This month

■ **Spotlight**
  Psychiatric and neurological paraneoplastic syndromes

■ **Cardiology**
  A 70-year-old gentleman with long-standing congestive heart failure

■ **Dermatology**
  A 46-year-old gentleman with itchy nodules on the scrotum and inner thigh

■ **Gastrointestinal Medicine**
  A 25-year-old male with complaints of bloody diarrhoea

■ **General Medicine**
  A 49-year-old female with complaints about losing her hair.

■ **Dermatology Review**
  Pathophysiology and treatment of atopic dermatitis
CME COURSES

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Answer Sheet

The Hong Kong Medical Association is dedicated to providing a coordinated CME programme for all members of the medical profession. Under the HKMA CME Programme, a CME registration process has been created to document the CME efforts of doctors and to provide special CME avenues. The Association strives to foster a vibrant environment of CME throughout the medical profession. Both members as well as non-members of the Association are welcome to join us. You may contact the HKMA Secretariat for details of the programme.

HKMA CME Bulletin – MONTHLY SELF-STUDY SERIES to help you grow!
Please read the following articles and answer the questions. Participants in the HKMA CME Programme will be awarded credit points under the Programme for returning the completed answer sheet via fax (28650943) or by mail to the HKMA Secretariat on or before 15 February, 2011. Answers to questions will be provided in the next issue of the HKMA CME Bulletin. (Questions may also be answered online at www.hkmacme.org)

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EDITORIAL

Happy New Year! I wish everyone happy learning in the new year.

Although learning is not for CME points, we do have to care about our CME points. Did you earn more than 30 points in 2010? We sent out letters in October to remind doctors who had not gained enough CME points. Did you find it useful? At the beginning of a new year, it is again time to plan your CME activities. Of course, one of the main sources of CME points is from this *CME Bulletin*, or from answering the monthly CME questions on-line. However, please be reminded again that the number of CME points from self-study is capped at 20. So it is a must to attend some seminars to gain enough points to be CME-certified. Don’t think that this “qualification” is not very useful and CME has not been made compulsory. The about-to-come Primary Care Registry is going to use CME as a major criterion for entrance into, and staying in the Registry. So it is better for us to get prepared.

To plan CME activities for a whole year, you will find the HKMA CME diary very useful. You can refer to your records in the diary from last year and then select topics that you are interested in, or that fulfill your needs. Interest and needs are different. An example is the featured article of this issue. It is about paraneoplastic topics. It is very interesting, but might not fulfill the needs of a family doctor in his daily practice.

The HKMA Community Networks have been organizing seminars and workshops which provide valuable opportunities for local doctors to meet regularly. They are also good sources of CME points. