-kong LO

MB.,BS (HKU), MRCP, FRCP, FHKCP, FHKAM(Med)
Specialist in Dermatology & Venereology

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Answers to May 2013 Issue

Adjuvant Treatment for Early Breast Cancer

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

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Reference: 1. K.D. Kaufman et al., Eur J Dermatol 2002; 12:35-49. 2. Data on File (MSD, Hong Kong)

01-2014-PPC-11-HK-1058-P

Bullous Pemphigoid: a Local Review

Dr. Mimi M CHANG

MBChB, MRCP(UK), FHKCP, FHKAM (Medicine)

Specialist in Dermatology and Venereology, Department of Medicine and Therapeutics, Prince of Wales Hospital

Dr. Mimi M CHANG

Introduction

Bullous pemphigoid (BP) is the most common autoimmune blistering disease, especially in the elderly. It is associated with autoantibodies targeted against hemidesmosomal antigens in the basement membrane zone, resulting in subepidermal blistering. BP is characterised clinically by pruritic tense non-scarring blisters on the extremities and trunk, and histologically, by subepidermal clefts with variable infiltrates including eosinophils. On direct immunofluorescence (IMF), linear IgG and/or C3 deposits are found along the dermo-epidermal junction (DEJ)[1,2].

Epidemiology

Bullous pemphigoid typically affects the elderly after 70 years of age, though it may rarely present in children and young adults[2]. It was estimated that the risk of developing BP after 90 years of age is 300 fold higher than 60 years of age[3,4]. There is no known ethnic, sexual or racial predilection. With ageing population, the incidence is found to be rising, ranging from 6 to 21 new cases per million population per year in Europe[5-7]. In Asia, we found a similar incidence in Singapore (7.6 new cases per million per year) and Taiwan (annual prevalence 0.0027%) [8,9]. In Hong Kong, an early study in the 1990s found 149 new cases in all Department of Health clinics in 8 years[10]. Recently, two large cohort studies in the two university hospitals reflected a growing incidence of 9.2 to 11.2 per million per year[12,13,14]. In the Prince of Wales Hospital, 121 new cases of BP were diagnosed in 2002 to 2011. The majority of patients were elders with mean age of 79.9 years and poor premorbid states (78% were ADL-partially dependent or bed-bound)[14].

Pathophysiology

Circulating and tissue bound pathogenic autoantibodies of IgG subtype (less commonly, IgE, IgA, IgM) attack the hemidesmosomes of the basement membrane zone (BMZ). The two target autoantigens are, BP 180, a transmembrane type XII glycoprotein, and BP 230, an intracellular cytoplasmic protein[1]. The binding of autoantibody to antigen results in activation of the complement cascade, mast cell degranulation and neutrophils recruitment, causing release of proteolytic enzymes that degrade the BMZ, resulting in subepidermal blistering. Autoantibodies also directly interfere with hemidesmosomal cell-cell adhesion. Autoreactive T and B lymphocytes also play a role in BP [15,16].

Clinical presentation

The hallmark of BP is pruritic tense blisters occurring on erythematous or normal-looking skin. They are of variable sizes, and can occur in a localised or widespread fashion, favouring the trunk, flexures, proximal extremities and acral areas (Fig 1). Mucosal involvement, mainly oral mucosa, occurs in less than 10% of BP. In the early phase of the disease, eczematous or urticated plaques are found, and this non-bullous phase may last for weeks and is often misdiagnosed as eczema, urticaria, drug eruption or scabies infestation. In the late phase, excoriations and erosions predominate. Lesions are typically non-scarring and heal with post-inflammatory hyperpigmentation and sometimes milia formation. Other clinical variants of BP, such as, pretibial BP, erythrodermic, vegetating, vesicular, ulcerative, nodular, and dyshidrosiform forms are rare[16]. Bullous pemphigoid can occur in pregnancy (gestational pemphigoid) and in patients with lichen planus (lichen planus pemphigoides).

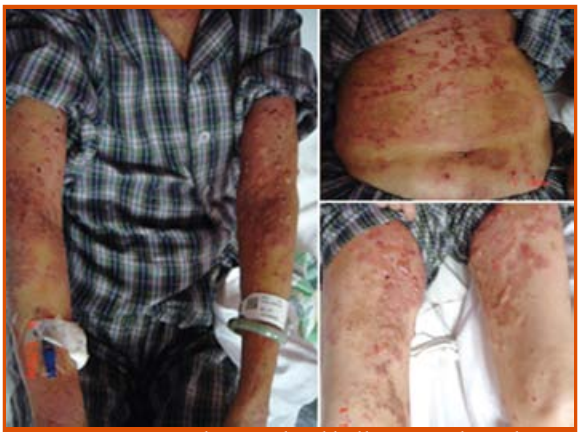

Figure 1. Patient with generalised bullous pemphigoid presenting with typical erythematous large tense blisters

Diagnosis and histology

The diagnosis of BP hinges on classical clinical features, typical histology and direct immunofluorescence (DIF). The combination of the clinical, histological and DIF findings gives a sensitivity of 90% and specificity of 83% and good positive-predictive value of 95-to 99%.[2]

A skin biopsy from a fresh blister stained with haematoxylin and eosin shows classical histology

subepidermal clefting and dermal infiltrates including eosinophils (Fig2). In early lesions, eosinophilic spongiosis is found. The histological diagnosis has to be confirmed with perilesional DIF showing the typical IgG and/or C3 along dermoepidermal junction[1,16] (Fig 3 & 4). It must be noted that the biopsy should include intact epidermis, and sent fresh, or in saline, and processed within 24 hours for DIF reading. It can also be transported in Michel's medium which allows a delay of processing of up to two weeks. Recent immunohistochemistry studies suggested that in formalin-fixed tissues, detection of C3d deposits was useful for diagnosis[17,18].

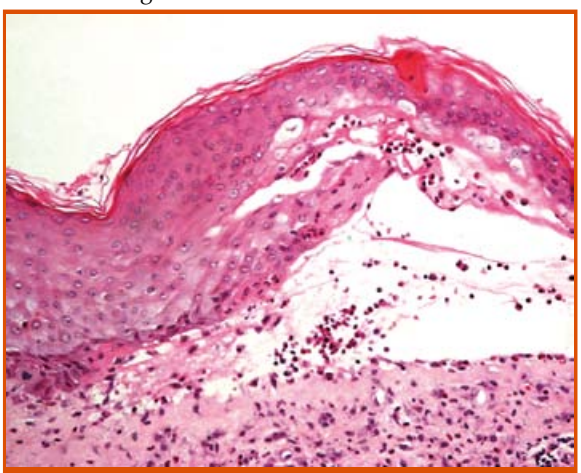

Fig 2. Histology of an eosinophil-rich subepidermal blister H & E. Lower magnification 200x.

