THE HONG KONG 香港醫訊
MEDICAL DIARY
www.fmshk.org
VOL.15 NO.5 MAY 2010

Update in Management of Psoriasis & Psoriatric Arthopathy

OFFICIAL PUBLICATION FOR THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG ISSN 1812 - 1691

Blue Cross 藍十字
Member of BEA Group

Medical Professional Protection

A NEW CHOICE of insurance to meet your needs at last!

Free 1-year Travel Insurance (worth HK$1,680)

Blue Cross is proud to present you the *Medical Professional Protection Insurance* (MPPI), a new option of MPL insurance specially tailored for physicians registered in Hong Kong based on the local malpractice situation.

Key Benefits:

- Up to 30% premium saving [1]
- Fixed premium rate for the first 2 years [2]
- Choice of 3 protection limits [3] to meet your practice's needs
- No claim discount [4], even for new enrolment
- Early bird privilege of a full-year comprehensive travel insurance for free till 30 Sept 2010 [5]
- Comprehensive coverage for medical professional liability, public relations cost, medical data, accidental death and dismemberment, and needle-stick injury

Visit our website for more information, to apply or get a quote. Alternatively, you can simply call our representative at **3608 2869** to arrange a meeting for discussion.

Medical Professional Protection Insurance is a product developed by Blue Cross in collaboration with Aon and Allied World Assurance Company. Aon is a leading insurance broker for a diverse range of professional indemnity insurance solutions. Allied World is a global reinsurer specialising in healthcare liability insurance.

[1] The percentage of premium saving will vary depending on the chosen protection limit, specialty, years of experience and claim history of the applicant. [2] Eligible policies must take effect on or before 28 February 2011 and policyholders who have reported claims under the policy within the 1st policy year will not be entitled to this promotional offer. [3] 3 levels of protection limit: HK$25 million, HK$50 million and HK$75 million per claim with respective annual aggregate protection amounts up to HK$50 million, HK$100 million and HK$150 million in any one policy year. [4] The percentage of no claim discount will vary depending on the specialty, years of experience and claim history of the applicant or policyholder. [5] The minimum required premium fee will be HK$10,000.

40th Anniversary

Hotline: 3608 2869 **Fax: 3608 2990** **Website: www.bluecross.com.hk/mppinsurance**

Contents

Editorial

- **Update in the Management of Psoriatic Arthropathy** 3
  Dr. Mo-yin MOK

Medical Bulletin

- **The Clinical Spectrum and Diagnosis of Psoriatic Arthropathy** CME 5
  Dr. Mo-yin MOK
- **MCHK CME Programme Self-assessment Questions** 8
- **Psoriasis** 10
  Dr. King-man HO
- **Manifestation of Radiological Abnormalities in Psoriatic Arthritis** 17
  Dr. Y. WONG
- **Overcoming the Stigmata of Psoriasis** 22
  Prof. Peter WH LEE
- **Biologic Therapies for Psoriatic Arthritis** 26
  Dr. Gavin Ka-wing LEE

Life Style

- **Travels** 28
  Prof. CL LAI
- **Running Asia - Chronicles of a Runner's Wife** 30
  Dr. Siaw Ing YEO

Radiology Quiz

- **Radiology Quiz** 20
  Dr. WK TSO

Federation News 32

Society News 33

Medical Diary of May 34

Calendar of Events

- **Meetings** 36

Disclaimer

The Cover Shot

Takayama - November 2009

The Takayama air gleams sparkling cold;
Strange autumn light streams through small maple leaves,
Which glow in glorious orange, red and gold,
While morning mist drips amongst ancient eaves.

The traveller wanders through Shinto shrines,
Whose columns echo the boldly tinted trees,
Matching red with red, each with each refines,
Defining mystic possibilities.

Then fracturing the silent tranquil scene,
The mountain river rumbles busily,
People bustle through old streets with old routine,
While time fumbles with eager certainty.

Yet 'neath the darkly glistening maple shades,
Serenity returns as chaos fades!

Prof. CL LAI

Approved Indications: RA, PSA AND AS

Look at that!

Look at the difference
you can make with HUMIRA

P-3799-L-1700

*References: (1) Humira prescribing information. (2) Data on file Abbott Laboratories.

HUMIRA®
adalimumab

Make the difference

Over 10 years of clinical experience*

Abbott Laboratories Limited
20/F., AIA Tower, 183 Electric Road,
North Point, Hong Kong
www.HUMIRA.com

Tel: 2566 8711
Fax: 2219 8066
Further Information is available upon request.

Abbott
A Promise for Life

Published by
The Federation of Medical Societies of Hong Kong

Design and Production
A-PRO MULTIMEDIA www.apro.com.hk

Update in the Management of Psoriatic Arthropathy

Dr. Mo-yin MOK

MBBS(HK), MRCP(UK), FHKCP, FHKAM, FRCP(Glas), FRCPA(Australia)
Assistant Professor, Division of Rheumatology, Department of Medicine, Queen Mary Hospital
Editor

Dr. Mo-yin MOK

Skin psoriasis is a chronic inflammatory cutaneous condition that is commonly encountered in clinical practice. This condition may sometimes be mistaken as eczema. In this issue, we will read about the various types of psoriatic lesions, the differential diagnoses and the available treatment options.

The impact of psoriasis on its sufferers is huge. Patients who have extensive skin involvement often have impaired quality of life. Stigmatisation is a central experience in these patients with broad psychological and social impact. Recognition of their psychological stress is essential as this may be linked to treatment non-compliance and hence worsening of status of the psoriasis. In this issue, we will also read about the psychosocial distress experienced by these patients and the ways to alleviate their psychological burden.

About one-third of patients with skin psoriasis may also have concomitant psoriatic arthropathy. These patients are commonly characterised by the presence of nail dystrophy. In this issue, we will read about the five clinical subtypes of psoriatic arthropathy. In those patients who present with rheumatoid arthritis pattern of joint distribution, the diagnosis of psoriatic arthropathy can be missed if the slightest attention has not been given to the tiny skin plaques over the extensor aspect of the elbows, over the nuchal region covered by long hair in girls and marks of post-inflammatory hyperpigmentation of treated skin lesions over the limbs. Radiological features such as periosteal bony proliferation, osteolysis and ankylosis often help in the diagnosis of psoriatic arthropathy. In this issue, we will learn how to interpret plain radiographic findings of psoriatic arthropathy and the use of various imaging modalities in the management of this disease and the associated musculoskeletal conditions.

Over a few decades, topical therapy including corticosteroids and keratolytics has been the conventional treatment of skin psoriasis. Phototherapy and systemic agents are reserved for patients with extensive disease. In this "biologic era" in the field of rheumatology, biologic therapies targeted against various inflammatory mediators with higher efficacy than conventional therapy are now available for the treatment of psoriasis and psoriatic arthropathy. In this issue, we will read about the efficacy and safety of anti-tumour necrosis factor-alpha therapy and the recently available monoclonal antibodies against interleukin-12/interleukin-23 in the treatment of this disease, thus offering more hope to these patients.

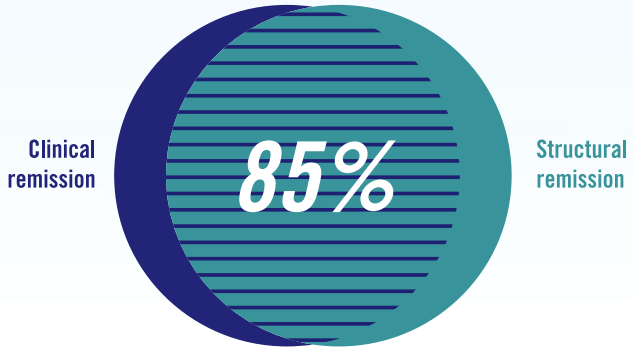
Hope that you enjoy reading these articles.

Enbrel® etanercept 恩利®

Leading the way to remission - your patients can achieve long-term success

Achieve remission in active early RA and long-standing RA*

- 85% of ENBREL + MTX patients achieving DAS remission (DAS <1.6) also achieved structural (radiographic) remission (change in total Sharp score ≤0.5)[1]
- **In the COMET trial at 52 weeks,** 50% and 80% of patients receiving Enbrel + MTX achieved clinical and structural remission (change in modified total Sharp Score ≤0.5) respectively.[2]



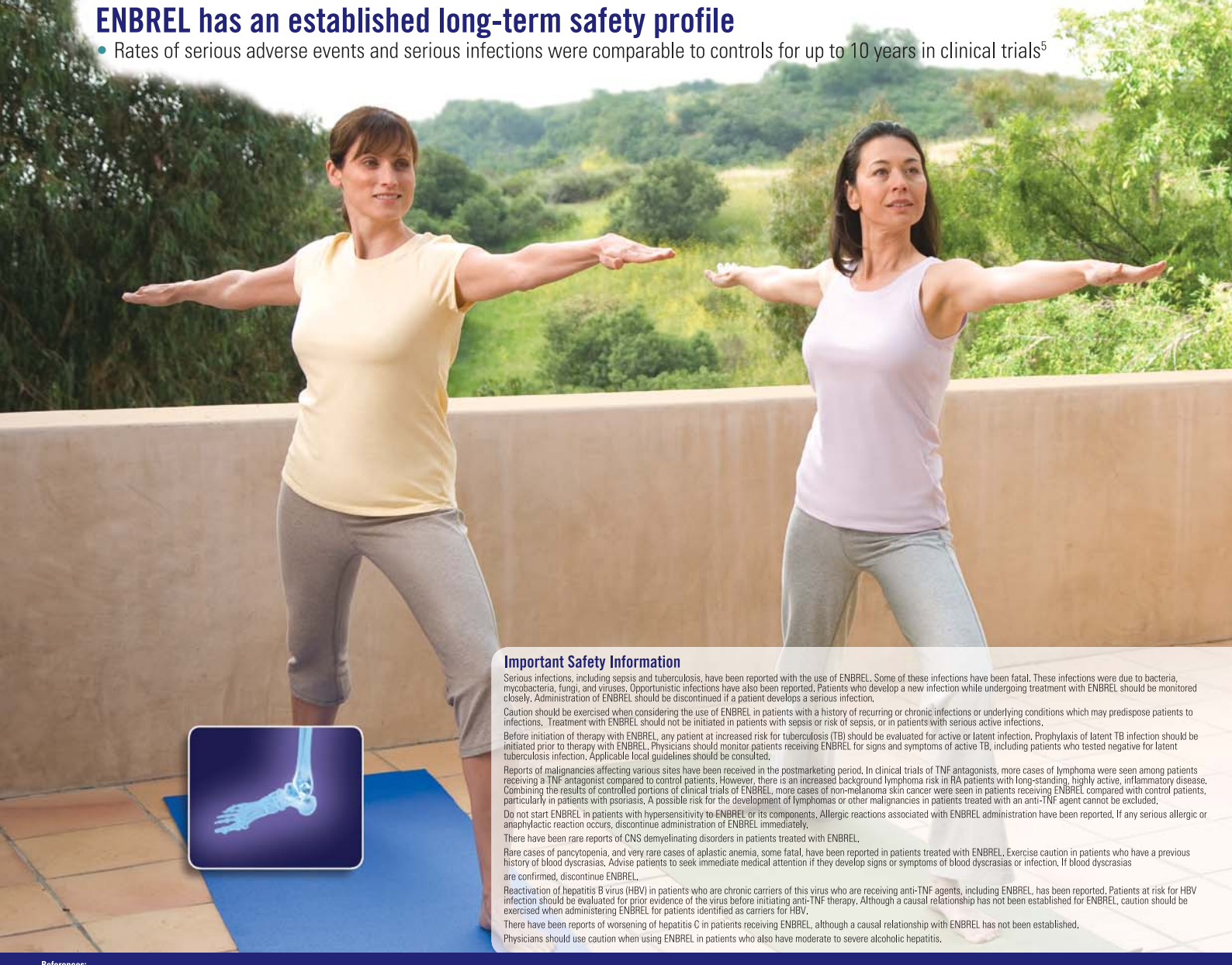
* Early RA: Median time for RA diagnosis to randomization was 7 months. Long-standing RA: Patients who had failed at least 1 DMARD other than MTX; median time for RA diagnosis to randomization was 5.3 years.

A unique mechanism of action

- ENBREL is a fully human soluble tumor necrosis factor (TNF) receptor[3]
- ENBREL is not associated with the production of neutralizing antibodies[3]
- Neutralizing antibodies may be associated with lower response rates[4]

ENBREL has an established long-term safety profile

- Rates of serious adverse events and serious infections were comparable to controls for up to 10 years in clinical trials[5]



Important Safety Information

Serious infections, including sepsis and tuberculosis, have been reported with the use of ENBREL. Some of these infections have been fatal. These infections were due to bacteria, mycobacteria, fungi, and viruses. Opportunistic infections have also been reported. Patients who develop a new infection while undergoing treatment with ENBREL should be monitored closely. Administration of ENBREL should be discontinued if a patient develops a serious infection.

Caution should be exercised when considering the use of ENBREL in patients with a history of recurring or chronic infections or underlying conditions which may predispose patients to infections. Treatment with ENBREL should not be initiated in patients with sepsis or risk of sepsis, or in patients with serious active infections.

Before initiation of therapy with ENBREL, any patient at increased risk for tuberculosis (TB) should be evaluated for active or latent infection. Prophylaxis of latent TB infection should be initiated prior to therapy with ENBREL. Physicians should monitor patients receiving ENBREL for signs and symptoms of active TB, including patients who tested negative for latent tuberculosis infection. Applicable local guidelines should be consulted.

Reports of malignancies affecting various sites have been received in the postmarketing period. In clinical trials of TNF antagonists, more cases of lymphoma were seen among patients receiving a TNF antagonist compared to control patients. However, there is an increased background lymphoma risk in RA patients with long-standing, highly active, inflammatory disease. Combining the results of controlled portions of clinical trials of ENBREL, more cases of non-melanoma skin cancer were seen in patients receiving ENBREL compared with control patients, particularly in patients with psoriasis. A possible risk for the development of lymphomas or other malignancies in patients treated with an anti-TNF agent cannot be excluded.

Do not start ENBREL in patients with hypersensitivity to ENBREL or its components. Allergic reactions associated with ENBREL administration have been reported. If any serious allergic or anaphylactic reaction occurs, discontinue administration of ENBREL immediately.

There have been rare reports of CNS demyelinating disorders in patients treated with ENBREL.

Rare cases of pancytopenia, and very rare cases of aplastic anemia, some fatal, have been reported in patients treated with ENBREL. Exercise caution in patients who have a previous history of blood dyscrasias. Advise patients to seek immediate medical attention if they develop signs or symptoms of blood dyscrasias or infection. If blood dyscrasias are confirmed, discontinue ENBREL.

Reactivation of hepatitis B virus (HBV) in patients who are chronic carriers of this virus who are receiving anti-TNF agents, including ENBREL, has been reported. Patients at risk for HBV infection should be evaluated for prior evidence of the virus before initiating anti-TNF therapy. Although a causal relationship has not been established for ENBREL, caution should be exercised when administering ENBREL for patients identified as carriers for HBV.

There have been reports of worsening of hepatitis C in patients receiving ENBREL, although a causal relationship with ENBREL has not been established.

Physicians should use caution when using ENBREL in patients who also have moderate to severe alcoholic hepatitis.

References:

1.van der Heijde D, Klareskog L, Landewe R, et al. Disease remission and sustained halting of radiographic progression with combination etanercept and methotrexate in patients with rheumatoid arthritis. Arthritis Rheum. 2007;56:3928-3939.

2.Emery P, Breedveld FC, Hall S, et al. Comparison of Methotrexate Monotherapy with a combination of Methotrexate and etanercept in active, early, moderate to severe rheumatoid arthritis (COMET): a randomised, double-blind, parallel treatment trial. Lancet 2008; 372:375-82

3.ENBREL Summary of Product Characteristics. Wyeth Pharmaceuticals.

4.Bartelds GM, Wijbrandts CA, Nurmohamed MT, et al. Clinical response to adalimumab: relationship to anti-adalimumab antibodies and serum adalimumab concentrations in rheumatoid arthritis. Ann Rheum Dis, Jul 2007; 66:921-26.

5.Weinblatt ME, Genovese MC, Moreland LW, et al. Efficacy and safety for up to 10 years of etanercept (ENBRELR) therapy in North American patients with early and long-standing rheumatoid arthritis. Arthritis Rheum. Poster presented at the annual meeting of the American Academy of Dermatology; February 1-5, 2008. San Antonio, Texas.

Pfizer

Pfizer Corporation Hong Kong Limited
16/F., Stanhope House, 738 King's Road, North Point, Hong Kong.
Tel: (852) 2811 9711 Fax: (852) 2579 0599
http://www.pfizer.com.hk

ENB-AD-802-2401-09

The Clinical Spectrum and Diagnosis of Psoriatic Arthropathy

Dr. Mo-yin MOK

MBBS(HK), MRCP(UK), FHKCP, FHKAM, FRCP(Glas), FRCPA(Australia)
Assistant Professor, Division of Rheumatology, Department of Medicine, Queen Mary Hospital

Dr. Mo-yin MOK

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 31 May 2010.

Psoriasis is a chronic skin condition which affects 2-3% of the population in Europe and the United States. Psoriatic arthritis (PsA) is an inflammatory arthritis associated with psoriasis. PsA is classified with the spondyloarthropathy (SpA) since these patients may present with spondylitis. However, other than the axial skeleton and the sacroiliac joints, PsA may also affect peripheral joints. PsA occurs in 6-39% of patients with psoriasis[1] compared to the prevalence of 0.5-1% of rheumatoid arthritis (RA), a prototypic inflammatory arthritis that affects peripheral joints. Unlike RA that shows a female preponderance, the gender ratio among PsA patients was almost equal with peak age of disease onset between 30-40 years.

Clinical Spectrum of PsA

Wright and Moll first described in 1876 the five patterns of PsA: (i) distal arthritis involving the distal interphalangeal (DIP) joints, (ii) oligoarthritis (<5 tender and swollen joints) involving small or medium-sized joints in an asymmetric distribution, (iii) a symmetric polyarthritis resembling RA, (iv) arthritis mutilans which is a deforming, destructive and disabling form of arthritis and (v) a SpA. Table 1 shows the reported frequency of different forms of PsA extracted from the largest cohort in the literature[2]. However, the joint pattern changes with time in over 60% of patients[3] and these patterns may also overlap in patients with longstanding disease.

Table 1. Distribution of different patterns of PsA from a cohort of 220[2]

Joint pattern of PsA	Percentage
Distal interphalangeal joints	12
Oligoarthritis	14
Polyarthritis	40
Spondyloarthritis	2
Arthritis mutilans	16

Nail Dystrophy in PsA

The clinical features of the five patterns of PsA may also be found in other articular diseases. The presence of two or more features of onychopathy is in favour of a psoriatic origin for arthritis. Nail pitting is the most common psoriatic nail lesion while onycholysis, nail bed discoloration, subungual hyperkeratosis, transverse grooves or longitudinal ridging may be observed. Nail dystrophy is the only clinical feature that can identify patients with psoriasis who will later develop arthritis. Nail lesions occur in around 90% of patients with PsA but only in 46% of psoriasis patients without arthritis[4].

Distal form of PsA

The distal pattern of PsA affects the DIP joints of the hands and can be very similar clinically and radiologically to erosive osteoarthritis of hands. DIP joint involvement in PsA is almost always accompanied by psoriatic nail changes of the corresponding finger. MRI scan revealed inflammation in the collateral ligaments, extensor tendons and entheseal insertions at the corresponding DIP joint with extracapsular contrast enhancement, bone oedema without cartilage damage and diffuse involvement of the nailbed in PsA[5]. The presence of psoriatic nail involvement is a clue to correct diagnosis of PsA clinically. X-rays may also help to differentiate between PsA and erosive osteoarthritis. The undulating osseous surfaces are usually closely applied in osteoarthritis while the lack of apposition of adjacent bony margins is a characteristic of PsA.

Oligoarticular form of PsA

The arthritis is inflammatory in nature and may show purplish red discoloration of the overlying skin, pain and significant early morning stiffness that improves with activity. Tenderness and joint effusion are usually less severe than in RA. The arthritis is typically asymmetric in distribution. Patients who initially presented with an oligoarthritis may later become polyarticular in disease involvement.

Polyarticular form of PsA

The polyarticular form of PsA may simulate RA. Table 2 shows the clinical features that help to differentiate between these two conditions. PsA tends to be asymmetrical and peripheral joints are more commonly involved in the 'ray' pattern i.e. all the joints of a single digit are affected, in contrast to the 'raw' pattern in RA where the same joints on both sides are involved. This may also explain the tendency to asymmetry that occurs in about half the cases of polyarticular disease in PsA. The presence of erythema on the inflamed joint is unusual in RA but may be seen frequently in PsA probably reflecting exaggerated angiogenesis characteristics of psoriatic disease[6]. Positive serology for rheumatoid factor and anti-cyclic citrullinated peptide (CCP) antibodies, though more prevalent in

RA, may be found in 10-15% of PsA patients[7]. Radiologically both PsA and RA are characterised by osseous erosions, however, irregular excrescence of bony proliferation and lack of juxta-articular osteoporosis strongly favour diagnosis of PsA.

Psoriasis and RA may coexist with a prevalence of 3:10000, the presence of nail dystrophy helps to distinguish patients with PsA from those with RA and psoriasis. In 7-30% of patients, arthritis may precede the appearance of psoriatic skin lesions and is referred to as "PsA sine psoriasis"[8]. In these patients, correct diagnosis of PsA will depend solely on the recognition of specific features of the articular disease.

Table 2. Comparison of clinical features between PsA and RA.

	PsA	RA
Demographics		
Peak age at presentation (years)	30-40	40-60
Female preponderance	Uncommon	Common
Clinical characteristics		
Distal interphalangeal joint involvement	Common	Uncommon
Symmetry	Less common	Common
Erythema over affected joints	Common	Uncommon
Spondylitis	Common	Uncommon
Enthesopathy	Common	Uncommon
Skin lesions	Common	Uncommon
Nail lesions	Common	Uncommon
Extra-articular manifestations		
Rheumatoid nodule	Not present	Present
Aortic valve disease/aortitis	Present	Not present
Eye involvement	Iritis, uveitis	Scleritis, episcleritis
Serological features		
Rheumatoid factor	Uncommon	Common
Anti-cyclic citrullinated peptide antibody	Uncommon	Common
Elevation in level of erythrocyte sedimentation rate / c-reactive protein	Lesser extent	Greater extent
Radiological features		
Osteopenia	Uncommon	Common
Osteolysis	Common	Uncommon
Ankylosis	Common	Uncommon

Spondyloarthropathy of PsA

PsA is classified with the SpA because of the presence of spondylitis in up to 40% of patients, other features of SpA (inflammatory enthesopathy, dacylitis, periosteal proliferation), the occurrence of extra-articular features common to the SpA (mucous membrane lesions, iritis, urethritis, diarrhoea, aortic root dilatation) and association with HLA-B27. The frequency of spinal involvement in PsA has been reported to be 2% as an isolated back disease to as high as 40% when associated with peripheral arthritis.

There is gender difference in disease expression among PsA patients with more advanced SpA among men. PsA patients who have spinal involvement are usually older than those without back involvement.Patients with SpA may present with inflammatory back pain and stiffness with restricted range of movement of the back. Clinical signs suggestive of sacroiliitis can be elicited from physical examination. PsA patients with SpA are mostly asymptomatic, are detected radiographically and may demonstrate full range of back movement. Compared to patients with ankylosing spondylitis, there is usually less severe neck pain and back pain, less limitation of movement and grade 4 sacroiliitis in PsA patients. PsA is characterised by the relative asymmetry of sacroiliac/spinal involvement and more extensive paramarginal syndesmophytes and/or periosteal reaction compared to ankylosing spondylitis or SpA related to inflammatory bowel disease. Cervical spine disease is found in 70% of PsA patients. Atlantoaxial subluxation is reported in 23% of patients and is related to prolonged disease duration.

Arthritis Mutilans

Arthritis mutilans is a destructive form of PsA and may lead to extensive and irreversible joint damage with appearance of the "telescoping" phenomenon and shortening of the digits within a short time (Figure 1). Severe joint or bone lysis leads to deformities with shortening and telescoping of digits.

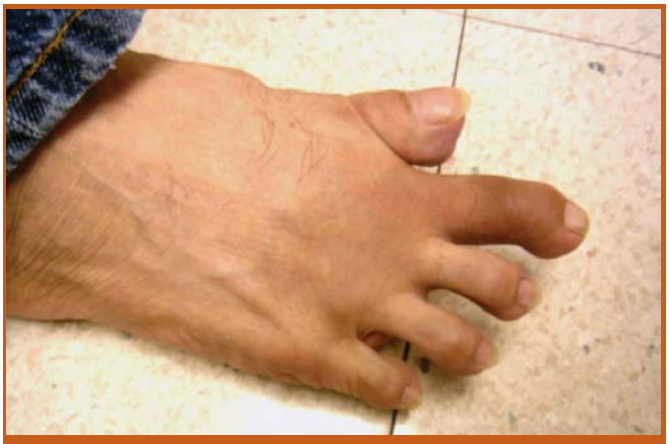


Figure 1. Telescoping of big toe and dactylitis of the second toe in a patient with PsA.

Enthesitis

The clinical presentation and radiological features of the oligoarticular and spinal patterns of PsA are similar and are often difficult to distinguish from other members of the SpA group such as reactive arthritis and inflammatory bowel disease related SpA. Enthesopathy, inflammation at the sites of tendon insertion, is particularly prominent in PsA affecting more frequently the plantar fascia or Achille's tendons. Enthesitis may be recognised radiographically as spurs and may also be identified using ultrasound scan. MRI scan reveals bone marrow oedema adjacent to the entheseal insertion sites representing a synovioentheseal inflammatory complex at the very early stage of PsA[9] corroborating the enthesis-based biomechanical hypothesis of disease pathogenesis.

Dactylitis

Dactylitis or 'sausage digits' is a typical feature of PsA (Figure 1) which presents as swelling of a whole digit with inflammation involving the distal and proximal interphalangeal and occasionally the metacarpophalangeal joints may be seen in 16-48% of patients. Dactylitis is usually less common in other SpA. MRI scan reveals features of synovitis with extensive inflammation and effusion in all the joints of a particular digit with an associated tenosynovitis in the whole digit and has been suggested to be a marker of disease progression in PsA[10].

Extra-dermal Extra-articular Features

Extra-articular manifestations of PsA are different from those of RA. Rheumatoid nodule is not found in PsA. Rheumatoid factor is present in around 10-15% of PsA patients compared to 70-80% of RA patients. Other extra-dermal, extra-articular features of PsA include iritis (7%), mouth ulcer, urethritis, colitis and aortic valve disease giving rise to morbidities in these patients.

Diagnosis of PsA

There are no specific laboratory tests which are diagnostic for PsA. Blood investigations may show anaemia of chronic illness or iron deficiency anaemia secondary to therapy with non-steroidal anti-inflammatory agents. Acute phase reactants in PsA are frequently normal or minimally elevated contributing little to diagnosis and may also be present in psoriasis patients without arthritis. Hyperuricaemia is not uncommon and is related to turnover of skin cells. Rheumatoid factor is usually negative but 10-15% of patients may have positive rheumatoid factor in low titre which is also present in similar proportion of patients without arthritis. Typical radiological features of PsA include pencil-in-cup appearance, irregular periosteal bone proliferation, resorption of the distal tuft and ankylosis and are diagnostic of the condition[11].

The diagnosis of PsA can be established in patients with an inflammatory arthritis who present with pre-existing skin psoriasis. Sometimes the skin lesions may be minimal and efforts should be made to look for skin lesions in the umbilical area, anal cleft, scalp and the ears. For patients who do not have skin lesions, the clinical radiographic features such as fluffy periostitis, lysis of terminal phalanges, pencil in cup appearance, gross destruction of isolated joint, ankylosis, and spondylitis may assist in diagnosis. The absence of juxta-articular osteoporosis, lack of symmetry and predilection for distal interphalangeal joints make RA less likely. The presence of dactylitis and enthesitis are more suggestive of PsA. Iritis and mucous membrane lesions may be more common in Reiter's disease.

The CASPAR Criteria

Historically, the Moll and Wright criteria have been used for the classification of PsA[12]. An international group of experts on PsA, the **C**l**AS**sification of **P**soriatic **AR**thritis (CASPAR) study group, has developed a new classification scheme (Table 3) based on extensive analysis on 588 patients with PsA and 534 other inflammatory arthritis has shown a specificity of almost 99% and sensitivity of 91.4%[13]. The CASPAR criteria are progressive by permitting the diagnosis of PsA sine psoriasis as well as in rheumatoid factor positive patients.

Table 3. CASPAR classification of psoriatic arthritis[13]

Established inflammatory musculoskeletal disease (joint, spine or entheseal) With 3 or more of the following:
1. Evidence of psoriasis
a. Current psoriasis
b. Personal history of psoriasis
c. Family history of psoriasis
2. Nail changes dystrophy (onycholysis, pitting, hyperkeratosis)
3. Negative rheumatoid factor
4. Dactylitis
a. Current
b. History
5. Radiologic evidence of juxta-articular new bone formation excluding osteophyte formation on plain x-rays of hand or foot

Clinical Course of PsA

It has always been believed that PsA is less aggressive compared to RA. However, destructive deforming arthritis has been reported in about 20% of PsA patients[8]. Radiological erosion was observed in 47% of patients within 2 years of disease onset and 55% patients have five or more deformed joints upon follow up for over 10 years[3]. 67% of established PsA patients had erosive disease in the clinic setting[14]. HLA antigens including HLA-B27 in the presence of HLA-DR7, HLA-B39 and HLA-DQw3 in the absence of HLA-DR7 were predictive of subsequent damage[15]. Clinical predictors for disease progression were found to include ≥ 5 swollen joints, high medication level particularly use of steroids at presentation to the clinic and female gender[16]. Other than its impact on functional status, patients with PsA also had impaired level of quality of life similar to patients with RA[17]. PsA patients were also found to be more predisposed to have accelerated atherosclerotic vascular disease[18]. The impact of PsA is also reflected by the increased mortality with a standardised mortality ratio of 1.62 with frequent cardiovascular deaths[19].

References

1. Shbeeb M, Uramoto KM, Gibson LE, O'Fallon WM, Gabriel SE. The epidemiology of psoriatic arthritis in Olmsted County, Minnesota, USA, 1982-1991. J Rheumatol 2000;27(5):1247-50.
2. Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK. Psoriatic arthritis (PSA)--an analysis of 220 patients. Q J Med 1987;62(238):127-41.
3. Kane D, Stafford L, Bresnihan B, FitzGerald O. A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. Rheumatology (Oxford) 2003;42(12):1460-8.
4. Gladman DD, Anhorn KA, Schachter RK, Mervart H. HLA antigens in psoriatic arthritis. J Rheumatol 1986;13(3):586-92.
5. Tan AL, Grainger AJ, Tanner SF, Emery P, McGonagle D. A high-resolution magnetic resonance imaging study of distal interphalangeal joint arthropathy in psoriatic arthritis and osteoarthritis: are they the same? Arthritis Rheum 2006;54(4):1328-33.
6. Gladman DD. Clinical aspects of the spondyloarthropathies. Am J Med Sci 1998;316(4):234-8.
7. Alenius GM, Berglin E, Rantapaa Dahlqvist S. Antibodies against cyclic citrullinated peptide (CCP) in psoriatic patients with or without joint inflammation. Ann Rheum Dis 2006;65(3):398-400.
8. Gladman DD, Antoni C, Mease P, Clegg DO, Nash P. Psoriatic arthritis: epidemiology, clinical features, course, and outcome. Ann Rheum Dis 2005;64 Suppl 2:ii14-7.
9. McGonagle D, Lories RJ, Tan AL, Benjamin M. The concept of a "synovio-entheseal complex" and its implications for understanding joint inflammation and damage in psoriatic arthritis and beyond. Arthritis Rheum 2007;56(8):2482-91.
10. Brockbank JE, Stein M, Schentag CT, Gladman DD. Dactylitis in psoriatic arthritis: a marker for disease severity? Ann Rheum Dis 2005;64(2):188-90.
11. Resnick D NA. Psoriatic arthritis. Diagnosis of Bone and Joint Disorders 1988;2nd edn. Philadelphia: WB Saunders:1171-99.
12. Moll JM, Wright V. Psoriatic arthritis. Semin Arthritis Rheum 1973;3(1):55-78.
13. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum 2006;54(8):2665-73.
14. Torre Alonso JC, Rodriguez Perez A, Arribas Castrillo JM, Ballina Garcia J, Riestra Noriega JL, Lopez Larrea C. Psoriatic arthritis (PA): a clinical, immunological and radiological study of 180 patients. Br J Rheumatol 1991;30(4):245-50.
15. Gladman DD, Farewell VT, Kopciuk KA, Cook RJ. HLA markers and progression in psoriatic arthritis. J Rheumatol 1998;25(4):730-3.
16. Gladman DD, Farewell VT, Nadeau C. Clinical indicators of progression in psoriatic arthritis: multivariate relative risk model. J Rheumatol 1995;22(4):675-9.
17. Husted JA, Gladman DD, Farewell VT, Cook RJ. Health-related quality of life of patients with psoriatic arthritis: a comparison with patients with rheumatoid arthritis. Arthritis Rheum 2001;45(2):151-8.
18. Eder L, Zisman D, Barzilai M, Laor A, Rahat M, Rozenbaum M, et al. Subclinical atherosclerosis in psoriatic arthritis: a case-control study. J Rheumatol 2008;35(5):877-82.
19. Wong K, Gladman DD, Husted J, Long JA, Farewell VT. Mortality studies in psoriatic arthritis: results from a single outpatient clinic. I. Causes and risk of death. Arthritis Rheum 1997;40(10):1868-72.

MCHK CME Programme Self-assessment Questions

Please read the article entitled "The Clinical Spectrum and Diagnosis of Psoriatic Arthropathy" by Dr. Mo-yin MOK 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 31 May 2010. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please choose the best answer.

1. Which condition is more prevalent in the general population?
a. Skin psoriasis
b. Rheumatoid arthritis
c. Spondyloarthropathy form of psoriatic arthropathy
d. Distal pattern of psoriatic arthropathy

2. Which of the following best classify psoriatic arthropathy ?
a. Connective tissue disease
b. Rheumatic manifestations of systemic diseases
c. Spondyloarthropathy
d. Reactive arthritis

3. The following are typical clinical patterns of psoriatic arthropathy except
a. Symmetrical polyarthritis
b. Asymmetrical oligoarthritis
c. Spondylitis
d. Atlantoaxial subluxation

4. The following features are present in psoriatic arthropathy sine psoriasis except
a. Skin psoriasis
b. Nail dystrophy
c. Dactylitis
d. Enthesitis

5. Which of the following extra-articular features is found in psoriatic arthropathy?
a. Rheumatoid nodule
b. Interstitial lung disease
c. Aortic valve disease
d. Scleromalacia perforans

6. The following features favour psoriatic arthropathy over rheumatoid arthritis except
a. Dactylitis
b. Enthesitis
c. Periungal erythema
d. Nail dystrophy

7. Compared to ankylosing spondylitis, the spondyloarthropathy of psoriatic arthritis is
a. More extensive in involvement
b. Less severe in symptoms
c. More symmetrical in involvement
d. More frequently associated with HLA-B27

8. Which of the following statement in regard to enthesitis is incorrect?
a. is inflammation of tendon insertion
b. may be detected by ultrasound scan
c. may show up as spur on plain X-ray
d. represents tenosynovitis and arthritis of the involved digit

9. The following are typical radiological features of psoriatic arthropathy except
a. Juxta-articular osteoporosis
b. Pencil-in-cup appearance
c. Lysis of terminal tuft
d. Unilateral sacroiliitis

10. The following are predictive factors for progression of psoriatic arthropathy except
a. Dactylitis
b. High level of medication at initial presentation to clinic
c. Male gender
d. Extensive joint involvement at disease onset

ANSWER SHEET FOR MAY 2010

Please return the completed answer sheet to the Federation Secretariat on or before 31 May 2010 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

The Clinical Spectrum and Diagnosis of Psoriatic Arthropathy

Dr. Mo-yin MOK

MBBS(HK), MRCP(UK), FHKCP, FHKAM, FRCP(Glas), FRCPA(Australia)
Assistant Professor, Division of Rheumatology, Department of Medicine, Queen Mary Hospital

1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

Name (block letters):_____ HKMA No.: _____

HKID No.:___ ___ - ___ ___ ___ ___ X X (x) HKDU No.: _____

Contact Tel No.:_____ DCHK No.: _____

Answers to April 2010 Issue

Facial Plastic Surgery in Otorhinolaryngology

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

Psoriasis

Dr. King-man HO

MBBS (HK), MRCP (UK), FHKCP, FHKAM (Medicine), FRCP (Glasgow, Edin), Dip Derm (London), Dip GUM (LAS)

Consultant Dermatologist, Social Hygiene Service, CHP
Specialist in Dermatology and Venereology

Dr. King-man HO

Abbreviation used:

HLA	: human leucocyte antigen
Th1	: type I helper T cell
IL	: interleukin
TNF	: tumour necrosis factor
PASI	: psoriasis area and severity index
BSA	: body surface area
DLQI	: dermatology life quality index
UV	: ultraviolet light
PUVA	: ultraviolet A with psoralen therapy

Introduction

Definition

Psoriasis is one of the prototypic papulosquamous skin diseases characterised by erythematous papules or plaques with silvery scales. It is a chronic inflammatory skin disease with increased epidermal proliferation related to dysregulation of the immune system.