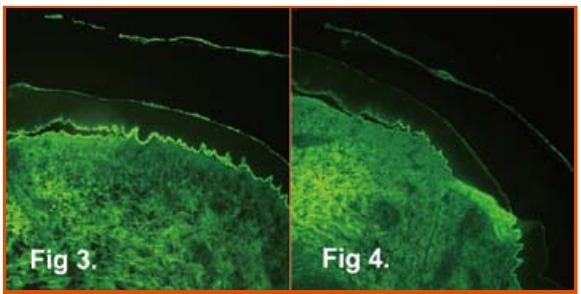

Fig 3. Direct immunofluorescence showing linear deposition of C3 (Fig 3) and IgG (Fig 4) along the dermo-epidermal junction.

In difficult cases, indirect immunofluorescence (IIF), or salt-split test, by incubating diseased human skin in 1M NaCl solution (antigen binding as roof-pattern of the epidermis in BP) can be considered[1,2]. Electron microscopy can also be used to differentiate BP from other subepidermal diseases such as epidermolysis bullosa acquisita. In Hong Kong, checking the serum antiskin antibody (ASA, an IIF using monkey oesophagus as substrate) is sometimes done and 70% of BP would be positive with stratified squamous epithelium (monkey oesophagus)[19]. However, ASA should not be used for the sole diagnostic purpose or routine monitoring for disease activity, as it is for pemphigus. Lately, serum measurements of enzyme-linked immunosorbent assays (ELISA) of BP180 (NC16A domain) have been found to be very specific and sensitive in the diagnosis of BP. [2,20-22]. BP230 ELISA is less sensitive and specific but has a confirmatory diagnostic value in negative cases[2]. The diagnostic sensitivity can be increased up to 100% when both are used together[29]. Commercial kits are available to aid the diagnosis and may be useful in disease monitoring and prognostication[20-22]. However, IF studies remain the gold standard for diagnosis[2,15].

Differential diagnosis

While BP is the most common autoimmune blistering disease in the elderly, other immunobullous diseases such as cicatrial pemphigoid, linear IgA bullous dermatosis, dermatitis herpetiformis, paraneoplastic pemphigus, may present with tense blisters and have to be distinguished from BP by clinical, histological and immunologic studies.

Non-autoimmune causes can be more common and have to be excluded, too. Conditions like acute or contact dermatitis, insect bite reaction, infection, drug eruption, vasculitis and bullae arising from mechanical or metabolic causes (diabetes, renal failure, porphyria), can be differentiated from BP by careful history taking, physical examination and infective workup.

Associated conditions and malignancy

BP has recently been noted to be associated with conditions including inflammatory skin dermatosis, diabetes, and neurological disorders (cerebrovascular disease, multiple sclerosis, dementia and Parkinsonism, possibly due to cross-reactivity with neuronal BP 180/230 expressed in the brain)[2,23]. Unlike pemphigus, the relationship of BP with malignancies is controversial[24,25]. Extensive screening for malignancy in an asymptomatic BP patient is not recommended[2,15]. Compared to out-patient patients locally, hospitalised BP patients (over 90%) had coexisting medical conditions (hypertension, followed by cerebrovascular disease, diabetes mellitus and dementia), rendering them more prone to treatment–related complications and possibly higher mortality[11-13].

Course and mortality

BP runs a waxing and waning course. Occasional spontaneous remissions occur in some localised forms, and rarely, generalised BP. Following conventional corticosteroid therapy, the disease is expected to be well-controlled with fair prognosis with a median treatment of 2 years and only 50% will remit within three years[2,15,26]. Untreated patients suffer from unrelenting skin infections, dehydration, electrolyte imbalance and sepsis. Contrary to common belief amongst physicians that BP is a benign disease, it does carry a significant morbidity and mortality. Large population-based studies showed significantly increasing age-adjusted mortality rate of BP, but decreased for pemphigus[7,27-29]. Hospitalised patients had a mortality up to six times greater than the general population[3,4,6]. The first-year mortality rate of BP ranged from 15-41%, and up to 50% in 5 years[3,4,6,14,27-33]. The majority of deaths were due to sepsis. Poor prognostic factors include old age, low performance status, and associated medical conditions[26,28,32]. Factors relating to clinical presentation, disease extent and choice of therapy did not seem to

affect the overall prognosis[26,28,32]. Similar prognosticating factors were noted in our local study, with presence of malignancy, poor premorbid state, hypoalbuminaemia and anaemia being significant in predicting lethal outcome[13].

Management

Treatment of BP depends on the extent of the disease and the aim is to control symptoms with minimal side effects where possible. For localised BP, topical treatment with high potency corticosteroid or tacrolimus is often successful[15,33,34]. For generalised disease, options are anti-inflammatory agents, immunosuppressives or immunomodulating agents, and procedures to remove circulating pathogenic antibodies[15].

The first-line treatment of generalised BP is corticosteroid, which has been used for more than 50 years. Both systemic steroid monotherapy (prednisolone) and super-potent topical steroid (clobetasol proprionate cream) are evaluated in controlled trials with good efficacy. Prednisolone (or prednisone) at 0.75-1mg/kg/day is effective in controlling BP within 1-4 weeks in 60-90% of cases[15]. Increasing the dose beyond that does not offer additional benefits in disease control but causes complications[35,36]. For mild to moderate disease, prednisolone at 0.5mg/kg and 0.3mg/kg respectively, is recommended in the UK[15], though there is no well-evaluated initial treatment dose available in the literature. Locally, the majority of the hospitalised Chinese patients (91.7%) were given prednisolone (0.5mg/kg/day) and this regime was effective in inducing remissions within 4 weeks[13].

Regarding topical steroid, a landmark randomised-control study that showed that superpotent topical steroid (clobetasol cream 40g daily tapering over 12 months) was superior than prednisolone 0.5mg/kg in controlling extensive disease, with a better side effect profile and overall survival, at the expense of systemic absorption[37]. Recently, a milder regimen with clobetasol 10 to 30g daily tapered over 4 months, was shown to be non-inferior in efficacy, with less systemic absorption and decreased the risk of death[38]. The European practice was difficult to apply locally for practical reasons (ability of the patients or availability of carers to apply prolonged treatment).