Epidemiology

Psoriasis is said to affect 2% of the world population. The prevalence is up to 5% in selected Western population. The prevalence in Chinese is estimated to be 0.3% to slightly more than 1%, the variation in estimation is accounted for by the methodological differences in these surveys.

Psoriasis has a bimodal age of disease onset. The first peak is around 20 and the second peak is around 60. People with disease onset around 20 year old have stronger genetic predisposition. They have a higher prevalence of having HLA-Cw6. The linkage to genetic factor is lower for the group with late onset disease.

Pathology and Pathogenesis

Pathology

The pathology of an established lesion of psoriasis is characterised by epidermal hyperplasia with squared-off rete ridges, parakeratosis, elongation of dermal papillae with thinning of the supra-papillary epidermis, dilated tortuous capillaries and perivascular lymphohistiocytic infiltration of the superficial dermis. In some of these lesions, collection of polymorphonuclear cells in the stratum corneum (known as Munro microabscess) and between keratinocytes (known as spongiform abscess of Kogoj) may be found.

Pathogenesis

The cell cycles of the epidermal cells in psoriatic lesions are greatly accelerated. Turnover of the basal cells, which divide every 1.5 days, in psoriatic skin is as fast as the rapidly growing cells of the small intestinal mucosa. The epidermal transit time is shortened from the normal 28 days to as short as 3-4 days. Such a change in epidermal cell cycle is thought to be related to the inflammatory cytokine released in the inflammatory process. Psoriasis is considered to be a prototypic Th1 disease. Increased amounts of Th1 cytokines viz interferon γ and IL-2 are observed. Early interventional researches adopted drugs such as cyclosporine A with pharmacological effects on this pathway, rendered proof of concept evidence to this observation. Studies have also demonstrated that keratinocytes and dendritic cells play an important role in innate and adaptive immune response of the skin. The innate immune response cytokines IL-1, 6 and TNFα are up-regulated in psoriatic skin. Biologic approaches to reduce the activity of TNFα are found to be useful in psoriasis. Recent studies show that Th17 cells, a novel subset of T cells, play a pivotal role in the pathogenesis of psoriasis. Recognition of the early dendritic cell-T cell interaction through IL-12 and IL-23 provides the new insight for the latest biological treatment, the monoclonal antibody against IL-12/IL-23, which results in a breakthrough in the management of psoriasis[1].

Clinical Features

Prototypic lesion

The typical lesion of psoriasis is a well-demarcated erythematous plaque with silvery scales on top of the plaque (figure 1). The affected patient may experience itchiness. The plaques may affect anywhere of the skin surface but the mucosa is normally spared. The scales may only be loosely attached and easily fall off from the skin. The disease may wax and wane, and not uncommonly is aggravated by trauma and irritation, infections, various drugs, seasonal changes and psychogenic stress.

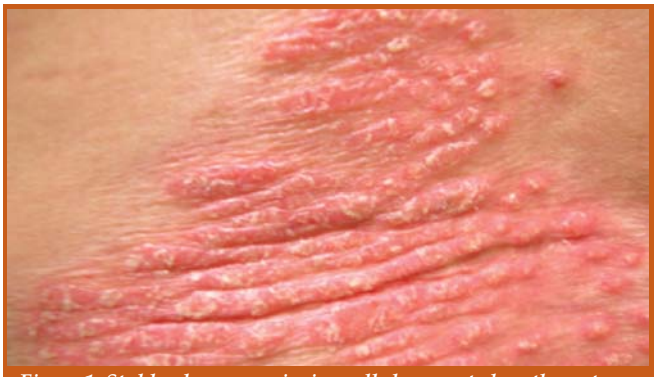


Figure 1. Stable plaque psoriasis: well-demarcated erythematous plaque with a silvery scale on top.

Chronic Stable Plaque Psoriasis

Sites of predilection of the characteristic plaques include the extensor surfaces of the elbows, knees, lower back and scalp. The genitalia and nails may also be affected. The plaques vary in size (figure 2). New lesions may be induced at traumatised skin such as surgical scar, or even scratch marks (known as Köbner phenomenon).

Figure 2. Diffuse involvement of the back in this patient. If more than 90% of the body surface is affected, it is referred as erythrodermic psoriasis.

Differential Diagnosis

Common conditions that may present as erythematous plaque include discoid eczema, lichen simplex chronicus, hypertrophic lichen planus and Bowen's disease.

Guttate Psoriasis

It is a variant characterised by small coins or even punctate lesions with less amount of scale affecting mostly young people. The disease may be precipitated by upper respiratory tract infection. Over half of these patients have some evidence of preceding streptococcal infection. A few may have prolonged disease remission after the acute episode.

Differential Diagnosis

Common conditions that may present as guttate papulosquamous lesions include pityriasis rosea, secondary syphilis and pityriasis lichenoides.

Unstable Psoriasis

Lesions are angry looking with more intense inflammation. These may be redder in colour with less scaling. Lesions may be less well-demarcated and occasionally exudation and crust are found[2]. Patients may experience more itchiness, irritation and even pain. Further progression to erythrodermic or pustular psoriasis can happen. Inappropriate use of corticosteroids, excessive irritation, sunburn are some of the factors not uncommonly associated with unstable psoriasis.

Differential Diagnosis

Common conditions that may present like unstable psoriasis include eczema and tinea incognito.

Erythrodermic Psoriasis

When more than 90% of the body is involved by psoriasis, it is defined as erythrodermic psoriasis. An affected patient is characterised by having generalised redness of skin and scaling. The colour may sometimes be described as dusky red. The face may occasionally be relatively spared. Individual plaques may not be obvious. Pustules may sometimes be found. Triggering factors are not uncommonly unidentified. Affected patients may have systemic symptoms.

Differential Diagnosis

Conditions that may present as erythroderma include eczema/dermatitis, cutaneous T cell lymphoma, drug reaction, pityriasis rubra pilaris and pemphigus foliaceus.

Pustular Psoriasis

Tiny superficial pustules with a background of erythema may occur. The roof of the pustules is easily broken. These pustules can be distributed throughout the whole skin surface or more localised especially in and around the unstable lesions. Some patients may have lesions with matted scales with a yellowish hue and if biopsy of these lesions is performed, the histology shows sheets of subcorneal polymorphs. Though very discrete pustules may not be seen clinically, these lesions may be described as pustular psoriasis by some clinicians. Steroid withdrawal* is the commonest precipitating factor encountered by the author as the cause of pustular psoriasis. Localised pustular psoriasis on the palms and soles is reported to be associated with smoking.

Differential Diagnosis

Conditions that may present as generalised pustulosis include infections, acute generalised exanthematous pustulosis and subcorneal pustular dermatosis.

*Not only when systemic steroids are given and tapered, but also when the patient has used super potent topical steroids for a long time, therefore, save for a few exceptional clinical situations, systemic steroids are contraindicated in psoriasis.

Psoriasis in Specific Body Locations

Scalp and face: The scalp is one of the most common sites affected by psoriasis. The typical plaques may extend slightly beyond the hairline (figure 3). Patients may present as recalcitrant dandruff. Some may involve the glabella region, eyebrows, and nasolabial fold, and in this situation it merges with seborrhoeic dermatitis. The term sebopsoriasis is coined to describe these cases. The external auditory canal is not uncommonly affected. Psoriatic lesions on the face may not be very well-demarcated nor is the silvery scale easily discernible.

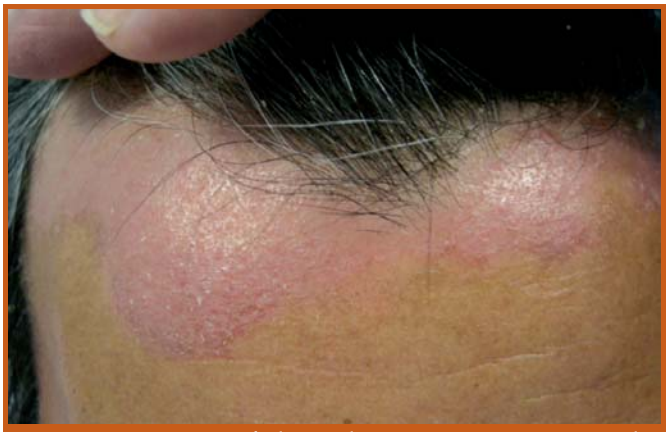


Figure 3. Psoriasis of the scalp. Lesion not uncommonly extends beyond the hairline. Psoriatic lesion on the face may not be very well-demarcated nor is the silvery scale easily discernible.

Differential Diagnosis
Conditions that may mimic psoriasis on the face or scalp include seborrhoeic dermatitis, lichen simplex chronicus (plaques on scalp) and tinea capitis.

Flexural regions: Lesions located at the axillae, inframammary folds, groins, intergluteal cleft and prepuce of the uncircumcised may present as shiny pink to red thin plaques or even patches. Fissure may sometimes be present. The scale is not discernible. Itchiness and irritation are not uncommon. The term inverse psoriasis is coined to describe these lesions.

Differential Diagnosis
Conditions that may mimic psoriasis in the intertriginous areas include seborrhoeic dermatitis, candida infection, intertrigo and extramammary Paget's disease.

Nail: Nail involvement has been reported in 10 - 80% of patients with psoriasis (figure 4). Features include onycholysis with or without the oil drop phenomenon, distal subungual hyperkeratosis, thimble pitting, crumbly poorly adherent nail, loss of lustre among other changes. The finger and toe nails can be affected. Patients with nail involvement may have a higher incidence of arthropathy. Psoriatic nails may also predispose to fungal infection. Nail disease can cause much concern to those affected because it can be symptomatic, have functional disability, cosmetically unacceptable but otherwise recalcitrant to all forms of treatment.

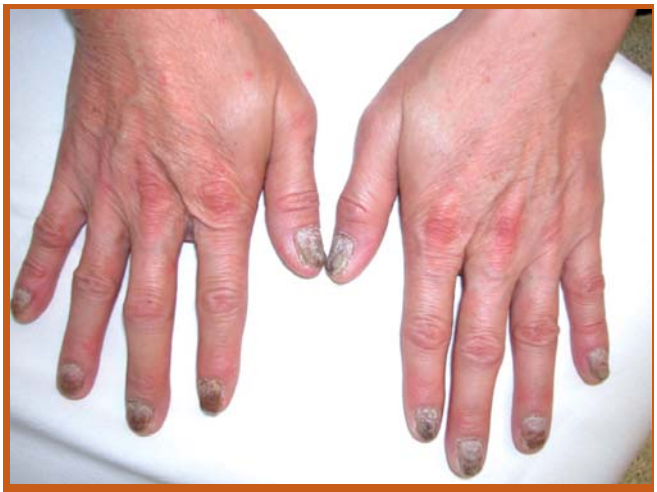


Figure 4. Nail psoriasis. Onychomycosis seldom presents as symmetrical involvement of the finger nails especially when all ten nails are involved. The unhealthy nails are more prone to secondary fungal infection and therefore should be excluded as appropriately.

Differential Diagnosis
Conditions that may mimic changes in nail psoriasis include fungal infection and various forms of idiopathic nail dystrophy.

Clues to differentiate these conditions are summarised in table 1. Psoriatic arthropathy is discussed in another article of this issue.

Table 1. Differential diagnosis and clinical clues to guide differentiation

Psoriasis presentation	Differential diagnosis	Clinical differentiation
Stable plaque	• Eczema (discoid and chronic lichenified) • Hypertrophic lichen planus • Bowen's disease	• Weeping in active discoid eczema; lichenification in lichen simplex, scales in lichen simplex are not as loosely attached and silvery as in psoriasis • Violaceous plaques mostly located on the shins, scales are not as loosely attached and silvery • Long standing solitary thin plaque without the characteristic silvery scale in psoriasis
Guttate	• Pityriasis rosea • Secondary syphilis • Pityriasis lichenoides chronicus (PLC)	• Oval papulosquamous plaques with long axis of lesions aligned in a fir tree distribution mostly on the trunk • Palm and sole involvement, mucosal lesions, systemic symptom, lymphadenopathy • More polymorphic in PLC, mica scale and Oblaten sign
Unstable	• Eczema • Tinea incognito	• Among the unstable psoriatic lesions, some may have otherwise typical morphology of psoriasis; history of using potent topical steroid or systemic steroid • Less plaque like and angry looking in tinea incognito
Erythrodermic	• Other causes of erythroderma such as eczema/dermatitis, cutaneous T cell lymphoma, drug reaction, pityriasis rubra pilaris (PRP), pemphigus foliaceus.	• History of psoriasis; nail changes of psoriasis; history of eczema or exposure to allergens; painful keratoderma, diffuse alopecia and leonine face in T cell lymphoma; starts as morbilliform rash in drug reaction; island of sparing, perifollicular erythema and orange hue in PRP; impetigo-like blisters and erosions followed by collarettes of scale or scale-like crusts in pemphigus foliaceus
Pustular	• Generalised: acute generalised exanthematous pustulosis • Subcorneal pustular dermatosis • Localised palmoplantar pompholyx; tinea pedis	• Sudden onset after taking drug; no past history of skin diseases; self limited • Predilection for axillae and around the groins • Vesicular lesions instead of pustular lesions
Scalp	• Seborrhoeic dermatitis • Tinea capitis	• Discernible plaque or patch in psoriasis • Bald patches in tinea with broken hair stump
Inverse	• Candidiasis • Tinea • Extramammary Paget's disease	• Satellite lesions with scalloped scale in candida intertrigo • Faintly discernible active margin in tinea • More wet looking and with erosion in extramammary Paget's disease
Nail	• Onychomycosis	• Chalky white subungual hyperkeratosis or superficial or proximal subungual whitish discolouration; the oil drop sign and thimble pitting in nail psoriasis

Disease Assessment

Clinician Based
The British guidelines define "severe" disease as PASI score of 10 or more (or a BSA of 10% or greater where PASI is not applicable) and a DLQI > 10, i.e. the rule of ten proposed by Finlay[3]. PASI and BSA are regarded as clinician-based assessment of severity. In recent years, patient reported outcome such as health related quality of life assessment by various tools is required by the

regulatory agencies for drug registration. DLQI and SF-36 are two of such instruments commonly used in clinical trials in psoriasis. PASI 75, which refers to a reduction of PASI score by 75% of the baseline, is employed as an endpoint assessment in modern clinical trials involving biologics. PASI is calculated as follows:

$\sum$[area score of the region X extent indicator of the region X (sum of severity indicator of the region)]
= 0.1 $Å_{Head}$ (E + S+ T) + 0.2 $Å_{Upper\ limbs}$ (E + S+ T) + 0.3 $Å_{Trunk}$ (E + S+ T) + 0.4 $Å_{Lower\ limbs}$ (E + S+ T) wherein Å refers to area of the suffix region according to a 7-point scale (0-6), E, S, T refers to the degree of erythema, scaling and thickness respectively according to a 5-point scale (0-4). The maximum score is 72.

Severity assessment of joint disease is discussed in the other chapter of the same issue.

Other Assessment

The occupation history is also important as this will give an idea of the social and functional disability associated with the skin condition of the index patient. Treatment and drug history will provide further information guiding the choice of various therapeutic modalities for a particular patient.

As a routine, the author also asks the patient how he/she feels about his/her skin disease and what his/her major concern is before the discussion of the various treatment options.

Laboratory investigations are not required in an otherwise simple and mild case of psoriasis. Skin biopsy is not usually required to establish the diagnosis of psoriasis unless in doubt. Skin scraping, hair plugging or nail clipping for fungal study may be performed as appropriate. If systemic therapy or phototherapy is contemplated, baseline investigations include complete blood picture, liver and renal biochemistry, hepatitis serology, anti-nuclear antigen, fasting sugar and lipid, and chest X-ray should be performed to guide choosing a preferred modality from the various possible options.

Table 2. Prevalence of diseases associated with metabolic syndrome (WHO case definition) in patients with psoriasis (Cohen et al. 2008)

Category	Prevalence		
	Psoriasis n = 16 850	Control n = 48 677	OR (95% CI)
Mean age, years (SD)	42.7 (20.3)	51.0 (19.1)	
Ischaemic heart disease % (n)	14.2 (2 387)	7.1 (3 479)	2.1 (2.0-2.3)
Diabetes mellitus % (n)	13.8 (2 324)	7.3 (3 556)	2.0 (1.9-2.1)
Hypertension % (n)	27.5 (4 627)	14.4 (7 017)	2.2 (2.2-2.3)
Obesity % (n)	8.4 (1 419)	3.6 (1 768)	2.4 (2.3-2.6)

SD = standard deviation; CI = confidence interval; OR = odds ratio

Recent epidemiological studies also reveal that people with psoriasis are associated with risk factors for cardiovascular diseases, table 2[4]. Although standard guidelines for screening of cardiovascular risk factors have not arrived, to take a good history covering these risk factors such as smoking habit, personal and family history of diabetes, hypertension, hyperlipidaemia and cardiovascular diseases may be contributory to the holistic management of a person with psoriasis. The affiliated service of the author also recommends routine measurement of the body mass index and blood pressure in patients newly presented with psoriasis.

Management

The Principle of Management

To understand the expectation of the patient is the most important first step in management. To communicate and educate the index case is the next important step. Appropriate skin care, avoidance of aggravating factors, the importance of keeping a good treatment history, cessation of smoking, avoidance of excessive alcohol drinking, reinforcement of the non-contagious nature and chronicity of the condition and conveying the message that psoriasis is amenable to very good control are the important contents in communication, especially in the first few encounters. Instruction on using especially the perplexing large number of topical drugs should be simple and clear. Printed information is invaluable.