Other adjuvant therapies involving immunosuppressants such as azathioprine, mycophenolate mofetil, methotrexate, cyclophosphamide, chlorambucil, dapsone, or tetracycline and nicotinamide combination, IVIG and plasma-exchange, were not shown to be superior to systemic steroid in terms of effectiveness[34,35], but may offer a steroid-sparing effect and minimise the side-effect profile. Of note, the tetracycline group with nicotinamide was shown in small randomised control trials to be efficacious but less toxic than systemic steroid[15]. The combination of doxycycline with topical and/or systemic steroid is used widely locally and in the UK[39] and a RCT is under way to evaluate doxycycline monotherapy with prednisolone. New emerging treatments with rituximab and omalizumab based on anecdotal reports to direct against pathogenic autoantibodies and IgE are under investigation[15].

General measures

Good skin care should be undertaken in handling patients with BP. Blisters should be left intact if possible to prevent secondary bacterial infections. In cases of large blisters affecting function, like those of the soles, they can be aspirated with sterile needles with the blister roof in place. Raw areas should be cleansed by antiseptics and covered by non-adhesive dressings[12]. Itchiness and pain should be adequately controlled by symptomatic treatment. Patients on long-term steroid should be given osteoporosis prevention (Calcium, vitamin D supplementation and bisphosphonate)[40]. Patients with underlying hepatitis B, prophylaxis with antivirals (while on immunosuppressants) and hepatology referrals are recommended

Table 2 Summary of treatment choice

For localized or mild disease	Very potent topical steroids alone (applied to lesional skin) (*strength of recommendation A*)*
	Systemic corticosteroids 0·3 mg kg^{-1} daily (weaning dose once control achieved) ± very potent topical steroids applied to lesional skin (*strength of recommendation A*)
	Anti-inflammatory antibiotics ± very potent topical steroids applied to lesional skin:
	Doxycycline 200 mg/day
	Oxytetracycline 1 g/day
	Lymecycline 408 mg twice daily
	Minocycline 100 mg/day
	Erythromycin 1–2 g/day
	(*strength of recommendation D*)
For moderate-to-severe disease	Systemic corticosteroids 0·5–1·0 mg kg^{-1} daily (weaning dose once control achieved) ± very potent topical steroids (*strength of recommendation A*)
	Very potent topical steroids 5–15 g twice daily to whole skin surface (if patient or carer is capable) (*strength of recommendation A*)
	Anti-inflammatory antibiotics ± very potent topical steroids applied to lesional skin (as above) (*strength of recommendation D*)
For disease of any severity not responding to existing treatment, or who relapse on unacceptably high doses of existing treatment	Consider switching to or the addition of:
	Systemic corticosteroids 0·5–1·0 mg kg^{-1} daily (weaning dose once control achieved) ± very potent topical steroids (*strength of recommendation A*)
	Anti-inflammatory antibiotics (as above) with or without nicotinamide 500–2500 mg daily (*strength of recommendation D*)
	Azathioprine 1–2·5 mg kg^{-1} daily (*strength of recommendation D*)
	Methotrexate 5–15 mg weekly (*strength of recommendation D*)
	Dapsone 50–200 mg daily (*strength of recommendation D*)
	Chlorambucil 0·05–0·1 mg kg^{-1} daily (*strength of recommendation D*)
For cases refractory to all the above, other modalities to be considered in exceptional circumstances	Mycophenolate mofetil 0·5–1 g twice daily
	IVIg
	Cyclophosphamide
	Plasmapheresis
	(also see main text)

Conclusion

BP is the most common autoimmune blistering disease, especially in the elderly. There is a wide range of infective, inflammatory, autoimmune, drug-induced or systemic conditions that may mimic BP. Clinico-pathological correlation with histologic and immunofluorescence confirmation is necessary before embarking on systemic treatment. Hospitalised patients with BP have multiple comorbidities and usually present with generalised involvement, more severe disease, recurrent relapses and a high mortality. There is a significant morbidity and case fatality especially in the first year. Patients with poor prognostic factors (bed-bound status, anaemic, hypoalbuminaemic, with malignancy) should be monitored closely. Careful considerations in using systemic immunosuppressants are necessary.

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Radiology Quiz

Radiology Quiz

Dr. Agnes WONG

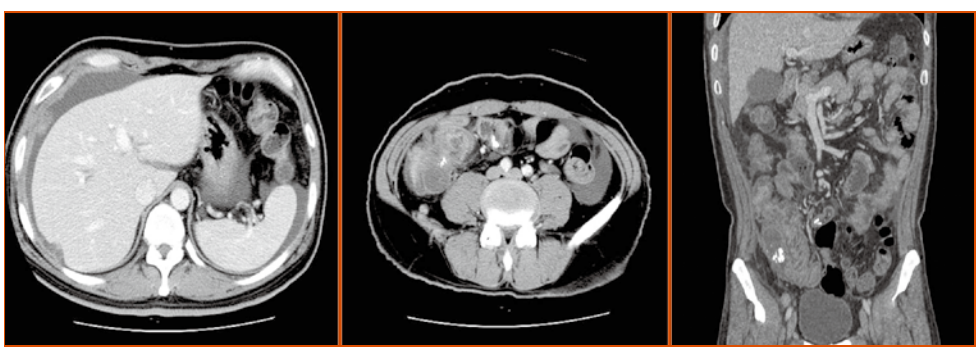
Department of Radiology, Queen Mary Hospital

Clinical information:

M/48, admitted for right lower quadrant pain for 1 week.
P/E: tenderness and guarding over right lower quadrant.
Afebrile. Normal white cell count. ? acute appendicitis.