Treatment Options

Treatment for psoriasis can be classified as:

1. **Topical drugs:** topical steroids, vitamin D analogues, tar, dithranol (which may have been withdrawn from the local market), keratolytics, calcineurin inhibitors, tazarotene (vitamin A analogue which is not available in the local market)
2. **UV light therapy:** UVB including nBUVB, PUVA, targeted phototherapy such as UVB delivered with the laser system (Excimer® 308 nm)
3. **Traditional systemic therapy:** methotrexate, systemic retinoid, cyclosporine A, (others include hydroxyurea, 6-thioguanine, mycophenolate mofetil, fumaric acid esters, these are less extensively used and their use is regarded off-label by the manufacturers, fumaric acid esters are not available in the local market)
4. **Biologic therapy:** etanercept, infliximab, adalimumab which target TNFα; ustekinumab which targets IL-12 & 23

Management Hierarchy

A combination of topical therapies is usually used as the first line treatment in patients with limited area involvement i.e. <10-20% BSA. If the condition is not under satisfactory control by topical therapy or the disease is too extensive, phototherapy or systemic therapies may be considered. Poor adherence to topical therapy is not uncommon. Therefore, before considering systemic treatment, skillful counselling is almost a must not only to confirm poor adherence, but also to know the expectation and concern of the patient. In a patient who has severe disease (c.f. the aforementioned rule of ten) which fails phototherapy and systemic therapy, or who is intolerant to these therapies, or who has significant, coexisting comorbidity (e.g. significant impairment of renal or liver function and unable to attend 2x/week for phototherapy in a designated dermatology clinic) which precludes the use of these treatments, biologic therapy can be considered[5].

As the adverse effects of these treatments differ or overlap one another, and some of these treatments have

recommended ceiling cumulative exposure such as not more than 150 to 200 treatment sessions in PUVA, treatment rotation is not uncommonly adopted. For recalcitrant cases, a combination of systemic including biologic therapies may be used. A failure of treatment with one biologic does not necessarily mean knocking out the whole group of biologics. The strength and weakness is summarised in table 3.

Conclusion

Psoriasis is a chronic inflammatory skin disease which causes significant functional impairment to those affected. A constellation of treatments is now available which can achieve disease control in most people with psoriasis. The best available evidence in management should, however, be applied on an individual basis.

References

1. PCM Kerkhof, J Schalkwijk. Psoriasis in Dermatology 2nd ed by JL Bolognia, JL Jorizzo, RP Rapini. Mosby Elsevier 2008
2. Psoriasis in Dermatology 2nd ed by O Braun-Falco, G Plewig, HH Wolff and WHC Burgdorf Springer NY 1996
3. Finlay AY. Current severe psoriasis and the rule of tens. Br J Dermatol 2005;152:861-7
4. Cohen AD, Sherf M, Vidavsky L, Vardy DA, Shapiro J, Meyerovitch J. Association between psoriasis and the metabolic syndrome. A cross-sectional study. Dermatology. 2008;216(2):152-5.
5. Smith CH, Anstey AV, Barker JNWN, Burden AD, Chalmers RJG, Chandler DA, et al. British Association of Dermatologists' guidelines for biologic interventions for psoriasis 2009. Br J Dermatol 2009;161:987-1019

Table 3. Highlights and comparison of treatments for psoriasis

Treatment	Short notes for Practical use	Strength	Weakness
Topical			
Steroids	• Still the mainstay of topical treatment • A wide variety of choice with respect to potency and formulations; the most potent is more than 600 times the least one • No more than twice daily use recommended • Co-formulated with other active ingredients such as calcipotriol, salicylic acid	• Effective & user friendly	• Tachyphylaxis dissociated with adverse effects • Systemic effects after prolonged use of potent topical steroids • Caution: use on the face and intertriginous regions; use of super potent topical steroid in unstable psoriasis
Vit D analogues	• Calcipotriol; calcitriol • Calcipotriol is co-formulated with betamethasone dipropionate • May be used during the steroid drug holiday	• The main non- steroidal topical preparation • No evidence for tachyphylaxis	• Not all patients like vit D analogues because of irritation and less quick in action compared to topical steroids • Caution: maximum weekly amount 100 gm for calcipotriol (210 gm for calcitriol)
Tar	• Commonly used for bathing, though gel formulation for direct application is available • Combined with phototherapy in the classic Goeckerman regime	• Non-steroidal topical preparation	• Not user friendly because of its smell and appearance • Caution: use in unstable psoriasis
Others	• Dithranol is not available in the local market • Calcineurin inhibitors can be used to treat facial and flexural psoriasis • Sulphur and salicylic acid are commonly used as de-scaling agents but seldom used alone		
Phototherapy			
	• These include UVB (broad or narrow band); PUVA; targeted phototherapy	• Treatments do not have substantial systemic adverse effects • PUVA is a very effective treatment • UVB therapy can be use in pregnancy	• Patient needs to present personally to the clinic for treatment 2-3x weekly • Patient needs to wear glasses that filter UV light for the whole day after PUVA • Less than 150-200 lifetime cumulative exposure is recommended for PUVA • Not suitable for unstable, erythrodermic, or pustular psoriasis
Systemic			
Methotrexate	• Weekly dosing with dose ranges from 5 mg to 25 mg • Commonly dosed together with folate supplement	• Convenient, user friendly • Effective, inexpensive traditional systemic treatment • Long experience • Can be used in a variety of psoriatic state	• Idiosyncratic reaction leading to pancytopaenia, paradoxic skin necrosis, hence,slow incremental dosing is commonly adopted • Teratogenicity and effect on male fertility • In real life situation it may be difficult to use as so many people may have deranged liver function such as fatty liver
Systemic retinoid	• Acitretin is the retinoid of choice in psoriasis • Dose seldom exceeds 1 mg/kg/day	• Long experience • Very effective when combined with PUVA • Severe acute adverse event uncommon	• Seldom achieves very satisfactory disease control • Fair patient acceptability because of constellation of minor adverse effects • One of the most potent teratogen known; in female, written consent is required by the manufacturer
Cyclosporine A	• 2.5 to 5.0 mg/kg/day;	• Not usually hepatotoxic • May be effective in arthritis	• Extensive drug interaction with a variety of drugs • Seldom used for more than 2 years because of its nephrotoxicity • Relapse is common upon treatment cessation
Biologics	• Etanercept, infliximab and adalimumab target TNFα • Ustekinumab targets IL 12 & 23 • Available in the local market • Apart from infliximab which is delivered by intravenous route, the others can be given subcutaneously	• Neither toxic to the liver nor to the kidney in contrast to the conventional systemic drugs • Dosing can be spaced out e.g. quarterly dose of ustekinumab is required after the initial induction • Effective in arthropathy	• Apart from adverse effects such as demyelinating disease, proneness to infections of which TB is of most concern • Induction of ANA positivity • Long term adverse effects are unknown

NB. Readers are encouraged to refer to the insert and standard textbook for details of the adverse effects of these treatments.

10 Years Experience in Balancing

POWER & SAFETY

- Proven efficacy in various pain models[1,2,3,4]
- Lower incidence of GI side effects versus NSAIDs*[5]
- The only coxib approved on all continents[6-11]
- Over 1 million prescriptions filled for CELEBREX each month[12]

CELEBREX 100 mg & 200 mg capsules are also available

*NSAIDs = nonsteroidal anti-inflammatory drugs

References: 1. Graham DY, Chan FK. Gastroenterology 2008;134:1240-1257. **2.** Nadarajah A, et al. Singapore Med J 2006; 47:534. **3.** Petri M, et al. J Rheumatol 2004;31:1614-20. **4.** Cheung R, et al. Clin Ther 2007;29 [Theme Issue]:2498-2510. **5.** Silverstein FE, et al. JAMA 2000;284:1247-1255. **6.** European Medicines Agency (EMEA. http://www.emea.europa.eu/. **7.** Food and Drugs Administration (FDA). http://www.fda.gov. **8.** Announcement of the launch of the selective COX-2 inhibitor Celecox(r) tablets in Japan. June 11, 2007. Available at: http://www.astellas.com/global/about/news/2007/pdf/070611_eg.pdf. Accessed September 12, 2008. **9.** Australian Therapeutic Goods Administration. http://www.tga.gov.au/ **10.** COFEPRIS. http://www.cofepris.gob.mx. Accessed September 4, 2008. **11.** Package insert of Celebrex as registered in South Africa. Available at: http://www.arthritis.co.za/celebrex_package.html. Accessed September 4, 2008. **12.** About Celebrex. 2008. Available at URL: http://www.celebrex.com/content/about_CELEBREX.jsp?setShowOn=../content/about_CELEBREX.jsp. Accessed June 26, 2008. **Full prescribing information is available upon request.**

Pfizer Corporation Hong Kong Limited
16/F., Stanhope House, 738 King's Road, North Point, Hong Kong
Tel: (852) 2811 9711 Fax: (852) 2579 0599
Website: www.pfizer.com.hk

TOTAL PAIN MANAGEMENT

CEL09035P

LEXUS

No.1 Luxury SUV!

HYBRID

10 Best Cars of the Year 2009
Car & Driver

The Best Japanese SUV 2009
Car Plus

SUV of the Year 2009
Top Gear

RX series continued to be the best-selling Luxury SUV in Q1 2010. Now the RX450h 2010 Upgrade Edition with even more advanced features is available.

Come and check out the special offers now!

RX450h
2010 Upgrade

New features of the RX450h 2010 Upgrade

• Advanced rear parking camera • Ample back trunk storage space

More upgraded features for the Sport version : • New power back door • LED headlamps

* No.1 Luxury SUV — The Lexus RX series is the best-selling SUV model in its class based on 2009 and Q1 2010 sales result.

LEXUS HYBRID DRIVE

You've Got the Power!

Book your test drive on www.lexus.com.hk or visit our Lexus premium outlets today...

Hong Kong: Shop A, G/F Harcourt House, 39 Gloucester Road, Wanchai. Tel: 2511-9430 (P Free Valet Parking)

New Territories: G/F Mita Centre, 552-566 Castle Peak Road, Kwai Chung. Tel: 2880-4151 (P Free Valet Parking)

www.lexus.com.hk Inchcape

Manifestation of Radiological Abnormalities in Psoriatic Arthritis

Dr. Y. WONG

Department of Radiology, Baptist Hospital, Hong Kong

Introduction

Psoriatic arthritis is a distinctive kind of arthritis in patients with psoriasis. This disorder has a wide radiological spectrum and should be recognised at early stage of the disease. The presence of articular abnormalities in psoriatic patients varies from 2% to 6%. The reported prevalence of psoriasis among patients with polyarticular arthritis ranges from 3% to 5%.[1] Five varieties of psoriatic arthritis have been categorised by the work of Wright and Moll.[2,3] They include polyarthritis with distal interphalangeal joint involvement; symmetrical seronegative polyarthritis simulating rheumatoid arthritis; monoarthritis or asymmetrical oligoarthritis; sacroiliitis and spondylitis; and arthritis mutilans. In certain cases, the diagnosis of psoriatic arthritis cannot be made solely based on radiological findings. Some patients have disease patterns that differ from the five classical types. In one group of patients, dactylitis or enthesitis are the predominant abnormalities.[4]

Typical Sites of Radiological Abnormalities

Psoriatic arthritis involves the synovial and cartilaginous joints, as well as the attachment of tendons and ligaments to the bones in the appendicular and axial skeleton. It shows similar distribution of abnormalities as Reiter's syndrome and ankylosing spondylitis, and differs from rheumatoid arthritis.

The most typical sites of abnormalities are the interphalangeal joints of hands and feet, metacarpophalangeal and metatarsophalangeal joints, calcaneus, sacroiliac joints and spine. Less frequently changes may be found in the knees, ankles, sternocalvicular, and costovertebral joints. The disease rarely affects the hip and glenohumeral joints.

The distribution of psoriatic arthritis can vary, with certain distinctive characteristics. An asymmetrical or unilateral joint involvement is more common in psoriasis than rheumatoid arthritis. The upper and lower extremity joints are involved in psoriasis, in contrast to Reiter's syndrome with predominantly lower extremity involvement. The abnormalities in phalangeal tufts and calcaneus in psoriatic arthritis are characteristic.

In the axial skeleton, the abnormalities predominantly affect the spine and sacroiliac joints. The symphysis pubis and tendinous insertions at the pelvis may also demonstrate abnormalities.

Radiological Abnormalities at Peripheral Joints

The radiological abnormalities of psoriatic arthritis include erosive arthropathy at the distal interphalangeal joints of the hands.[5] The erosion starts at the periphery of the articulation and extends into the centre. (Fig. 1) Asymmetrical involvement of the interphalangeal joints of the hands and feet are common. Irregular periosteal bony proliferation resulting in periostitis is a typical presentation. (Fig.2) There is also presence of ankylosis of the joints, with lack of osteoporosis. The resorption of the tufts of the distal phalanx in the hands and feet is characteristic for psoriatic arthritis.[6] The progressive osteolysis may progress to destruction of most of the phalanx. The eroded small bones are irregular in outline. The expansion of the base of the distal phalanx combined with the middle phalanx gives "pencil and cup" appearance.

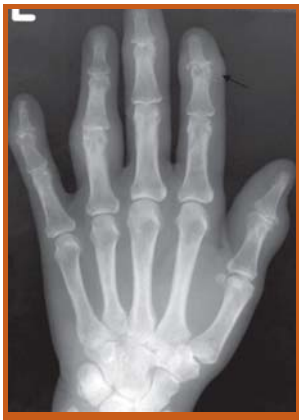


Fig.1 Distal interphalangeal joint in psoriatic patient with narrowing of joint space, marginal and central erosions (arrow). Mild flexion deformity is noted.

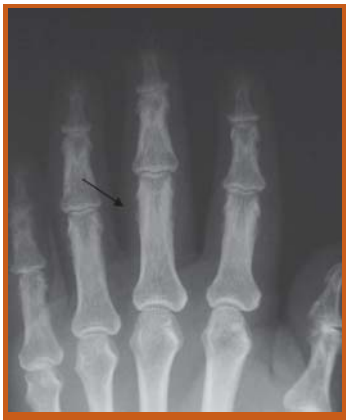


Fig.2 Radiograph shows soft tissue swelling, with bony erosion and proliferation compatible with periostitis (arrow) in psoriatic arthropathy.

High-resolution ultrasound and ultrasound together with power Doppler are sensitive in diagnosing synovitis in the case of established psoriatic arthritis.[7] Magnetic resonance imaging (MRI) is useful in assessing the synovitis in cases of psoriatic arthritis. The MR appearance of synovitis may be indistinguishable from that of rheumatoid arthritis even with dynamic imaging techniques.[8] However, the type and site of the lesions, as well as other typical abnormalities like enthesitis can help to differentiate psoriatic arthritis from other arthritis.[9] Other forms of MRI abnormalities include bone marrow oedema at the subchondral and diaphyseal regions; the latter one is relatively specific to psoriatic arthropathy.[9]

Enthesitis

One characteristic feature of psoriatic arthritis is inflammation of the entheses, at the attachment sites of tendons, ligaments, fascia and joint capsule to bones. The locations include the posterior and inferior surfaces of the calcaneus, femoral trochanters, ischial tuberosities, medial and lateral malleoli, ulnar olecranon, and the anterior surface of the patella.[10] Oriente et al have found peripheral enthesitis in 20% of their patients with psoriatic arthritis with a peak value of 30% in the spondylitic pattern.[11] In calcaneus, the bone erosion at the plantar aspect results in sclerosis of bone with irregular and poorly defined enthesophytes at the attachment of the plantar ligament and aponeurosis. The enthesophytes will be sharply delineated in outline, and occasionally becomes eburnated.

Recently, ultrasound has been used as a sensitive imaging modality to assess the enthesitis. Lehtinen et al[12] and Balint et al[13] first described sonographic features in limb enthesitis, showing a high frequency of asymptomatic abnormal findings. The abnormalities include loss of normal fibrillary echogenicity, with increased thickness or intralesional focal changes at tendon insertion. Power Doppler can show abnormal hyperaemia and vascularisation in enthesitis.[14] (Fig.3) MRI with its fat saturation sequence can demonstrate early enthesitis, which presents as diffuse bone marrow and soft tissue oedema.[15] The enthesitis is characterised by extracapsular inflammation at the insertions of ligaments and tendons with bone marrow oedema at attachments. Enhancement of ligament and bursa after intravenous injection of gadolinium contrast is also present. MRI is also a more sensitive imaging tool in assessing the treatment response in enthesitis.[16]

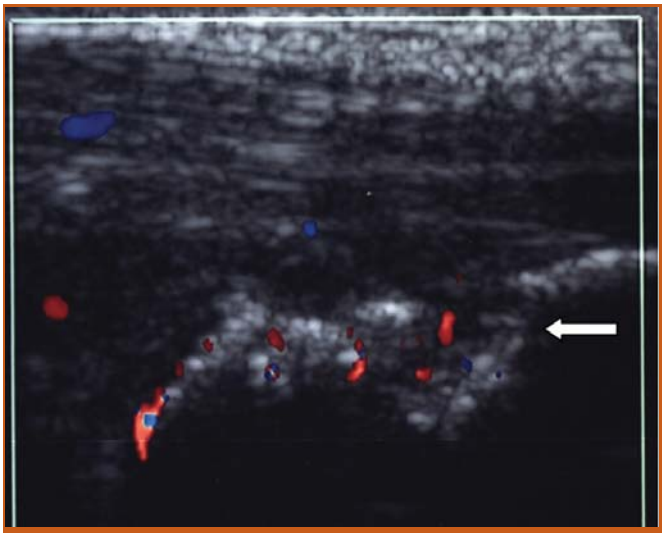


Fig.3 Ultrasound colour Doppler shows cortical irregularity with increase in thickness and colour flow at Achille's tendon insertion in calcaneum. The findings are suggestive of active enthesitis (arrow). (Courtesy of Dr. L. F. Chau)

Vertebral Abnormalities

Sundaram and Patton have described paravertebral ossification, which was noted in 17% of psoriasis patients.[17] The typical paravertebral ossification around the lower thoracic and upper lumbar vertebrae can be seen as an early manifestation of the disease. The ossification typically presents as fluffy and curvilinear radiodensity on the side of the vertebrae which is parallel with the vertebral body and intervertebral discs. (Fig.4) The ossification can merge with disc tissues. Its location away from the vertebral coloumn distinguishes the lesion from syndesmophytosis of ankylosing spondylitis. De Vlam and Mielant believe that the ossification shows less involvement of the apophyseal joint. The normal posterior spinal mobility leads to greater tensile forces anteriorly and promotes paravertebral inflammatory and bone formation. In ankylosing spondylitis, the involvement of the apophyesal joints reduces spinal mobility and leads to syndesmophytes formation.[18]

The cervical spine may demonstrate discovertebral junction and apophyseal joint erosion, as well as bone ankylosis and atlantoaxial subluxation. The atlantoaxial subluxation changes in psoriatic arthritis may resemble rheumatoid arthritis.[19]

Sacroiliac Joint Abnormalities

The reports on the prevalence and patterns of sacroiliac joint abnormalities in psoriasis show lots of discrepancies due to patient selection, techniques of radiological examinations, and image interpretations.