Questions:

1. What are the findings?
2. What is the diagnosis?



(See P.33 for answers)

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- Rapid elimination of zoster-associated pain with Famvir® 250mg TID, 1.4 times faster than acyclovir 800mg PID[4]
- Significantly earlier reduction in pain with Famvir® than valacyclovir in patients aged 50 years or older[5]
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- Significantly shortened the duration of Post-Herpetic Neuralgia by half for Herpes zoster[7]
- Recurrence free after one year in 70% patients for genital herpes with Famvir® 250mg[8]

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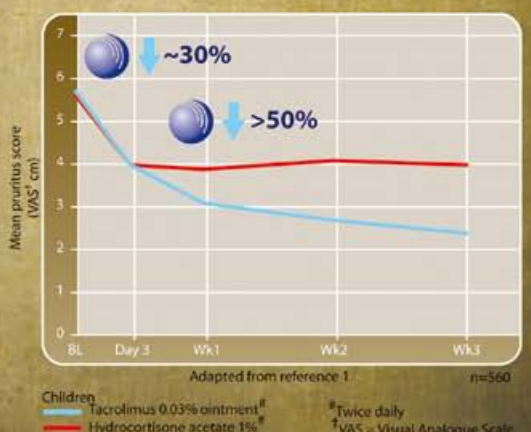

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Recent Advances in the Treatment of Pigmentary Conditions

Dr. Henry CHAN

Henry HL Chan, MBBS, MD, PhD, FRCP(London, Edin, Glas), FHKCP, FHKAM(Medicine)

Specialist in Dermatology
Hon Clinical Professor and Hon Consultant
Division of Dermatology, Department of Medicine, University of Hong Kong, Queen Mary Hospital

Dr. Henry CHAN

Tattoos can be very resistant to treatment and despite the use of QS lasers that produce high power energy with an extremely short pulse duration (nanosecond, 10-9s), the results have been sub-optimal. Other lasers have been used to further enhance the treatment outcomes including ablative or non-ablative fractional resurfacing with variable successes. The intention to combine QS lasers with fractional technology is to allow more rapid clearance via the transepidermal means. While the addition of a fractional laser immediately after QS lasers can also reduce the texture change known to be associated with lasers for tattoo removal,[1] (Figure 1) nonetheless, the degree of clearing remains unimpressive and there is a desire to look for better therapeutic measures.

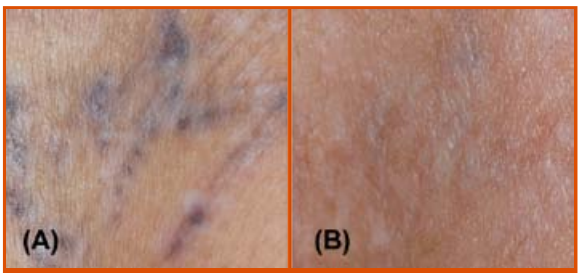

Figure 1 (A) Before treatment (B) After seven treatments with QS1064 Nd:YAG/ QS Ruby /QSAlexandrite and two treatments with non-ablative fractional resurfacing

More recently, it has been proposed to repeat treatment of the same tattoo with a QS laser after a 20 minutes interval. The intention is that immediate whitening that developed after QS lasers consists of stream pockets, generated as a result of the photothermal effect(rapid change of thermal gradient leading to heat induced cavitation and bubbles formation). Immediate whitening prevents further laser penetration into the dermis and by resting for 20 minutes before further laser exposure, it has been suggested that this can allow more rapid clearance. Recent studies did indicate better success if the same tattoo is treated 4 times at 20 minute intervals within the same session.[2] This so called R 20 method did gain initial popularity but several issues occurred. First of all, it was very time and space consuming; the results in the author's hands were not as impressive as in previous publications and the amount to bill the patient can also be controversial.

In light of the R20 approach, others have invented a R0 approach, whereby application of perfluorodecalin prior to laser irradiation has been shown to prevent bubble formation.[3] Perfluorodecalin is a liquid hydrocarbon and has a high gas solubility. It can improve optical clearance after QS laser treatment by absorbing the bubbles formed and prevent immediate whitening. Multiple passes can be performed without the need to wait for the immediate whitening to subside. It has been suggested that this is more superior than the R20 method in terms of time consumption and clinical outcome.

It has been over a decade when picosecond lasers were first found to be effective in tattoo removal.[4] By generating 10-12 s of pulse duration, a picosecond laser is more powerful than the current nanosecond lasers. It produces more significant photothermal and photomechanical effects. Despite such early proof of concept, no device has been developed commercially due to the high cost and the difficulty in producing a reliable laser. A commercially available picosecond Alexandrite laser has just been launched in the United States and recent studies indicated that after 1 to 2 treatment sessions, 75% of clearance can be achieved even for tattoos that failed to respond to previous therapy.[5] Another study did reveal the risk of pigmentary changes with post-inflammatory hyperpigmentation(PIH) developed among 13% of treated patients and hypopigmentation in 20%.[6] Given the fact that patients involved in that particular study were skin type II and III, the risk of pigmentary changes among Asians is likely to be significantly greater. The author's left forearm skin was treated with the same device last autumn and developed PIH that is still visible now. Although pigmentary disturbance can be of a concern, such a high energy device can be a game changer for dermal pigmentary conditions including naevus of Ota and Hori's macules, conditions that are particularly common among our population. Another interesting concept is the extreme photomechanical effect that can have the potential to induce skin rejuvenation. QS 0164 Nd:YAG lasers have been used for skin rejuvenation and treatment of melasma and whether picosecond laser can have similar effects remains to be seen. It is worthwhile to mention that besides the picoseconds Alexandrite laser, another company is currently developing a picosecond 532/1064 Nd:YAG laser. The use of picoseconds lasers among Asians will benefit with further investigations.

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Hiking Again

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If you have good memory, you should remember my recommendation of some hiking trails in different parts of Hong Kong in 2008. Have you started your journey yet? These trails could be found all over the territory. No matter where you live, there should be one nearby. You may find the ones in the urban regions which are more accessible and often shorter for even less experienced hikers. Or you may take a walk on longer trails in the New Territories and Lantau Island to challenge yourself.

I still remember bringing you to see a variety of spectacular rocks in 2010. Have you visited the peculiar rocks on Cheung Chau? Did you find the "Lizard", the "Elephant", the "Vase" and the "Face"? What about Poi Toi Island? Have you seen the "Flying Coffin", the "Turtle and Mock" and the "Buddha's Palm"? Are they interesting? Once you take your first step on any trail, you will enjoy the leisure of hiking and feel the greatness of nature.

This time, I would like to bring you to new attractions where nature-lovers can relax and enjoy not the rocks but the water- Bride's Pool (新娘潭) and Ng Tung Chai Waterfall (梧桐寨瀑布).