In plain radiographs, about 10 to 25% of patients with moderate to severe psoriatic skin diseases have sacroiliac joint abnormalities.[20,21] Bilateral sacroiliitis are more common than unilateral involvement.[22] The radiological abnormalities include sclerosis and erosions, first involving the ilial side. Joint space narrowing and ankylosis will happen in the late stage. However, the presence of ankylosis will be less than classical ankylosing spondylitis. Bone proliferation at the tendon insertion point including the iliac crests and ischial tuberosities is also frequently seen.

MRI is much more sensitive in the detection of early sacroiliitis. The changes include bone marrow oedema, sacroiliac joint erosion and sclerosis. Muche and colleagues[23] confirmed that sacroiliitis was very common in psoriatic arthritis as revealed by MR. They most often involve the dorsocaudal part of the joint in early disease, with subchondral bone marrow oedema a frequent finding. (Fig. 5) Williamson showed that sacroiliitis was present in 38% of a group of patients, some with absence of symptoms.[24]

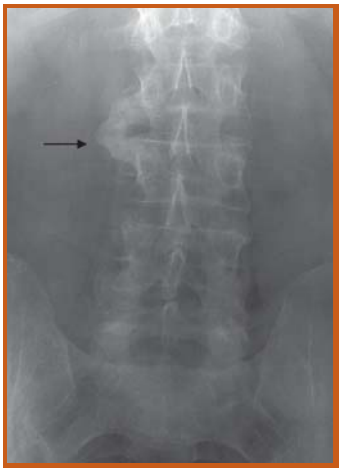


Fig.4 Curvilinear paravertebral bone ossification (arrow) at intervertebral space in upper lumbar level, which is characteristic of psoriatic arthritis. There is also presence of bilateral sacroiliitis with obliteration of the joint space and ankylosis, also typical for psoriatic arthropathy. (white arrow)

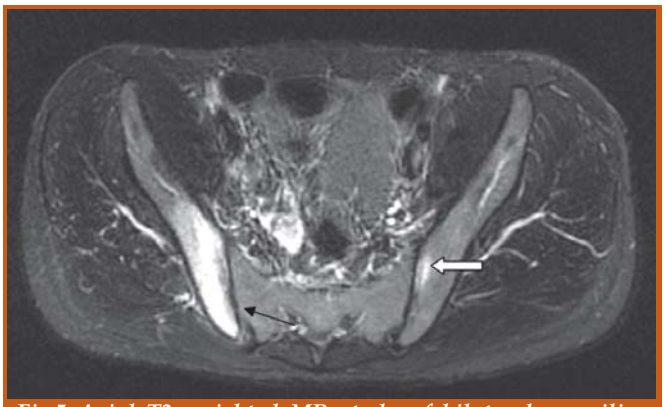


Fig.5 Axial T2 weighted MR study of bilateral sacroiliac joints in a psoriasis patient shows bone marrow oedema and erosion at the dorsocaudal aspect of the right sacroiliac joint. (arrow) The left sacroiliac joint also shows presence of bone marrow oedema in the ilium. (white arrow) The MR findings are characteristic of bilateral sacroiliitis.

Dactylitis

Dactylitis has been defined as one of the dominant clinical findings in psoriatic arthritis.[4,10] In the past, the sausage-like appearance was thought to be due to the presence of concomitant flexor tenosynovitis and arthritis of the metacarpophalangeal and interphalangeal joints. Olivieri and his group had demonstrated dactylitis in fingers and toes and showed the presence of tenosynovitis with effusion.[25-27] The flexor tendons were more often involved than extensor tendons, with small joint synovitis uncommon (from 6% to 27%). The same group also showed that peritendinous soft tissue oedema was causing the digital swelling, and bone marrow oedema was not seen at the entheseal insertions of the flexor and extensor tendons. They concluded that the dactylitis is due to flexor tenosynovitis and that distension of the joint capsule is not an indispensable condition for 'sausage-like' feature in dactylitis. They showed that clinical examination is a sufficient method for diagnosing tenosynovitis as it showed 100% sensitivity and specificity compared with MRI.

Bony proliferation with periostitis in metaphysis and diaphysis in the hands and feet is a late striking feature of psoriatic arthropathy following dactylitis.[29] Periosteal bone formation may cause significant cloaking of the phalanx, metacarpal or metatarsal bones. The changes are likely secondary to tenosynovitis.[25]

Differential Diagnosis

The radiographic abnormalities in psoriatic arthritis are similar to other seronegative spondyloarthropathies, namely ankylosing spondylitis and Reiter's syndrome. The Reiter's syndrome shows less frequent ankylosis than psoriasis and ankylosing spondylitis. The osteolysis in the terminal phalanx is also characteristic for psoriasis.

In psoriatic arthritis, there is often asymmetrical involvement of the upper and lower extremities with predilection for small joints in the hands and feet. In Reiter's syndrome, lower limb involvement is more common. For ankylosing spondylitis, axial involvement is more frequent.

For the spinal involvement, ankylosing spondylitis usually has symmetrical bilateral sacroiliitis, with squaring of the vertebral body and apophyseal joint involvement which is not frequent in psoriasis and Reiter's syndrome.

Asymmetrical joint involvement, absence of osteoporosis, and bony proliferation are typical features that distinguish psoriatic arthritis from rheumatoid arthritis. Paravertebral ossification and sacroiliitis are rarely seen in rheumatoid arthritis.

Summary

Psoriatic arthritis possesses certain radiological features such as asymmetrical DIP joint involvement, enthesitis, dactylitis and periostitis. The involvement of synovial and cartilaginous joints is also a common finding. MRI is more sensitive in detecting early bone marrow oedema, bony erosion, synovitis, and sacroiliitis. Ultrasound can also be used in the diagnosis of early enthesitis and dactylitis. Both MRI and ultrasound are useful imaging techniques to monitor the response in treatment of psoriasis.

References

1. Hellgren L: Association between rheumatoid arthritis and psoriasis in total populations. Acta Rheumatol Scan 15:316, 1969.
2. Wright V: Rheumatisim and psoriasis: A re-evaluation. Am J Med 27:454, 1959.
3. Moll JMH, Wright V: Psoriatic arthritis. Semin Arthritis Rheum 3:55, 1973.
4. Salvarani C, Cantini F, Olivieri I et al: Isolated peripheral enthesitis and / or dactylitis: A subset of psoriatic arthritis. J Rheumato 24: 1106, 1997.
5. Gold RH, Bassett LW, Theros EG: radiologic comparison of erosive polyarthritides with prominent interphalangeal involvement. Skeletal Radiology 8:89, 1982.
6. Martel W, Stuck KJ, Dworin AM, et al: erosive osteoarthritis and psoriatic arthritis: A radiologic comparison in the hand, wrist and foot. AJR 134(1): 125, 1980.
7. Fiocco U, Cozzi L, Rubaltelli L, et al. Long-term sonographic follow-up of rheumatoid and psoriatic proliferative knee joint synovitis. Br J Rheumatol 35:155,1996.
8. Cimmino MA, Parodi M, Innocenti S, et al. Dynamic magnetic resonance of the wrist in psoriatic arthritis reveals imaging patterns similar to those of rheumatoid arthritis. Arthritis Res Ther 7: R725, 2005;
9. Cimmino MA, Parodi M, Zampogna G, Paparo F, Silvestri E, Garlaschi G et al. Magnetic resonance imaging of the hand in psoriatic arthritis. J Rheumatol Suppl 83:39, 2009.
10. Salvarani C, Cantini F, Olivieri I et al: Distal extremity swelling with pitting edema in psoriatic arthritis. Evidence of 2 pathological mechanisms. J Rheumatol 26: 1831; 1999.
11. Oriente P, Biondi-Oriente C, Scarpa R. Psoriatic arthritis: clinical manifestations. Baillieres Clin Rheumatol 8: 277, 1994.
12. Lehtinen A, Taavitsainen M, Leirisalo-Repo M. Sonographic analysis of enthesopathy in the lower extremities of patients with spondylarthropathy. Clin Exp Rheumatol 12: 143, 1994.
13. Balint PV, Kane D, Wilson H, et al. Ultrasonography of entheseal insertions in the lower limb in spondyloarthropathy. Ann Rheu Dis 61: 905, 2002.
14. D'Agostino MA, Said-Nahal R, Hacquard-Bouder C et al. Assessment of peripheral enthesitis in the spondyloarthropathies by ultrasonography combined with power Doppler: a cross-sectional study. Arthritis Rheum 48: 523, 2003.
15. McGonagle D, Gibbon W, O'Connor P, Green M, Peases C, Emery P: Characteristic MRI entheseal changes of knee synovitis in spondylarthropathy. Arthritis Rheum 41: 694, 1998.
16. McGonagle D, Marzo-Ortega H, O'Connor P, Gibbon W, Pease C, Reece R, Emery P: The role of biomechanical factors and HLA B27 in magnetic resonance imaging determined bone changes in plantar fascia enthesopathy. Arthritis Rheum 46: 489, 2002.
17. Sundaram M, Patton JT: Paravertebreal ossification in psoriasis ad Reiter' disease. Br J Radiol 48:628, 1975.
18. De Vlam K, Mielant H, Veyes EM: Association between ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis: Reality or fiction? Clin Exp Rheumatol 14:5, 1996.
19. Lassoued S, Hamidou M, Fournie A, et al: Cervical spine involvement in psoriatic arthritis. J Rheumatol 16:251, 1989.
20. Harvie JN, Lester RS, Little AH: Sacroiliitis in severe psoriasis. AJR 127:579,1976.

21. Maldonado-Cocco JA, Porrini A, Garcia-Morteo O: Prevalence of sacroiliitis and ankylosing spondylitis in psoriasis patients. J Rheumatol 5:311,1978.
22. Helliwell PS, Hickling P, Wright V: do the radiological changes of classic ankylosing spondylitis differ from the changes found in the spondylitis associated with inflammatory bowel disease, psoriasis, and reactive arthritis. Ann Rheum Dis 57:135, 1998.
23. Muche B, Bollow M, Francois RJ et al. Anatomic structures involved in early- and late-stage sacroiliitis in spondylarthritis: a detailed analysis by contrast-enhanced magnetic resonance imaging. Arthritis Rheum 48(5); 2004.
24. Williamson L, Dockerty JL, Dalbeth N, McNally E, Ostlers S, Wordsworth BP. Clinical assessment of sacroiliitis and HLA-B27 are poor predictors of sacroiliitis diagnosed by MR in psoriatic arthritis. Rheumatol 43:85,2004.
25. Olivieri I, Barozzi L, Favaro L, Pierro A, De Matteis M, Borghi C, Padula A, Ferri S, Pavlica P. Dactylitis in patients with seronegative spondyloarthropathy: assessment by ultrasonography and magnetic resonance imaging. Arthritis Rheum. 39:1524,1996.
26. Olivieri I, Barozzi L, Pierro A, De Matteis M, Padula A, Pavlica P. Toe dactylitis in patients with spondyloarthropathy: assessment by magnetic resonance imaging. J Rheum 24:926,1997.
27. Olivieri I, Salvarani C, Cantini F, Scarano E, Padula A, Niccoli L, Ciancio G, Barozzi L. Fast spin echo-T2-weighted sequences with fat saturation in dactylitis of spondyloarthritis. Arthritis Rheum 46:2964,2002.
28. Olivieri I, Scarano E, Padula A, Giasi V. Dactylitis involving most of the fingers. Clin Exp Rheumatol.21:406, 2003.
29. Forrester DM, Kirkpatrik J: Periostitis and pseudoperiostitis. Radiology 118:597,1976.

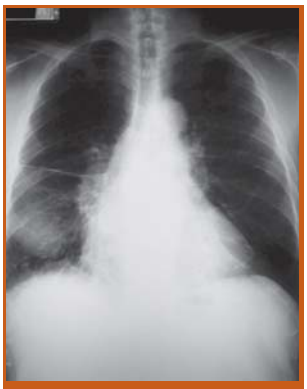
Radiology Quiz

Radiology Quiz

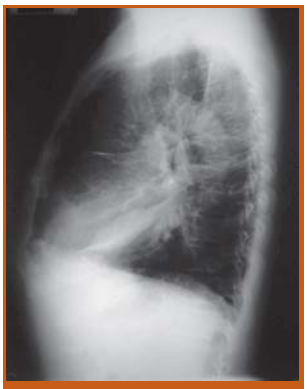
Dr. WK TSO

Chief of Service, Department of Radiology, Queen Mary Hospital

Dr. WK TSO



Case 1



Case 2

Case

M/71.
Admitted because of shortness of breath and ankle oedema.
He had right pleural effusion and signs of congestive cardiac failure on admission.
This chest X-Ray was taken before transfer of the patient for convalescence.

Questions:

What are the radiological findings?
What further investigation will you suggest to confirm the diagnosis?

(See P.37 for answers)

Outstanding Enterprise Award

HealthBaby awarded "The Best Biotechnology Company"

The 10TH《CAPITAL》Outstanding Enterprise Award was audited by a group of credible jury from government, political and business sectors, the result was ensured with accuracy and justice.

HealthBaby passed the FDA surprising inspection

HealthBaby USA branch (PacifiCord) successfully passed FDA (The Food and Drug Administration) rigorous and surprising inspection in January 2010. It proves that the processing and storage systems of HealthBaby meet international and professional standard.

Website:http://www.fda.gov/BiologicsBloodVaccines/SafetyAvailability/TissueSafety/ucm183755.htm

Excellent Quality Proved

Hence, 70%* parents entrusted HealthBaby.

***Source: Synovate Healthcare 2009 Cord Blood Bank Survey**

HealthBaby 生寶臍帶血庫
Most Accredited Cord Blood Bank in HK

Service Hotline
3188 8899
www.healthbaby.hk

HealthBaby laboratory is:
HOKLAS
PRODUCTIVITY AND QUALITY CERTIFICATE OF MERIT
caringcompany

HK | MACAU | TAIWAN | THAI | USA

Overcoming the Stigmata of Psoriasis

Prof. Peter WH LEE

Hon. Professor, Department of Psychiatry, The University of Hong Kong
Adjunct Professor, Department of Psychology, Chinese University of Hong Kong
Hon. Consultant in Clinical Psychology, Hong Kong Sanatorium & Hospital

Prof. Peter WH LEE

Stigmata and their Clinical Implications in Psoriasis:

Emotional and stress reactions to psoriasis are associated with physiological correlates, which in turn act upon organ vulnerability and debility resulting in flare-ups and exacerbations. In patients with psoriasis, stress in the form of stigmata has been indicated as a principal predictor of disability (Richards et al., 2001, Vardy et al., 2002).

Stigma and self stigma may affect the course of illness exacerbation and/or relative acquiescence in psoriasis. Fortune et al. (1997) noted that disability in psoriasis was best accounted for by anticipatory and maladaptive avoidant coping behaviour and experiences of rejection. Depression, common in psoriasis patients decreases the threshold for itch perception via increasing central nervous system opiate levels (Gupta, 1999). Yet, depression was found to have only a moderate correlation with symptom severity. Instead depression was more accurately predicted by perceived stigmata associated with deprivation of social touch (Gupta et al. 1998), being female, a stronger belief in perceived severity of the consequences of psoriasis, and poor coping strategies (Fortune et al., 2002). Likewise, presence of distress in the form of uncontrolled and excessive worrying slowed clearance of psoriasis using a standard therapy of psoralen plus ultraviolet A treatment while combined standard therapy plus psychological management yielded superior results at up to 6 months follow-up (Fortune et al., 2003).

Goffman (1963) cautioned that stigma "is deeply discrediting", and a stigmatised person may be reduced "from a whole and usual person to a tainted, discounted one". Link, Yang, Phelan, and Collins (2004) further noted that labelling, stereotyping, cognitive separation, emotional reactions, status loss and discrimination may all be involved. Psoriasis may be more accurately seen as a recurrent medical condition as well as an adverse and salient social (and internal) stimulus for the afflicted. The adverse consequences of psoriasis include not only physical sufferings, but also the precarious management of visible regions, coping with a subjectively all-consuming disease, psychological co-morbidity, and social vulnerability (Schmid-ott et al., 2005). Multiple interpersonal concerns were reported by psoriasis patients. Krueger et al. (2001) provided survey results which indicated that 27% of the patients had difficulties with sexual activities, 81% were embarrassed with visible psoriasis, and 88% expressed concerns about the disease worsening. Likewise, Langley et al. (2005) in their review of the literature indicated that up to a third of patients with psoriasis suffered from pathological worry and anxiety which impinge on "all aspects" of thepatients' daily life.

Stigmata are multi-faceted. A useful tool for measuring stigma is the Questionnaire on Experience with Skin Complaints (QES) by Ginsburg & Link (1989). Six dimensions of stigmata were measured including:

- Interference of skin symptoms and self-esteem: feelings of being worthless, alone or unclean;
- Outward appearance and situation-caused retreat: experience of lack of physical attractiveness or sexual desirability, special ways of dressing, avoidance of public situations;
- Rejection and devaluation: anticipated and perceived negative reactions of others;
- Composure: calmness and confidence in a satisfactory life despite the psoriasis;
- Concealment: tendencies toward hiding the diagnosis and keeping the disease secret;
- Experienced refusal: feelings of stigmatisation in specific situations such as shopping or usual public transport.

An all Encompassing Clinical Management Approach Advocated

Psoriasis is thus more effectively managed with a combined medical as well as social learning perspective, incorporating variables such as the perceived degree of social rejection, suspicion and misguided fear of infection. Indeed, Vardy et al. (2002) provided data to indicate that severity of perceived stigma mediated the impact of severity of psoriasis on quality of life. Effective management and harm containment of the all-encompassing adverse impact of psoriasis should best incorporate an understanding from an insider's perspective on "how it is like" to live with psoriasis. Therapeutic aims should include targeted attempts to reduce the challenges of psoriasis on the person's self image, social functioning and emotional reactions.