Bride's Pool 新娘潭

Bride's Pool is very renowned in Hong Kong not only for its beautiful scenery but also its sorrowful story behind. I believe most of you have heard of it. The Bride's Pool is at the middle of the Bride's Pool Road (新娘潭路) in between Tai Mei Tuk (大尾督) and Luk Keng (鹿頸). On weekdays, there are plenty of parking spaces for drivers. However, the car park is always full at weekends and holidays. Instead, we can take bus 275R at the Tai Po Market Station (大埔墟火車站), which only operates on Sundays and holidays. The Bride's Pool is situated near the Plover Cove Reservoir (船灣淡水湖) and you can follow the Bride's Pool Natural Trail (新娘潭自然教育徑) to visit it. The trail begins with a nice bridge which is located at the top of the Bride's Pool. You can exercise and enjoy nature at the same time by taking the walk with the singing birds and many other enticing insects. There are explanatory signs along the trail to introduce the local geology and ecology. Surrounding the trail is the dense woodland which protects hikers from the sunshine. Walking along the side of the stream, you can surely feast upon many amazing landscapes and the large water-eroded potholes. The whole trail is smooth and takes around an hour to complete. Therefore, it is suitable for beginners and families.

Ng Tung Chai Waterfall 梧桐寨瀑布

Ng Tung Chai Waterfall (梧桐寨瀑布) is the highest fall in Hong Kong. It is located on the north slope of Tai Mo Shan (大帽山), the highest mountain in Hong Kong. There are totally four waterfalls – starting from the lowest called the Bottom Fall to the Middle Fall, the Main Fall and the Scatter Fall at the highest. One can start by taking bus 64K at the Tai Po Market Station and then get off at the Ng Tung Chai Bus-stop. It is very easy to find the signpost that shows the direction to the falls. The first thing you can see is a traditional Chinese building called Man Tak Yuen (萬德苑). Honestly speaking, I do not know what Man Tak Yuen means. Following the route and the signpost next to Man Tak Yuen, you will get to the Bottom Fall (井底瀑). It is a short waterfall which is not very strong. Then following the pathway you can reach the Middle Fall (中瀑), which is also called the Horse Tail Fall (馬尾瀑). It looks like the tail of a horse, particularly at the end of the fall. It is around 10metres high. Although the Middle Fall is not the highest, I think it is the strongest fall and the most magnificent one, especially in summer. When you continue climbing uphill, you will soon reach the Main Fall (主瀑). It is also called the Long Fall (長瀑). It is more than 30 metres high and is the highest waterfall in Hong Kong. There is a platform nearby where I believe you can take the best photos of the Main Fall.

Path to Scatter Fall

The hiking path heading to the Scatter Fall (散髮瀑) is rough and a little bit slippery. The path is officially closed because of the landslides years ago and it is still full of small and sharp stones. However, we can still pass it with care to visit the Scatter Fall. It is a small and short waterfall and is named because it is like a scatter of a young beautiful lady's hair. At an altitude of 465 metres on Tai Mo Shan, it takes about 3 to 4 hours to reach the Scatter Fall from the bus-stop. After visiting the Scatter Fall, you can return to the bus-stop on the same route. If you still have surplus energy, you can continue climbing upwards to reach the top of Tai Mo Shan. It takes another 3 to 4 hours. However this is not meant for city people who seldom do exercises. So you had better get well prepared before taking the challenge to reach its top and it is suggested that you take it slowly.

"Playing with hills and never with water" (嬉山莫嬉水) should always be in your mind. Although the government has constructed the hiking trails along the beautiful falls, you should take note of the weather or any special announcement on flooding if you go in rainy seasons.

Main Fall

Middle Fall

Bottom Fall

Lizard Island Queensland 昆士蘭

Lizard Island 被 Australian Gourmet Traveller 2012 Travel Awards 評為「澳洲最佳渡假村」，位於世界十大渡假及潛水勝地著名的大堡礁上。旅客可欣賞到最蔚藍清澈的海天美景。渡假村提供各種珊瑚礁和水上活動，包括潛水到著名的鱈洞 (Cod Hole) 或從海灘直接浮潛，欣賞珊瑚花園和顏色鮮豔的熱帶魚，體驗充滿活力的大堡礁。[Lizard Island 四日兩夜國泰套票連膳食由 $22,500 起 (經濟倉) / $45,600起 (商務倉)] *

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[Longitude 131° 四日兩夜國泰套票連膳食及導賞遊由 $22,600起 (經濟倉) / $53,700起 (商務倉)]*