Illness management is more effective to the extent that the external signs of psoriasis detrimental to the social standing (stigma) and self esteem (self stigmata) could be reduced. The two interlinking processes are likely to be mutually potentiating. Perceived stigma and social rejection prompt fear, self depreciation, avoidance,

anxiety, uncertainty, insecurity feelings, depression and demoralisation. Self stigma that arises from the patients' own rejection of the condition as well as projection of rejection by others may aggravate self devaluation and "spread" of negativity. The phenomenon of spread has been well reported in the health psychology literature. Spread refers to rejection and devaluation of the entire person over and beyond the confines of the symptoms. Pain and itchiness aside, psoriasis diminishes a person's social standing and self esteem. Personal weaknesses may be further opened up leading to a vicious cycle of fixation and worry about the sight, pruritus and uncertain prognosis of psoriasis, anxiety and depressed mood, reduced quality of life, enhanced physiological stress reactions, negative fixation, symptom flare-ups and exacerbations. On the other hand, overcoming and accepting the sometimes unavoidable stigmata of psoriasis may lead to greater peace of mind, less emotional upheavals, and greater treatment adherence. Acceptance alongside realistic reduction of perceived stigmata reduces depressed mood and anxiety, more positive overall quality of life, reduced inflammations and exacerbations.

The Unity of Mind and Body

The unity of mind and body and their inevitable interactions is very well illustrated in conditions such as psoriasis. Few clinicians would disagree that psychological or behavioural factors play a role in almost every medical condition, especially in conditions that are visible and stigmatising and those with accompanying adverse psychological aftermaths of low self esteem, depression, anxiety, avoidance, and social awkwardness.

While the diathesis or illness susceptibility is clearly of genetic origin, some individuals may become more vulnerable and may even be designated as being skin reactors. Individuals with alexithymia having more difficulties in identifying feelings and describing feelings, with poor emotional regulation and excessive preoccupation with physical symptoms and external events were implicated as being at greater risk (Richards et al., 2005).

On a broader consideration, today's terminology has discarded the term "psychosomatic illness" which implicated only a limited number of disease conditions as having a psychosomatic origin. Instead, the more encompassing term of "psychosomatic approach" to illness management is regarded as being more clinically realistic. The DSM diagnostic nomenclature (APA, 1994) further delineates the multifarious psychosomatic processes under the diagnostic category of "psychological factors affecting general medical condition". Specifically, psychological factors may affect a medical condition through exacerbating the underlying disorder, reducing coping effectiveness, prompting maladaptive behaviours, intensify stress related physiological responses and lead to further outbreaks of the underlying disorder. In psoriasis, psycho-behavioural-neuro-immunological factors implicating chronic inflammatory changes, symptom exacerbation, IL-22, TH-17 cells, and depression may be implicated in its recurrent cycle of initiation, progression, aggravation and relative acquiescence (Leibovici et al, 2010).

Improved Awareness and Training Required in Detecting Psychological Distress

A misleading feature of psoriasis is that it does not even have to be visible for the patient to fear and anticipate social rejection, and also that it does not need to be objectively severe to warrant severe disability and distress (Ginsburg, 1995). It is thus not surprising to note that while most clinicians would agree that psychological factors may affect the course and management of psoriasis, they are also poor in detecting psychological distress in their patients. A low consensus between the respective patients' and their physicians' reports of presence of severe psychological distress was noted (Richards et al., 2004). Indeed regardless of the doctor's empathy level, severe psychological distress in 61% of their patients was not identified. Even when severe anxiety and depressive reactions were noted, in the majority of cases, no further action was taken following the consultation. Sampogna et al. (2003) also provided disappointing data to indicate that dermatologists did not have an accurate perception of the extent of psychiatric disturbances in their patients with skin conditions.

Richards et al. (2004) thus asserted that "It is of key importance that psychological distress is appropriately recognised and addressed in a holistic or biopsychosocial approach to patient management". They thus advocated the use of specific guidelines and education in psychological detection skills as well as routine administration of psychometric screening tools such as the HADS.

Janowski and Pietrzak (2008) provided helpful indications to guide clinicians in referring psoriasis patients for psychological interventions including:

- Presence of psychiatric and behavioural disorders (depression and anxiety disorders, suicidal ideation) as co-morbidity with psoriasis;
- History indicating psychological stress as a psoriasis-triggering or aggravating factor;
- Significantly decreased quality of life, where social relationships, sexual functioning and self-esteem are seriously affected;
- Increased pruritus;
- Increased feelings of stigmatisation as indicated by sensitised attention to potential rejecting behaviours of others, biased interpretation of others' behaviours and intentions, or anticipatory expectations of unfavourable reactions from others;
- Psoriasis being unresponsive to standard pharmacological treatments;
- Children and adolescents with psoriasis (given psoriasis being an increased risk for disturbances of normal psychosocial development).

Concluding Remarks and Recommendations

Instead of a cross-sectional approach where symptoms are managed as and when they arise, a longitudinal approach to psoriasis with understanding of the individual patient's vulnerability, high stress points,

sense of stigmata, and factors associated with exacerbation is advocated. The aim is to equip the patient with awareness of aggravating and stress factors inherent in themselves and in their life circumstances, and learn to short-circuit stress responses and aggravations at the earliest stage. Self monitoring of illness aggravating correlates in the form of life events, behavioural and emotional responses are useful. For example, psychological variables may maintain and exacerbate psoriasis by eliciting poor compliance and scratching behaviours. A general attitude of negative affectivity may prompt more helpless and depressive responses, rendering adherence to treatment unreliable (Charman and Horne, 1997).

A systems approach to managing psoriasis is also proposed. The clinician needs to recognise that psoriasis is at the same time a lifelong medical illness as well as an illness with vast social and personal implications. Understanding the patient's perceptions regarding the illness, his/her unique life circumstances, as well as reactions to psoriasis facilitates better physician-patient communication, trust and collaboration.

Effective management of negative emotions needs to be built into the overall management plan. Anger, depression and anxiety are common emotional conditions of maladjustment. Anger leads to non-adherence, emotional instability, and increased scratching behaviours. Depressed mood leads to and may be aggravated by social avoidance, low self-esteem, sense of helplessness and hopelessness. Anxiety leads to dread, insecurity and uncertainty feelings, inhibition, and reduction in meaning and gratification in daily living. All in all, vulnerability is accentuated by chronic negative emotions, leading to unsatisfactory illness control and management. The cognitive behavioural approach to management of emotions postulates that extreme, distorted and over-inclusive thoughts underlie negative emotions. Understanding and managing the patient's conceptions about his/her illness and associating coping styles facilitates emotional management and a less stormy illness course (Fortune et al., 2002, 2004; Zachariae et al. 1996).

Specific interventions have also been indicated to be useful. For example, behavioural methods for habit reversal in reducing scratching had been indicated as being useful. Illness control was demonstrated to improve significantly with targeted psychological therapies (autogenic training, relaxation, self control, stress management, and management of illness attribution) compared with interventions consisting only of information giving and psychoeducation (Ehlers et al., 1995).

Psychological screening and interventions clearly hold the promise for being a useful adjunctive treatment in the overall management of psoriasis.

References

- American Psychiatric Association (1994): Diagnostic and statistical manual of mental disorders, fourth edition, text revision (DSM-IV-TR), APA.
- Charman D., Horne D.L. (1997): Atopic dermatitis, in Baum A., Newman S., Weinman J., West R., McManus C. (eds.) Cambridge Handbook of Psychology, Health and Medicine, Cambridge University Press, UK.
- Ehlers A., Stangier U., Gieler U. (1995): Treatment of atopic dermatitis: a comparison of psychological and dermatological approaches to relapse prevention, Journal of Consulting and Clinical Psychology, 63(4), 624-635.
- Fortune, D.G., Richards, H.L., Griffiths, C.E.M., & Main, C.J. (2002): Psychological stress, distress and disability in patients with psoriasis: Consensus and variation in the contribution of illness perceptions, coping and alexithymia, British Journal of Clinical Psychology, 41, 157-174.
- Fortune, D.G., Richards, H.L., Griffiths, C.E.M. (2002): Psychological stress, distress and disability in patients with psoriasis: consensus and variation in the contribution of illness perceptions, coping and alexithymia. British Journal of Clinical Psychology, 41: 157-174.
- Fortune D.G., Richards H.L., Kirby B. (2003): Psychological distress impairs clearance of psoriasis in patients treated with photochemotherapy. Archives of Dermatology, 139: 752-756.
- Fortune D.G., Richards H.L., Griffiths C.E.M., Main C.J. (2004): Targeting cognitive-behaviour therapy to patients' implicit model of psoriasis: Results from a patient preference controlled trial, British Journal of Clinical Psychology, 43, 65-82.
- Ginsburg I.H. Link B.G. (1989): Feelings of stigmatisation in patients with psoriasis. Journal American Academy of Dermatology, 20: 53-63.
- Ginsburg I.H. (1995): Psychological and psychophysiological aspects of psoriasis. Dermatology Clinics of North America, 13: 793-804.
- Goffman, E. (1963). Stigma: Notes on the management of spoiled identity. New York: Simon & Schuster Inc.
- Gupta M.A., Gupta A.K., Watteel G.N. (1998): Perceived deprivation of social touch in psoriasis is associated with greater psychologic morbidity: an index of the stigma experience in dermatologic disorders. Cutis, 61:339-342.
- Gupta M.A., Gupta A.K. (1999): Depression modulates pruritus perception: a study of pruritus in psoriasis, atopic dermatitis and chronic idiopathic urticaria. Annals of New York Academy of Sciences, 885: 394-395.
- Langley R.G.B., Krueger G.G., Griffiths C.E.M. (2005): Psoriasis: epidemiology, clinical features, and quality of life. Annals of Rheumatological Diseases, 64 (Supplement ii), ii18-ii23.
- Link, B.G., Yang, L.H., Phelan, J.C. & Collins, P.Y. (2004). Measuring mental illness stigma. Schizophrenia Bulletin, 30, 511-541.
- Richards H.L., Fortune D.g., Griffiths C.E.M. (2001): The contribution of perception of stigmatisation to disability in patients with psoriasis. Journal of psychosomatic Reserach, 50: 11-15.
- Richards H.L. Fortune D.G., Weidmann A., Sweeney S.K.T. Griffiths C.E.M. (2004): Detection of psychological distress in patients with psoriasis: low consensus between dermatologist and patient, British Journal of Dermatology, 151: 1227-1233.
- Richards H.L., Fortune D.G., Griffiths C.E.M. (2005): Alexithymia in patients with psoriasis: clinical correlates and psychometric properties of the Toronto Alexithymia Scale-2-. Journal of Psychosomatic Research, 58: 89-96.
- Sampogna F., Picardi A., Melchi C.F. (2003): The impact of skin diseases on patients: comparing dermatologists' opinions with research data collected on their patients. British Journal of Dermatology, 148: 989-995.
- Schmid-Ott G., Kunsebeck H.W., Jager B., Sittig U., Hofste N., Ott R., Malewski P. Lamprecht F. (2005): Significance of the stigmatization experience of psoriasis patients: a 1-year follow-up of the illness and its psychosocial consequences in men and women, Acta Dermatologic Venereology, 85: 27-32.
- Vardy D., Besser A., Amir M. (2002): Experiences of stigmatization play a role in mediating the impact of disease severity on quality of life in psoriasis patients. British Journal of Dermatology, 147: 736-742.
- Veibovici V., Canetti L., Yahalomi S., Cooper-Kazaz R., Bonne O., Ingber A., Bachar E. (2010): Well being, psychopathology and coping strategies in psoriasis compared with atopic dermatitis: a controlled study, European Journal of Dermatology and Venereology, advanced publication.
- Zachariae R., Oster H., Bjerring P., Kragballe K. (1996): Effects of psychologic intervention on psoriasis: A preliminary report, Journal of American Academy of Dermatology, 34: 1008-1015.

香港醫學組織聯會

適健中心 The Centre on Health & Wellness
(香港復康會屬下單位)

合辦

催眠治療臨床應用工作坊(進階培訓)

適當運用催眠治療於心理輔導的歷程中，除了能有效處理一些在日常生活裡(意識層面)出現的癥狀問題外如：失眠及痛症等，還能夠爲那些被潛藏的情緒困擾，找出根源及給予治本之果效。

在臨床研究中，更指出催眠治療對創傷性經歷帶來的情緒如：焦慮與恐懼等、身心痛症及慢性痛症.....均有明顯的效果。

日期及時間	:	2010 年 5 月 14 日 至 6 月 4 日 (逢星期五, 5 月 21 日除外) 晚上 6:30 至 9:30
地點	:	香港灣仔軒尼詩道 15 號溫莎公爵社會服務大廈 4 字樓會議室
課程收費	:	$950 (整個課程共 3 節，每節 3 小時)
導師	:	尹婉萍小姐 (註冊認可催眠治療培訓導師、註冊臨床催眠治療師、註冊社工)

課程大綱

日期/節數	題目
2010 年 5 月 14 日 (第一節)	• 催眠治療與心理輔導的配合 (一) 評估的重要性及提問的技巧 • 催眠治療提示的運用(一)簡接提示與喻意的運用
2010 年 5 月 28 日 (第二節)	• 催眠治療與心理輔導的配合 (二) 身心反應與情緒反應的互動關係 • 催眠治療提示的運用(二)與自我催眠的配合 • 催眠治療與創傷性經歷的處理
2010 年 6 月 4 日 (第三節)	• 加深導入催眠意境的方法 • 舒導抑壓性的情緒 • 處理突發性的情緒 • 催眠治療與緩減痛症

如對此課程有任何查詢，
可致電香港醫學組織聯會秘書處 2527 8898 或電郵至 info@fmshk.org

有興趣之人士可登入本網站 **www.fmshk.org** 下載報名表格

Biologic Therapies for Psoriatic Arthritis

Dr. Gavin Ka-wing LEE

Dr. Gavin Ka-wing LEE

Introduction

Psoriatic arthritis (PsA) is a disease with diverse manifestations. Oligoarthritis is a common pattern identified; however, it could take the form of rheumatoid-like disease or inflammatory spondylitis. Distal interphalangeal joint involvement and arthritis mutilans are distinctive features of PsA. Enthesitis and dactylitis should also be aware of. It is important to recognise the different forms of presentation and to realise that therapeutic options are not equally effective across different patterns of PsA.

Moreover, traditional disease modifying anti-rheumatic drugs (DMARDs) have limitations and more effective agents are very much needed. With the advances in the understanding of immunological disturbances in inflammatory arthritis and the success of using anti-TNF in the management of rheumatoid arthritis, investigators have identified biologic agents that have filled at least to some extent the gaps in the treatment of PsA.

Different anti-TNF agents have been reported to be efficacious in treating PsA, which was also supported by systemic reviews and meta-analyses of the data available in the literature[1-4]. Therefore, various anti-TNF agents have been approved in different national regulatory agencies for their indication in PsA. Other non anti-TNF agents have been tested and would be discussed accordingly.

Anti-TNF in the Management of Psoriatic Arthritis (Table 1)

Etanercept

A soluble TNF receptor-FC fusion protein, etanercept, was shown to be effective in a relatively small (No. of subject = 60) double blind placebo-controlled study in improving clinical signs (PsARC, ACR20 etc) and inflammatory markers (ESR and CRP)[5]. The efficacy in using 25mg twice weekly etanercept was reconfirmed by a larger scale consisting over 200 patients with PsA[6]. Radiological progression was also shown to be inhibited by the active treatment group in this large scale trial.

Infliximab

In an open label study of infliximab treatment for PsA, it had significant reduction of inflammation as detected by MRI[7]. This chimeric monoclonal anti-TNF antibody administrated by intravenous infusion was tested in the IMPACT and IMPACT2[8,9]. Signs and symptoms were shown to be significantly improved in the active treatment group as compared to the placebo. Both the sustainability of its results and the inhibition of radiological progression were further demonstrated in the 2-year extension of IMPACT study[10].

Table 1.

Agent		Trial	No. Active/control	Duration (weeks)	Outcome
Etanercept	Mease 2000		30/30	12	PsARC, ACR 20/50
	Mease 2004		101/104	24	ACR 20, PsARC, HAQ-DI
Infliximab	Antoni 2005	IMPACT	52/52	16	ACR 20/50/70, PsARC, HAQ-DI
Adalimumab	Antoni 2005	IMPACT 2	100/100	16	ACR 20/50/70, PsARC, HAQ-DI, PASI 50/70/90, SF-36
	Mease 2005	ADEPT	151/162	24	ACR20/50/70, TSS, PsARC, PASI50/70/90, HAQ-DI, SF-36
	Genovese 2007		51/49	24	ACR20/50/70, TSS, PsARC, HAQ-DI, SF-36
Golimumab	Kavanaugh 2009	GO-REVEAL	292/113	24	ACR20/50/70, PASI50/70/90, HAQ-DI, SF-36, NAPSI, MASES

Adalimumab

Adalimumab is a humanised monoclonal anti-TNF antibody given by subcutaneous injection on every other week. It has been tested in a pivotal trial, ADEPT, which is a 24-week randomised double-blind, parallel group, placebo-controlled trial. It demonstrated significant improvement in joint and skin manifestations[11]. Modified total Sharp score of radiographic structural damage was inhibited at week 24 in the adalimumab arm. Another study conducted in PsA patients who failed DMARDs showed that addition of adalimumab (vs placebo) resulted in improvement in disease control[12].

Golimumab

Golimumab is an humanised monoclonal antibody against TNF-α , which is administrated subcutaneously on a monthly basis. Efficacy and safety in using golimumab for the treatment of PsA has been demonstrated in GO-REVEAL, a 24-week randomised placebo controlled trial[13]. Patients with active treatment (golimumab 50mg or 100mg) had significantly higher proportions in achieving ACR20, ACR50 and ACR70. The beneficial effects on skin involvement, nail disease and enthesitis were also documented in this trial.

Biologic Therapies Other than Anti-TNF

Alefacept

An approved agent for the treatment of plaque psoriasis, Alefacept, is a fusion protein of the first extracellular domains of human lymphocyte function-associated antigen 3 (LFA-3) and Fc portion of IgG1. Alefacept inhibits T cell activation by blocking co-stimulation CD2-LFA-3. It was tested in a setting of weekly intramuscular injection for 12 weeks in combination of methotrexate[14]. However, statistically significant different improvement was only achieved in ACR 20 but not ACR 50 or ACR 70. An open-label extension was also reported recently[15]. Nevertheless, it has not been accepted as one of the options used in the treatment of PsA.

Ustekinumab

An inhibitor of interleukin 12/23, ustekinumab binds to the P40 subunit of these two interleukins preventing their binding to the 12Rβ1 receptor on the surface of T cell, NK cells and antigen presenting cells. It has already been approved for the management of plaque psoriasis. A crossover trial was conducted in 146 PsA subjects, which showed promising results in terms of its efficacy in improving the manifestations of joint inflammation, skin disease, enthesopathy and dactylitis[16]. A larger scale study is needed to confirm its usefulness in the management of PsA.

Clinical Guidelines and Recommendations

Guidance in using anti-TNF in PsA has been published by professional societies, like the British Society for Rheumatology (BSR)[17]. Patients with pure axial disease are suggested to adopt the guidance similar for patients with ankylosing spondylitis. Patients with peripheral arthritis who continue to have persistent active disease (defined as ≥ 3 SJC and ≥ 3 TJC on 2 separate occasions 1 month apart) despite an adequate trial of 2 standard DMARDs individually or in combination (sulphasalazine, methotrexate, cyclosporin or leflunomide) should be considered for the use of anti-TNF. Apart from the above key principles, the guidance also provides details on the definition of an adequate trial of DMARDs, exclusion criteria, criteria for withdrawal of therapy and assessment during anti-TNF therapy.

More recently treatment recommendations for PsA have been developed by an international organisation, namely, the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA)[18]. The recommendations have a comprehensive coverage of clinical manifestations of PsA including peripheral arthritis, skin and nail diseases, axial disease, dactylitis and enthesitis. It is apparent that the use of biologics in particularly anti-TNF is recommended in patients with moderate to severe degree of all these manifestations (Table 2).

Despite of all these enthusiasms, one should remember the potential adverse effects that may happen with the use of anti-TNF. Infections, in particularly tuberculosis(TB), are known complications. Proper TB screening has been proved to significantly lower the risk of developing TB during the course of anti-TNF therapies. Clinical assessment of cardiac function and the possibility of having an underlying malignancy or an autoimmune disorder such as lupus should all be carried out. Pre-treatment serologic testing for hepatitis B and C status are also required.

Conclusion

The treatment of PsA has significantly changed by the development of biologic therapies. Clinicians should all be aware of the erosive and progressive nature of PsA. All patients with moderate to severe disease should not be denied of the chance for a better control of their disease by using biologic therapies if clinically appropriate. Therefore, early detection, early treatment and timely referral to specialists could not be over-emphasised.

References

1. Kavanaugh AF, Ritchlin CT. Systemic review of treatments for psoriatic arthritis: an evidence based approach and basis for treatment guidelines. J Rheumatol 2006;33:1417-1421.
2. Soriano ER, McHugh NJ. Therapies for peripheral joint disease in psoriatic arthritis. A systematic review. J Rheumatol 2006;33:1422-1430.
3. Nash P. Therapies for axial disease in psoriatic arthritis. A systematic review. J Rheumatol 2006;33:1431-1434.
4. Saad AA, Symmons DPM, Noyce PR, Ashcroft DM. Risks and benefits of tumor necrosis factor- inhibitors in the management of psoriatic arthritis: Systematic review and metaanalysis of randomized controlled trials. J Rheumatol 2008;35:883-890.
5. Mease PJ, Goffe BS, Metz J, VanderStoep A, Finck B, Burge DJ. Etanercept in the treatment of psoriatic arthritis and psoriasis: a randomised trial. Lancet 2000;356:385-390.
6. Mease PJ, Kivitz AJ, Burch FX, et al. Etanercept treatment of psoriatic arthritis: safety, efficacy, and effect on disease progression. Arthritis Rheum 2004;50:2264-2272.
7. Antoni C, Dechant C, Hanns-Martin Lorenz PD et al. Open-label study of infliximab treatment for psoriatic arthritis: clinical and magnetic imaging measurements of reduction of inflammation. Arthritis Rheum 2002;47:506-512.
8. Antoni CE, Kavanaugh A, Kirkham B, et al. Sustained benefits of infliximab therapy for dermatologic and articular manifestations of psoriatic arthritis: results from the infliximab multinational psoriatic arthritis controlled trial (IMPACT). Arthritis Rheum 2005;52:1227-1236.
9. Antoni C, Krueger GG, de Vlam K, et al. Infliximab improves signs and symptoms of psoriatic arthritis: results of the IMPACT 2 trial. Ann Rheum Dis 2005;64:1150-1157.
10. Antoni CE, Kavanaugh A, van der Heijde D, et al. Two-year efficacy and safety of infliximab treatment in patients with active psoriatic arthritis: findings of the infliximab multinational psoriatic arthritis controlled trial (IMPACT). J Rheumatol 2008;35:869-876.
11. Mease PJ, Gladman DD, Ritchlin CT, et al. Adalimumab for the treatment of patients with moderately to severely active psoriatic arthritis. Arthritis Rheum 2005;52:3279-3289.
12. Genovese MC, Mease PJ, Thomson GT, et al. Safety and efficacy of adalimumab in treatment of patients with psoriatic arthritis who had failed disease modifying anti-rheumatic drug therapy. J Rheumatol 2007;34:1040-1050.
13. Kavanaugh A, McInnes I, Mease P, et al. Golimumab, a new human tumor necrosis factor antibody, administered every four weeks as a subcutaneous injection in psoriatic arthritis. Arthritis Rheum 2009;60:976-986.
14. Mease P, Gladman DD, Keystone EC. Alefacept in combination with methotrexate for the treatment of psoriatic arthritis. Arthritis Rheum 2006;54:1638-1645.
15. Mease PJ, Reich K. Alefacept with methotrexate for treatment of psoriatic arthritis: open-label extension of a randomised, double-blind, placebo controlled study. J Am Acad Dermatol 2009;60:402-411.
16. Gottlieb A, Menter A, Mendelsohn A, et al. Ustekinumab, a human interleukin 12/23 monoclonal antibody, for psoriatic arthritis: randomised, double-blind, placebo-controlled, crossover trial. Lancet 2009;373:633-640
17. Kyle S, Chandler D, Griffiths CEM, et al. Guideline for anti-TNF-therapy in psoriatic arthritis. Rheumatology 2005;44:390-397.
18. Ritchlin CT, Kavanaugh A, Gladman DD, et al. Treatment recommendations for psoriatic arthritis. Ann Rheum Dis 2009;68:1387-1394

Table 2

Systemic Rx	Peripheral arthritis	Skin and nail disease	Axial disease	Dactylitis	Enthesitis
NSAID	✓	x	✓	✓	✓
DMARDs	SSZ, MTX, CycA, LEF	MTX, CycA	x	?	?
Biologics	Anti-TNF	Anti-TNF, Alefacept	Anti-TNF	Anti-TNF	Anti-TNF

Travels

Prof. CL LAI

Prof. CL LAI

Asked to write about leisure activities for the medical community, I immediately thought of Travels. I have two kinds of travels in mind: one spatial and the other mental. I cannot do without either of them, especially the latter kind!

Being raised in a rather closeted environment (my parents would not even consider sending me to live in a hostel for my university studies), my first travel outside Hong Kong had to wait till I graduated from my medical studies in 1970! And it was - you would hardly believe this - to Macau for one night! Which is the reason why I planned and saved and super-planned for a one week 'flying' visit to Rome, Florence and Venice, at the very first opportunity I could grab, and this was only after completing my first year as a registrar at the University Department of Medicine, in July 1972. In those days interns did not have a single day of leave throughout their internship; I had to wait till I had been a registrar for a full year before I was allowed a one week holiday. And even that one week hard-earned holiday was 'unofficial'. As a result though my ward physician gave me his full blessing before I took my first real travel abroad, I was terrified when I was one full day late for returning to work due to a flight delay from Venice to Rome! My colleagues all warned me on my return that Professor David Todd, who was then the virtual head of the Department, was very angry about my escape abroad! Well, I survived Professor Todd's anger, but only just!

Since then I have become famous in my Department for my annual, and often more frequent than annual, travels abroad. For how can one not pine to visit all the beautiful, and often more than beautiful, places on our wonderful planet! The aquamarine depths of the oceans surrounding the Thai islands, the ever-changing colours of the lakes of Jiuzhaigou, the innumerable shades of reds and oranges of the autumn maple leaves in Japan (which are smaller and more delicate than maple leaves elsewhere), the awesome chasm of the Grand Canyon, the wonderful chaos of the Times Square, the Georgian and Victorian buildings of London which seemed to me such old friends even on my very first visit in 1974, the majesty and symmetry of Haussmann's plan for Paris, the ancient architecture of old Rome where each cobblestone one treads might have been touched by Julius Caesar or Cicero, the mystic centre of Jerusalem so heavily and tragically laden with religious fervour (Muslim Dome of the Rock, Christian Via Dolorosa, and Hebrew Wailing Wall), and, back to nature, the deep dark glory of Homer's wine-red Aegean Sea... My list of just ten places is less than adequate to express my yearning to experience the natural and man-made wonders of the world! I have not mentioned the compressed complexities of the South Island landscape of New Zealand, the seething restless surface of the Yellowstone Park nor the bluer-than-blue Tuscan sky! But how can I even attempt to do justice to the infinite variety of the earth by just a few hundred words!

Maybe you have been wondering why I chose to visit Italy on my first sneak 'flying' visit. The answer is very simple. Well before I could travel abroad without depending on my parents' permission and fund, I had been travelling mentally for over one decade, by reading any books I chose to read! I was saturated with Michelangelo and his Vatican frescos and his sculptures scattered over Rome and Florence before my escapade in 1972. What freedom one could/can achieve just by sitting in a chair. I could read novels and histories and art-books right under my mother's watchful eyes without her suspecting that I was not diligently studying my school textbooks. And try to figure out the miracle of tracking the innermost workings (and foibles) of the mind of any genius just by looking at the written words printed on a page! I love books probably more than even travelling. My love for books, i.e, non-medical books of almost any variety, is so great that I find it difficult to walk by a bookshop without entering for a good browse, in case any new good books have arrived in the last few days! My love for books extends to the smell and the feel of the pages of a paperback. I recently read that Philip Roth, the American novelist who surely should be awarded a Nobel Prize, predicts that computer comics, digital wares and blockbuster films will soon replace the public appetite for books, good old books with pages to turn over with your own fingers. I sincerely hope this is not going to be true!

One of my favourite motto to medical students is 'No knowledge is useless'. By this I do not just mean a broad-based medical knowledge; I include all we can possibly learn from the wisdom and insights of other people's superior mental faculties. In my early teens, I learned to love the gymnastics of the English language from Charles Dickens; I learned to look at the murky intricacies of the human mind from Iris Murdoch and Feodor Dostoyevsky; I learned about wild passions from Emily Bronte. In the ensuing five decades [you already know my (old) age from my year of graduation!], I learned more, much more, from my mental odyssey with such diverse authors (in alphabetical order) as: Saul Bellow and his massive epics of American struggles, Boccaccio and his pornographic 'Decameron', Joseph Conrad and his

mindscape of darkness, Charles Darwin and his revolutionary evolution findings (albeit written in clotted prose), George Eliot and her more-than-masculine intellect, TS Eliot and his elegant, difficult verse lines, Flaubert and his meticulous human depiction, Thomas Hardy and his sardonic universe, Hermann Hesse and his strange journeys into the mind, Marcel Proust and his wordy meanderings that sparkle with new insights, and Sophocles and his astonishing detective drama of Oedipex who kills his father and marries his mother unknowingly (the origin of Freud's Oedipex complex)... And of course towering above them all, there is the all encompassing illimitable intellect of William Shakespeare, whom I first approached with much trepidation, only to be overwhelmed by his tremendous human insights, verbal dexterity and poetic profundities. (Incidentally I still approach Shakespeare with some trepidation!)

But once again, how can I do justice to the mental travelling through books by just a few hundred words. The rest, as Shakespeare says, is silence...

Running Asia - Chronicles of a Runner's Wife

Dr. Siaw Ing YEO

Dr. Siaw Ing YEO

Honorary Clinical Associate, Division of Rheumatology and Clinical Immunology, Department of Medicine, The University of Hong Kong

Serendipity was what started William running. We were living in East London at the time. The route of the London Marathon partly traverses the wind tunnels that meander between the towering heights of Canary Wharf. I hadn't realised how serious this running business was then. One evening, the runner returned with an ankle the size of a football, the colour of blueberries and feel of Jell-O. He'd continue running despite having jammed the ankle in a pothole off East India Road. He was off training for 6 weeks.

When we moved to Hong Kong, I had imagined that I would just be a bystander. Not so. I sat in Victoria Park for a considerable time one morning, awaiting his return during the last Standard Chartered Hong Kong Marathon. I was waiting at the wrong spot.

But it was pleasant to witness the startling change of night sky into a lovely shade of pink as the sun appeared, and hear the merry chirp of birds, fresh from their sleep. The Sunday regulars (a group of old-aged pensioners) were not so happy, however, to have me camp on the bench where they meet to socialise. I soon got the message and moved off to more welcoming pastures!

1. Running along the Island East Corridor at dawn

It was so much more interesting when William decided to chase the marathons across Asia. At last, I could be a tourist. First stop was Kuala Lumpur, Malaysia. It was hot and humid, but the shopping was satisfying. Satay, laksa and durian were a bonus. The marathon route was mainly composed of a series of seemingly interminable highways. But a group of mothers with their strollers along the 10km route was a surprising and heartwarming sight.

The Borneo Marathon is held in Kota Kinabalu annually. This is where you will find the tallest mountain in Southeast Asia, Mount Kinabalu. The run takes you through the local fish market and the newly built Likas Stadium. The smell of the morning's catch is as breathtaking as the wide open sea views. One also gets to enjoy the obligatory carbohydrate-loading pasta party the evening before.

In Singapore, the queues for runners' packs were perfectly formed and participants behaved in an orderly fashion. The event started at 5:30 a.m. I was snuggled up comfortably in the hotel until it was time to venture out to Rangoon Road. Any tourist to the Lion City must eat at Ng Ah Soi's Bah-Kut-Teh joint. This is reportedly the place that turned down our Chief Executive, Mr Tsang's request for a bowl of fragrant soup. That aside, they furnish you with your own stove to keep the kettle boiling and tea flowing. The aroma of the herbs was strangely refreshing and the ribs tender yet meaty. There was torrential rainfall and we were trapped but completely happy and satiated.

2. The infamous Bah-Kut-Teh

The Grand Palace in Bangkok is an amazing starting and finishing point. They have been organising the Standard Chartered Marathon here for 20 years. The full marathon started at an ungodly hour of 2 a.m. Crossing the two bridges across the Chao Phraya River was, I am told, an awesome experience. I can't say much for the terrifying ride in the river taxi though. The river was busy and the taxi was packed to the hilt with locals and tourists, no safety equipment in sight. One caution for runners though. Do avoid the lovely spicy food before the run. It makes runner's diarrhoea so much more prolific and unpredictable!

3. The Grand Palace, Bangkok

Frankly, I don't mind this running malarkey. It takes me places I've never been before and I get to savour firsthand the local customs and fare. Also there are hundreds of photos which I can share with friends on Facebook.

It's really painless too. I'm not the runner with the stiff muscles, refractory plantar fasciitis, aching ilio-tibial band or the upset stomach. In fact, I'm rather looking forward to accompanying the marathon runner in 2010. New York, Boston, Sydney, Berlin, anyone?

Driving Talk

Dr. HK MONG talking on the topic of Safe, Smart & Advanced Driving

Mr. Stephen HUNG talking on the topic of Non-compliance & Consequences

On Friday, 19 March 2010, the Federation was honoured to have invited Dr. HK MONG, Chairman of the Institute of Advanced Motorists Hong Kong and Mr. Stephen HUNG, Chairman of the Criminal Law and Procedure Committee of the Law Society of Hong Kong to deliver a talk on Safe, Smart & Advanced Driving and Non-compliance & Consequences. The talk was well attended by medical professionals and lawyers. The two speakers shared their precious experience with the participants on the topics and addressed their hot questions.

The talk finished with a lucky draw in which Lexus sponsored a pair of gentlemen and ladies watches, and National Australia Bank sponsored an iPod and 4 concert tickets. The Federation was also thankful to Lexus and National Australia Bank's sponsorship for a delicious refreshment at the event and souvenirs for the speakers and participants. Everyone had a fruitful evening with information attained from the talk and souvenirs from our sponsors.

We are delighted to announce that a Test Drive will be held in July to put the theories into practice.

The speakers at Q & A

The speakers and the Federation President, Dr. Raymond LO

Winners of the Lucky Draw

Rental Fees of Meeting Room and Facilities at The Federation of Medical Societies of Hong Kong

(Effective from October 2009)

Venue or Meeting Facilities	Member Society (Hourly Rate HK$)			Non-Member Society (Hourly Rate HK$)		
	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays
Multifunction Room I *(Max 15 persons)*	150.00	105.00	225.00	250.00	175.00	375.00
Council Chamber *(Max 20 persons)*	240.00	168.00	360.00	400.00	280.00	600.00
Lecture Hall *(Max 100 persons)*	300.00	210.00	450.00	500.00	350.00	750.00
Non-Peak Hour: 9.30 am - 5.30 pm Peak Hour: 5.30pm - 10.30pm						
LCD Projector	500.00 per session					
Microphone System	50.00 per hour, minimum 2 hours					

Word from the President

The Federation is dedicated towards enhancing the sharing and exchange between our member societies and health professionals. We shall be inviting our member societies to introduce their work and mission in our coming issues of the Medical Diary. The first society to introduce their profile in our Diary this year is our newly joined student member -- the Chinese University Medical Society. The young generation is the pillar of our profession in future. We shall continue to actively engage our new and existing members in Federation activities for professionals and the public.

On behalf of the Medical Society, the Chinese University of Hong Kong, I would like to express our pleasure in joining the FMSHK.

The Medical Society is a non-profitable student association founded in 1982. It is organised by a group of aspiring medical students, which serve about 700 members in the Faculty of Medicine.