*套票價格以最少二人成行，包括機票、酒店及膳食，有關稅項及燃油附加費不包括在內。有效期由即日至2013年12月15日。品味遊保留最終之決定權。

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
						★ MPS Workshop - Mastering Your Risk **1**
★ HKMA Table -Tennis Tournament 2013 (Day 1) ★ MPS Workshop - Mastering Difficult Interactions with Patients **2**	★ Cutting edge: (1) Learning points and critique from AUA (2) Exposure or encounter? **3**	★ HKMA Kowloon West Community Network - Third Session of the Certificate Course on Alzheimer's Disease: Dementia Case Demonstration ★ HKMA Tai Po Community Network - Management of COPD and Asthma ★ FMSHK Officers' Meeting ★ HKMA Council Meeting **4**	★ HKMA Shatin Doctors Network - From Hypertension to Coronary Artery Disease ★ HKMA Central, Western & Southern Community Network - Third Session of the Certificate Course on Orthopaedics: Osteoarthritis of Major Joints and Management ★ MPS Workshop - Mastering Shared Decision Making **5**	★ HKMA Hong Kong East Community Network - Primary Prevention of Allergy ★ MPS Workshop - Mastering Professional Interactions **6**	**7**	★ Refresher Course for Health Care Providers 2012/2013 ★ HKMA Trip to Inner Mongolia for young doctors and medical students **8**
★ HKMA Trip to Inner Mongolia for young doctors and medical students **9**	★ HKMA Trip to Inner Mongolia for young doctors and medical students **10**	★ HKMA Trip to Inner Mongolia for young doctors and medical students ★ HKMA Tai Po Community Network - How to Improve Pediatric Allergic Rhinitis and Asthma? **11**	★ HKMA Trip to Inner Mongolia for young doctors and medical students ★ Tuen Ng Dragon Boat Race **12**	★ HKMA Kowloon East Community Network - Management of Acne Vulgaris and Acne Scar ★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2013 – Latest Development in Corneal Transplantation **13**	**14**	★ HKMA Kowloon East Community Network – Second Session of the CME Course for Health Personnel 2013: Management of Degenerative Joint Diseases in Primary Care Setting ★ 9th Annual General Meeting cum Scientific Seminars **15**
★ HKMA Table-Tennis Tournament 2013 (Day 2) ★ Hong Kong Primary Care Conference **16**	**17**	★ HKMA Kowloon West Community Network – Final Session of the Certificate Course on Alzheimer's Disease: Drug Therapy and Non-pharmacological Intervention for Dementia ★ HKMA Tai Po Community Network - Management of Allergic Rhinitis and its Comorbidity **18**	★ MPS Workshop – Mastering Difficult Interactions with Patients **19**	★ MPS Workshop – Mastering Shared Decision Making ★ FMSHK Executive Committee Meeting **20**	**21**	★ Certificate Course on Bringing Better Health to Our Community 2013 (Session 2) - Management of Thrombocytopenia in Primary Care Setting; Transient Ischemic Attack – What Can be Done in Primary Care ★ MPS Workshop – Mastering Professional Interactions **22**
★ FMSHK Annual Scientific Meeting 2013 - Obesity related disorders: an emerging epidemic **23** ★ HKMA Tenpin Bowling Tournament 2013 **30**	★ Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013 **24**	★ Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013 **25**	★ Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013 ★ HKMA Central, Western & Southern Community Network - Final Session of the Certificate Course on Orthopaedics: Keyhole Spine Surgery ★ MPS Workshop – Mastering Your Risk **26**	★ Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013 ★ MPS Workshop – Mastering Adverse Outcomes **27**	★ Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013 ★ HKMA Yau Tsim Mong Community Network – Update on Long Term Follow-up and Real Life Effectiveness Studies of Quadrivalent HPV Vaccine **28**	★ The 6th Annual Scientific Meeting and 7th Annual General Meeting of the Hong Kong Society for Paediatric Immunology and Infectious Diseases (Bill Marshall & Roland Levinsky Memorial Lectures) **29**

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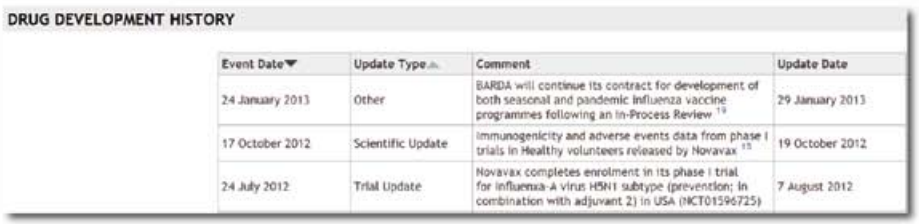

Date / Time	Function	Enquiry / Remarks
1 SAT 2:00 pm	**MPS Workshop - Mastering Your Risk** Organiser: The Hong Kong Medical Association, Speaker: Dr. Lee Wai Hung, Danny, Venue: Eaton Hotel	HKMA CME Dept Tel: 2527 8452 2.5 CME points
2 SUN 2:00 pm	**HKMA Table -Tennis Tournament 2013 (Day 1)** Organiser: The Hong Kong Medical Association, Chairman: Dr. KOO Hok Tin, Hilton, Venue: HKBU Wai Hang Sports Centre	Mr. Andie HO Tel: 2527 8285
2:30 pm	**MPS Workshop - Mastering Difficult Interactions with Patients** Organiser: The Hong Kong Medical Association, Speaker: Dr. Cheng Ngai Shing, Justin, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 2.5 CME points
3 MON 7:30 pm	**Cutting edge: (1) Learning points and critique from AUA (2) Exposure or encounter?** Organiser: HKMA Kowloon West Community Network & Hong Kong Alzheimer's Disease Association, Chairman: Dr. LEUNG Gin Pang, Speaker: Dr. CHAN Chun Chung, Ray, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Ms. Tammy HUNG Tel: 9609 6064 1 CME point
4 TUE 1:00 pm	**HKMA Kowloon West Community Network - Third Session of the Certificate Course on Alzheimer's Disease: Dementia Case Demonstration** Organiser: HKMA Kowloon West Community Network & Hong Kong Alzheimer's Disease Association, Chairman: Dr. LEUNG Gin Pang, Speaker: Dr. CHAN Chun Chung, Ray, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Hana YEUNG Tel: 2527 8285 1 CME point
1:45 pm	**HKMA Tai Po Community Network - Management of COPD and Asthma** Organiser: HKMA Tai Po Community Network, Speaker: Dr. NG Kin Chung, Alvin, Venue: Chiuchow Garden Restaurant, Shop 001-003, 1/F, Uptown Plaza, No.9 Nam Wan Road, Tai Po	Ms. Ivy LEUNG Tel: 3189 8782 1 CME point
8:00 pm	**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. Nancy CHAN Tel: 2527 8898
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. TSE Hung Hing, Venue: HKMA Head Office (5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
5 WED 1:00 pm	**HKMA Shatin Doctors Network - From Hypertension to Coronary Artery Disease** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Dr. WU Chee Wo, Venue: Marriott Hong Kong, Shatin	Mr. Ethan WONG Tel: 9866 4038
1:00 pm	**HKMA Central, Western & Southern Community Network - Third Session of the Certificate Course on Orthopaedics: Osteoarthritis of Major Joints and Management** Organiser: HKMA Central, Western & Southern Community Networ, Chairman: Dr. TSANG Chun Au, Speaker: Dr. NG Tze Pui, Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Ms. Hana YEUNG Tel: 2527 8285 1 CME point
6:30 pm	**MPS Workshop - Mastering Shared Decision Making** Organiser: The Hong Kong Medical Association, Chairman: , Speaker: Dr. Fung Shu Yan, Anthony, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 2.5 CME points
6 THU 1:00 pm	**HKMA Hong Kong East Community Network - Primary Prevention of Allergy** Organiser: HKMA Hong Kong East Community Network, Chairman: Dr. KONG Wing Ming, Henry, Speaker: Dr. Alfred TAM, Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, HK	Ms. Candice TONG Tel: 2527 8285 1 CME point
6:30 pm	**MPS Workshop - Mastering Professional Interactions** Organiser: The Hong Kong Medical Association, Speaker: Dr. Hau Ka Lam, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 2.5 CME points
8 SAT 2:30 pm	**Refresher Course for Health Care Providers 2012/2013** Organiser: , Chairman: , Speaker: Dr. LO Kwok Man, Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara Tsang Tel: 2354 2440 2 CME points
(9,10,11,12)	**HKMA Trip to Inner Mongolia for young doctors and medical students** Organiser: The Hong Kong Medical Association, Venue: Beijing and Inner Mongolia	Miss Phoebe WONG Tel: 2527 8285
11 TUE 1:45 pm	**HKMA Tai Po Community Network - How to Improve Pediatric Allergic Rhinitis and Asthma?** Organiser: HKMA Tai Po Community Network, Speaker: Prof. WONG Wing Kin, Gary, Venue: Chiuchow Garden Restaurant, Shop 001-003, 1/F, Uptown Plaza, No.9 Nam Wan Road, Tai Po	Ms. Ivy LEUNG Tel: 3189 8782 1 CME point
12 WED 8:00 am	**Tuen Ng Dragon Boat Race** Organiser: Shatin Sports Association Limited, Chairman: Dr. YAM Chun Yin, Abraham, Venue: Shatin Riverside	Ms. Dorothy KWOK Tel: 2527 8285
13 THU 1:00 pm	**HKMA Kowloon East Community Network - Management of Acne Vulgaris and Acne Scar** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. AU Ka Kui, Gary, Speaker: Dr. CHIU Lai Shan, Mona, Venue: Lei Garden Restaurant, Shop no. L5-8, apm, Kwun Tong, No. 418 Kwun Tong Road, Kowloon	Miss Hana YEUNG Tel: 2527 8285 1 CME point
2:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2013 – Latest Development in Corneal Transplantation** Organiser: The Hong Kong Medical Association, Speaker: Dr. Cheng Chak Kwan, Arthur, Venue: Function Room A, HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 1 CME point
15 SAT 1:30 pm	**HKMA Kowloon East Community Network – Second Session of the CME Course for Health Personnel 2013: Management of Degenerative Joint Diseases in Primary Care Setting** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. David Chao, Speaker: Dr. Ho Hon Shuen, Venue: Lecture Theatre, G/F, Block P, United Christian Hospital	Ms. Marina PUN Tel: 3513 4888 1.5 CME points
2:15 pm	**9th Annual General Meeting cum Scientific Seminars** Organiser: Hong Kong Society for Quality of Life, Chairman: Dr. Daniel YT Fong, Speakers: Prof. Feng-bin LIU& Dr. Winnie KW SO, Venue: Alumni Chamber, 7/F, William MW Mong Block, LKS Faculty of Medicine, The University of Hong Kong, 21 Sassoon Road, Pokfulam, Hong Kong	Mr. Andrew KWAN Tel: 6112 0106 1.5 CNE points
16 SUN 2:00 pm	**HKMA Table-Tennis Tournament 2013 (Day 2)** Organiser: The Hong Kong Medical Association, Chairman: Dr. KOO Hok Tin, Hilton, Venue: HKBU Wai Hang Sports Centre	Mr. Andie HO Tel: 2527 8285

Date / Time	Function	Enquiry / Remarks
16 SUN 8:15 am	**Hong Kong Primary Care Conference** Organiser: The Hong Kong College of Family Physicians, Chairmen: Dr. Lorna NG & Dr. William WONG, Venue: HKAM Jockey Club Building	Ms. Crystal YUNG Tel: 2861 0220
18 TUE 1:00 pm	**HKMA Kowloon West Community Network – Final Session of the Certificate Course on Alzheimer's Disease: Drug Therapy and Non-pharmacological Intervention for Dementia** Organiser: HKMA Kowloon West Community Network & Hong Kong Alzheimer's Disease Association, Chairman: Dr. CHAN Siu Man, Bernard, Speaker: Dr. TAM Kui Fu, Stanley, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Hana YEUNG Tel: 2527 8285 1 CME point
1:45 pm	**HKMA Tai Po Community Network - Management of Allergic Rhinitis and its Comorbidity** Organiser: HKMA Tai Po Community Network, Speaker: Dr. WONG Han Qian, Venue: Chiuchow Garden Restaurant, Shop 001-003, 1/F, Uptown Plaza, No.9 Nam Wan Road, Tai Po	Ms. Ivy LEUNG Tel: 3189 8782 1 CME point
19 WED 6:30 pm	**Hong Kong Primary Care Conference** Organiser: The Hong Kong College of Family Physicians, Chairmen: Dr. Lorna NG & Dr. William WONG, Venue: HKAM Jockey Club Building	HKMA CME Dept Tel: 2527 8452 2.5 CME points
20 THU 6:30 pm	**MPS Workshop – Mastering Shared Decision Making** Organiser: The Hong Kong Medical Association, Speaker: Dr. Fung Shu Yan, Anthony, Venue: Eaton Hotel	HKMA CME Dept Tel: 2527 8452 2.5 CME points
	FMSHK Executive Committee Meeting Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Nancy CHAN Tel: 2527 8898
22 SAT 1:00 pm	**Certificate Course on Bringing Better Health to Our Community 2013 (Session 2) - Management of Thrombocytopenia in Primary Care Setting; Transient Ischemic Attack – What Can be Done in Primary Care** Organiser: HKMA Yau Tsim Mong Community Network and Department of Family Medicine & General Outpatient Clinic and Department of Medicine, Kowloon Central Cluster, Speaker: Dr. CHAN Chi Chung; Dr. LO Wai Ting, Joyce, Venue: Block M, Lecture Theatre, Queen Elizabeth Hospital,30 Gascoigne Road, Kowloon, Hong Kong	Ms. Candice TONG Tel: 2527 8285
2:30 pm	**MPS Workshop – Mastering Professional Interactions** Organiser: The Hong Kong Medical Association, Speaker: Dr. Lee Wai Hung, Danny, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 2.5 CME points
23 SUN 9:30 am	**FMSHK Annual Scientific Meeting 2013 - Obesity related disorders: an emerging epidemic** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Ballroom, 3/F, Sheraton Hotel, 20 Nathan Road, Kowloon	FMSHK Secretariat Tel: 2527 8898
24 MON 9:00 am *(25,26,27,28)*	**Hong Kong Infectious Disease Week (Adult Infectious Disease Seminar & Paediatric Infectious Disease and Immunology Course 2013** Organiser: Hospital Authority Infectious Disease Centre, Infectious Disease Control Training Centre of Hospital Authority, Princess Margaret Hospital, Hong Kong Society for Infectious Diseases & Hong Kong Society for Paediatric Immunology and Infectious Diseases, Chairmen: Dr. Owen Tsang & Dr. C W Leung, Speakers: Prof. Matthew E Falagas, Prof. William Hope, Dr. Mark Nelson, Prof. Walter A Orenstein, Prof. David Isaacs, Dr. E Graham Davies & Dr. Delane Shingadia, Venue: Lecture Theatre, 7/F, Block H, Princess Margaret Hospital	Dr. Mike Kwan Tel: 2990 2872
26 WED 1:00 pm	**HKMA Central, Western & Southern Community Network - Final Session of the Certificate Course on Orthopaedics: Keyhole Spine Surgery** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. LAW Yim Kwai, Speaker: Dr. LEUNG Hin Shuen, Clarence, Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME point
6:30 pm	**MPS Workshop – Mastering Your Risk** Organiser: The Hong Kong Medical Association, Speaker: Dr. Hau Ka Lam, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept Tel: 2527 8452 2.5 CME points
27 THU 6:30 pm	**MPS Workshop – Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association, Speaker: Dr. Hung Chi Wan, Emily, Venue: Eaton Hotel	HKMA CME Dept Tel: 2527 8452 2.5 CME points
28 FRI 1:00 pm	**HKMA Yau Tsim Mong Community Network – Update on Long Term Follow-up and Real Life Effectiveness Studies of Quadrivalent HPV Vaccine** Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr. LAU Tai Wah, Venue: Jade Ballroom, Level 2, Eaton Smart, Hong Kong380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285 1 CME point
29 SAT 12:30 pm	**The 6th Annual Scientific Meeting and 7th Annual General Meeting of the Hong Kong Society for Paediatric Immunology and Infectious Diseases (Bill Marshall & Roland Levinsky Memorial Lectures)** Organiser: Hong Kong Society for Paediatric Immunology and Infectious Diseases, Chairman: Dr. CW Leung, Speakers; Dr. Delane Shingadia & Dr. Graham Davies, Venue: Centenary Ballroom, The Marco Polo Hong Kong Hotel, Tsimshatsui	Dr Mike KWAN Tel: 2990 2872
30 SUN 2:00 pm	**HKMA Tenpin Bowling Tournament 2013** Organiser: The Hong Kong Medical Association, Chairman: Dr. HO King Yip, Anthony, Venue: South China Athletic Association	Mr. Andie HO Tel: 2527 8285