Each year, we organise a wide range of events from academic, recreational to social. A charity event, the Medical Students' Festival, aims to raise fund for a beneficiary through holding different activities including a variety-show and a high table dinner among medical students. Different community health-check services are also coordinated in estate and different health-care centres throughout the year. Health Exhibition is an annual event, which aims at arousing the public's interest towards the importance of health. To extend our mission beyond Hong Kong, we also organise an international service trip and this year, we are heading to Vietnam to help the "Agent Orange" victims. All in all, our aim is to blend the doctors-in-training with community so that medical and health education can be directed to the public.

Joining the FMSHK, which is the umbrella organisation of medical associations, is not only a gain in connections with other medical professionals but also a chance for us to further extend our community service towards the public in a more organised way and a larger network. We hope that our interaction with FMSHK and other society members will yield a healthier Hong Kong.

Mr. Ashley Tsz-wai TSANG
President
Medical Society
The Chinese University of Hong Kong

News from Member Societies

The Hong Kong Society of Gastroenterology
Updated office-bearers for the year 2010-2011 are as follows: President: Prof. Benjamin WONG; Honorary Secretary: Dr. Judy Wai-chu HO; Honorary Treasurer: Dr. Wai-cheung LAO

The FMSHK would like to send its congratulations to the new office-bearers and look forward to working together with the societies.

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
						1
2	★ Patients with Severe Loin Pain and Septic Shock ★ HKMA Choir Rehearsal 3	★ FMSHK Officers' Meeting ★ 3rd Singing Course 4	★ HKMA - Wuhan Project "Practical Health Informatics Course for Doctors" (Session IV) ★ HKMA Yau Tsim Mong Community Network - The Impact of Post-prandial Hyperglycaemia in Diabetes and Cardiovascular Disease ★ MPS - Mastering Adverse Outcomes 5	★ HKMA Hong Kong East Community Network - Certificate Course: Practical Psychiatry for the General Practitioners (Session 7) ★ HKMA Council Meeting 6	★ Joint Surgical Symposium - To Cover the Uncovered in Plastic and Reconstructive Surgery ★ HKMA Kowloon East Community Network - Clinical Update Series on BPH and Diabetes Management: Optimizing Glycemic Control for T2DM 7	★ 3rd Table-Tennis Training Course ★ MPS - Mastering Your Risk 8
★ MPS - Mastering Adverse Outcomes 9	★ HKMA Choir Rehearsal 10	★ HKMA Kowloon West Community Network - Vaccination ★ 3rd Singing Course 11	★ Hong Kong Neurosurgical Society Monthly Academic Meeting Special Lecture - Central Nervous System Infections - A Microbiogical Perspective ★ HKMA Central, Western & Southern Community Network - "Certificate Course Management of Common Urological Problems for Primary Healthcare Providers" (II) 12	★ HKMA Hong Kong East Community Network - Certificate Course: Practical Psychiatry for the General Practitioners (Session 8) ★ MPS - Mastering Your Risk ★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2010 - Common Eye Diseases of Senior Citizens ★ 11th Regional Osteoporosis Conference - ISCD Bone Densitometry Courses and Certification Examinations 2010 & 1st ISCD Vertebral Fracture Assessment Course in Hong Kong 13	14	★ 3rd Table-Tennis Training Course ★ MPS - Mastering Your Risk ★ HKMA Kowloon East Community Network - Joint CME Course for Health Personnel 2010 on "Perinatal Psychiatry" ★ Refresher Course for Health Care Providers 2009/ 2010 15
★ MPS - Mastering Your Risk ★ HKMA Certificate Course on Family Medicine 2010 ★ HKMA Squash Tournament 16	★ HKMA Choir Rehearsal 17	★ FMSHK Executive Committee Meeting ★ 3rd Singing Course 18	★ MPS - Mastering Your Risk 19	20	21	★ 3rd Table-Tennis Training Course 22
★ MPS - Mastering Your Risk ★ 2010 Paediatric Update No.1 Common Childhood Sleep Disorders - an Update for Practising Paediatricians 23	★ HKMA Choir Rehearsal 24	★ MPS - Mastering Your Risk ★ HKMA Tai Po Community Network - Common Foot and Ankle Problem ★ 3rd Singing Course 25	26	★ HKMA New Territories West Community Network - Pain Management ★ Unusual Suspect & The Prince and the Beggar 27	28	★ MPS - Mastering Adverse Outcomes ★ 3rd Table-Tennis Training Course ★ HKCFP Annual Scientific Meeting 2010 "Meeting the Challenges of Non-Communicable Diseases" ★ HKMA Hong Kong East Community Network - PPI-CME Lecture 2010/2011 on "Emergency Medicine" 29
★ HKCFP Annual Scientific Meeting 2010 "Meeting the Challenges of Non-Communicable Diseases" ★ 2nd Seasonal Photo Sharing and Photo Competition ★ HKMA Tenpin Bowling Tournament ★ MPS - Mastering Your Risk 30	★ HKMA Choir Rehearsal 31					

Medical Diary of May

Date / Time	Function	Enquiry / Remarks
3 MON 7:30 pm - 8:30 pm	**Patients with Severe Loin Pain and Septic Shock** Organiser: Hong Kong Urological Association, Chairman: Dr. FU Kam Fung Kenneth, Speaker: Dr. YEUNG Hip Wo Victor, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. HUNG Hing Hoi / Ms. Tammy HUNG Tel: 2958 6006 / 9609 6064 Fax: 2958 6076 / 8344 5115 1 CME Point for College of Surgeons of Hong Kong
8:00 pm *(10, 17, 24, 31)*	**HKMA Choir Rehearsal** Organiser: The Hong Kong Medical Association, Venue: Rehearsal Hall, Sheung Wan Civic Centre	Ms. Candy YUEN Tel: 2527 8285
4 TUE 7:30 pm - 8:30 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. Paulina TANG Tel: 2527 8898 Fax: 2865 0345
8:00 pm *(11, 18, 25)*	**3rd Singing Course** Organiser: The Hong Kong Medical Association, Venue: 油麻地海燕音樂學院	Ms. Dora HO Tel: 2527 8285
5 WED 1:00 pm	**HKMA - Wuhan Project "Practical Health Informatics Course for Doctors" (Session IV)** Organiser: The Hong Kong Medical Association, Mr. Edmund TSE; Mr. Michael CHIU & Mr. Clifford TSE, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Carman WONG Tel: 2527 8285 1.5 CME Points
1:00 pm	**HKMA Yau Tsim Mong Community Network - The Impact of Post-prandial Hyperglycaemia in Diabetes and Cardiovascular Disease** Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr. CHAN Wing Bun, Venue: Shan Tung Room 2, 8/F., Langham Hotel, Mong Kok, Kowloon	Ms. Kitty LEE Tel: 2814 5100
(9, 29)	**MPS - Mastering Adverse Outcomes** Organiser: The Hong Kong Medical Association, Speakers: Dr. Justin CHENG & Dr. CHEUNG Kit Ying Andy, Venue: Mongkok or 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 for Participant 4 CME Points for Speaker
6 THU 1:00 pm *(13)*	**HKMA Hong Kong East Community Network - Certificate Course: Practical Psychiatry for the General Practitioners (Session 7)** Organiser: HKMA Hong Kong East Community Network, Hong Kong Society of Biological Psychiatry and Lundbeck Institute Hong Kong, Chairman: Dr. SHUM Ping Siu, Speakers: Various, Venue: Regus Conference Centre, 35/F., Central Plaza, 18 Harbour Road, Wanchai, Hong Kong	Ms. Jaclyn LEE / Ms. Carrie CHEUNG Tel: 2877 1106 1.5 CME Points for Participant 3 CME Points for Speaker
8:00 pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. HH TSE, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Christine WONG Tel: 2527 8285
7 FRI 8:00 am - 9:00 am	**Joint Surgical Symposium - To Cover the Uncovered in Plastic and Reconstructive Surgery** Organiser: Department of Surgery, The University of Hong Kong & Hong Kong Sanatorium & Hospital, Chairman: Prof. William WEI, Speakers: Dr. CHUNG Hon-Ping, Dr. LIU Hin-Lun, Venue: Hong Kong Sanatorium & Hospital	Department of Surgery, Hong Kong Sanatorium & Hospital Tel: 2835 8698 Fax: 2892 7511 1 CME Point (Active)
1:00 pm	**HKMA Kowloon East Community Network - Clinical Update Series on BPH and Diabetes Management: Optimizing Glycemic Control for T2DM** Organiser: HKMA Kowloon East Community Network, Speaker: Dr. CHAN Wing Bun, Venue: Kwun Tong	Miss Alice TANG / Miss Carman WONG Tel: 2527 8285 1 CME Point for Participant 2 CME Points for Speaker
8 SAT 4:00 pm *(15, 22, 29)*	**3rd Table-Tennis Training Course** Organiser: The Hong Kong Medical Association	Ms. Dora HO Tel: 2527 8285
2:30 pm *(13, 15, 16, 19, 23, 25, 30)*	**MPS - Mastering Your Risk** Organiser: The Hong Kong Medical Association, Speakers: Dr. CHEUNG Kit Ying Andy & Dr. Justin CHENG, Venue: Mongkok or 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 for Participant 4 CME Points for Speaker
11 TUE 1:00 pm	**HKMA Kowloon West Community Network - Vaccination** Organiser: HKMA Kowloon West Community Network, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Carman WONG Tel: 2527 8285
12 WED 7:30 am	**Hong Kong Neurosurgical Society Monthly Academic Meeting Special Lecture - Central Nervous System Infections - A Microbiogical Perspective** Organiser: HK Neurosurgical Society, Speaker: Dr. Samson S.Y. WONG, Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Y.C. PO Tel: 2990 3788 Fax: 2990 3789 2 CME Points for College of Surgeons of Hong Kong
1:00 pm	**HKMA Central, Western & Southern Community Network -"Certificate Course Management of Common Urological Problems for Primary Healthcare Providers" (II)** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. LAM Ming Yuen, Speaker: Dr. WONG Bok Wai Byron, 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 / Miss Carman WONG Tel: 2527 8285
13 THU *(14, 15, 16)*	**11th Regional Osteoporosis Conference - ISCD Bone Densitometry Courses and Certification Examinations 2010 & 1st ISCD Vertebral Fracture Assessment Course in Hong Kong** Venue: Novotel Century Hotel Hong Kong and Hong Kong Convention & Exhibition Centre	ROC 2010 Conference Secretariat, c/o International Conference Consultants, Ltd., Tel: (852) 2559 9973 Fax: (852) 2547 9528 Email: roc2010@icc.com.hk, Websites: www.oshk.org.hk / www.hkgerisoc.org
15 SAT 1:30 pm	**HKMA Kowloon East Community Network - Joint CME Course for Health Personnel 2010 on "Perinatal Psychiatry"** Organiser: HKMA Kowloon East Community Network; Hong Kong College of Family Physicans & United Christian Hospital, Chairman: Dr. AU Ka Kui Gary, Speaker: Dr. May MIAO, Venue: Lecture Theatre, G/F, Block F, United Christian Hospital, Kowloon	Ms. Gary WONG Tel: 3513 4821

Date / Time	Function	Enquiry / Remarks
15 SAT 2:30 pm	**Refresher Course for Health Care Providers 2009/ 2010** Organiser: The Hong Kong Medical Association and Our Lady of Maryknoll Hospital, Speaker: Dr. NG Kin Chung, Venue: Training Room II, 1/F., OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon, Hong Kong	Ms. Clara TSANG Tel: 2354 2440 2 CME Points for Participant 4 CME Points for Speaker
16 SUN 2:00 pm	**HKMA Certificate Course on Family Medicine 2010** Organiser: The Hong Kong Medical Association, Speakers: Dr. CHEUNG Kit Ying Andy & Dr. MAK Ki Yan, Venue: Queen Elizabeth Hospital, Kowloon	Miss Viviane LAM Tel: 2527 8452 3 CME Points for Participant and Speaker
2:00 pm	**HKMA Squash Tournament** Organiser: The Hong Kong Medical Association, Venue: Kowloon Cricket Club	Ms. Dora HO Tel: 2527 8285
18 TUE 8:00 pm - 10:00 pm	**FMSHK Executive Committee 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. Paulina TANG Tel: 2527 8898 Fax: 2865 0345
23 SUN	**2010 Paediatric Update No.1 Common Childhood Sleep Disorders - an Update for Practising Paediatricians** Organiser: Hong Kong College of Paediatricians, Speakers: Dr. Daniel KK NG & Dr. June Sin Hang CHAN and Prof. Yun Kwok WING, Venue: Hospital Authority Head Office Lecture Theatre	Ms. Vanessa WONG Tel: 2871 8871 Fax: 2785 1850 3 CME points Category A (Hong Kong College of Paediatricians)
25 TUE 1:30 pm	**HKMA Tai Po Community Network - Common Foot and Ankle Problem** Organiser: HKMA Tai Po Community Network, Speaker: Dr. CHAN Tun Kut, Venue: Tai Po	Mr. Roget LAW Tel: 2811 9711 1.5 CME Points for Participant 3 CME Points for Speaker
27 THU 1:00 pm	**HKMA New Territories West Community Network - Pain Management** Organiser: HKMA New Territories West Community Network, Chairman: Dr. YAN Kam Sun Charlie, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F, Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long, New Territories	Miss Alice TANG Tel: 2527 8285
6:30 pm - 8:00 pm	**Unusual Suspect & The Prince and the Beggar** Organiser: Hong Kong Thoracic Society / ACCP(HK & Macau Chapter), Chairpersons: Dr. LO Ho Yin & Dr. LAU Kam Shing, Speakers: Dr. TONG Chun Wai & Dr. Raymond TSO, Venue: LG1, Lecture Room, Ruttonjee Hospital, Hong Kong	Dr. James C.M. HO / Dr. Johnny W.M. CHAN Tel: 2255 4999 Fax: 2872 5828
29 SAT *(30)*	**HKCFP Annual Scientific Meeting 2010 "Meeting the Challenges of Non-communicable Diseases"** Organiser: The Hong Kong College of Family Physicians, Chairman: Dr. CHEUNG Man Kuen, Speakers: Various, Venue: HKAM Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong	Ms. Teresa LIU / Mr. Patrick WU Tel: 2528 6618 Fax: 2866 0616 Email: asm2010@hkcfp.org.hk
1:30 pm	**HKMA Hong Kong East Community Network - PPI-CME Lecture 2010/2011 on "Emergency Medicine"** Organiser: HKMA Hong Kong East Community Network & HA Hong Kong East Cluster, Speakers: Dr. WONG Tai Wai & Dr. LI Keung, Venue: HKEC Training Centre for Healthcare Management & Clinical Technology, Pamela Youde Nethersole Eastern Hospital, Hong Kong	Miss Alice TANG / Miss KWONG WL Tel: 2527 8285/ 2595 6941
30 SUN 2:00 pm	**2nd Seasonal Photo Sharing and Photo Competition** Organiser: The Hong Kong Medical Association, Venue: HKMA Head Office, 5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Dora HO Tel: 2527 8285
2:00 pm	**HKMA Tenpin Bowling Tournament** Organiser: The Hong Kong Medical Association, Venue: South China Athletic Association	Ms. Dora HO Tel: 2527 8285

Meetings

20/6/2010	**Annual Scientific Meeting 2010** Organiser: Hong Kong Society of Dermatology and Venerology, Enquiry: Ms. Chloe WONG, Tel: 2155 8557 / 2116 4348, Fax: 2559 6910, Email: meeting.hk@asia.cmpmedica.com
10/7/2010	**Hong Kong Surgical Forum - Summer 2010** Organisers: 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: 2255 4885 / 2255 4886, Fax: 2819 3416, E-mail: hksf@hku.hk, Web-site: http://www3.hku.hk/surgery/forum.php

Answer to Radiology Quiz

Answer:

Chest X-Ray Findings:
Cardiac outline is enlarged. Upper zone blood vessels are dilated. Horizontal fissure is slightly thickened. Right costophrenic angle is blunted.

The above findings are compatible with congestive cardiac failure.
However, an opacity is seen at right lower zone.

Further Investigation:
Lateral CXR which shows a lenticular opacity at the inferior part of the oblique fissure.

Diagnosis is **loculated effusion at oblique fissure.**

Discussion:

Fluid may become loculated in the interlobar fissure. It is not common but may occur in cardiac failure. It may be mistaken as a lung tumour in the frontal projection. A lateral view will usually avoid this mistake. This type of loculated effusion may disappear rapidly on treatment and this will confirm the diagnosis avoiding any unnecessary investigations. These loculated effusions are therefore also known as 'pseudo-' or 'vanishing' tumours.

Dr. WK TSO

Chief of Service, Department of Radiology, Queen Mary Hospital

MSD

Scientific Update in the LANCET!!

Clinical data in women aged 24 - 45 was first published in the LANCET[1] and is now included in product circular of GARDASIL®[2,]*

Help reduce the burden of the following diseases:

- **Cervical Cancer**
- **Vulvar Cancer**
- **Vaginal Cancer**
- **Genital Warts**

caused by HPV 6, 11, 16, 18

* See Clinical data (efficacy and safety) in Product Circular Section XIII. CLINICAL PHARMACOLOGY and XV. SIDE EFFECTS

Selected Safety Information:
GARDASIL® [Quadrivalent HPV (Types 6, 11, 16, 18) Recombinant Vaccine] is indicated in 9-26 years old girls and women for the prevention of cervical, vulvar, and vaginal cancers; precancerous or dysplastic lesions and genital warts caused by HPV Types 6, 11, 16 and 18. It should be administered intramuscularly as 3 separate doses at 0, 2nd, 6th month. It is contraindicated in individuals with hypersensitivity to any vaccine ingredients or after a previous dose of GARDASIL and is not recommended for pregnant women. Pregnancy should be avoided during the vaccination period. This vaccine will not protect against diseases that are not caused by HVP and is not intended to be used for treatment of active genital warts; cervical, vulvar, or vaginal cancers; CIN, VIN, or VaIN. Routine cervical screening should be continued. Common adverse reaction in clinical trials were fever, injection-site pain, swelling, erythema, pruritus & bruising which were mild to moderate. Post-marketing reports: dizziness, headache, syncope, nausea & vomiting. **Before prescribing, please consult the full prescribing information.**

References:
1. N. Munoz MD, J Luna MD et al. Lancet 2009; 373:1949-57. 2. Product Circular (GARDASIL, MSD)

Merck Sharp & Dohme (Asia) Ltd.
27/F., Caroline Centre, Lee Gardens Two, 28 Yun Ping Road, Causeway Bay, Hong Kong
Tel: (852) 2574 4241 Fax: (852) 2834 0756

03-2012-GRD-10-HK-1008-J