Upcoming Meeting

10-13/7/2013	**9th Asian Dermatological Congress 2013** Organisers: Asian Dermatological Association, Hong Kong College of Dermatologists & the Hong Kong Society of Dermatology and Venereology, Chairman: Prof. Henry HL CHAN, Venue: Hong Kong Convention & Exhibition Centre, Enquiry: ADC 2013 Secretariat Tel: 3151 8900

Answers to Radiology Quiz

Answers:

1. - An irregular multiloculated hypodense mass is seen arising from the caecum, with multiple internal calcifications. Terminal ileum appears oedematous. Normal appendix is not seen.

   - Multiple soft tissue density masses are noted over perihepatic, subhepatic spaces, right para-colic gutter and mesenteric regions. These could represent peritoneal deposits. Scalloping of liver contour is also seen. Small amount of ascitic fluid with increased mesenteric stranding noted.

   - No focal liver masses. Gallbladder, pancreas, spleen, kidneys, adrenals appear normal. Psoas muscles are unremarkable.

   - No pneumoperitoneum.

2. Overall features are suggestive of CA appendix with peritoneal metastases and pseudomyxoma peritonei, which is confirmed on operation.

Dr. Agnes WONG
Department of Radiology, Queen Mary Hospital

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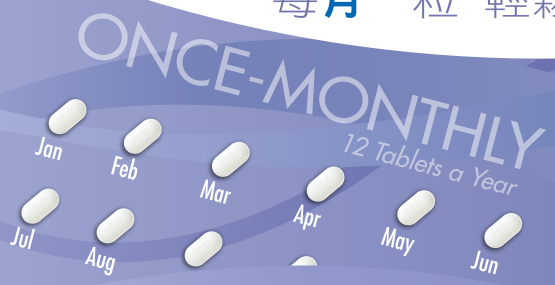
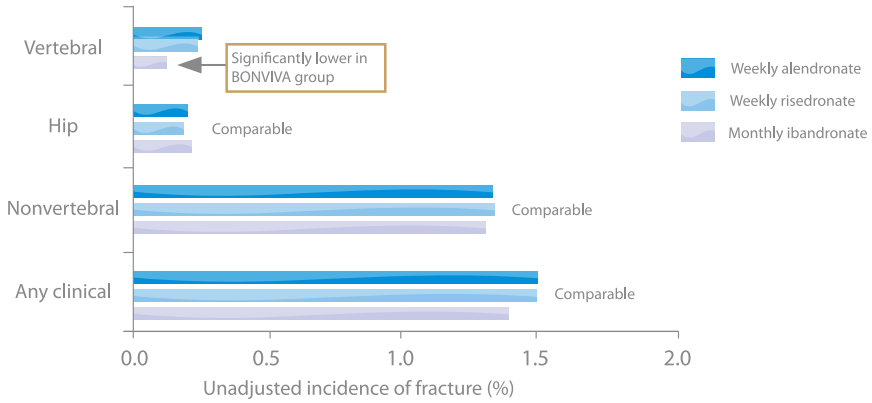


The VIBE* Study

Proven efficacy: Once-monthly Bonviva vs. weekly bisphosphonates (BP)

One year retrospective observational fracture study[1]



Bonviva-treated patients had statistically lower incidence of vertebral fractures.[1]

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*The eValuation of IBandronate Efficacy (VIBE) study was a retrospective claims database study with a 12-month observational period that included women ≥45 years of age (n=64,182), newly prescribed monthly oral ibandronate (Bonviva) (n=7345) or weekly oral BPs (alendronate 35 mg or 70 mg, or risedronate 35 mg) (56,837) for a period between April 1, 2005 and December 31, 2005. Ref:1. Bone. 2009;44:758–765. **Full prescribing information available upon request**

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Unit 3706-8, 37/F, AIA Tower 183 Electric Rd North Point Hong Kong 香港北角電氣道183號友邦廣場37樓3706-8室
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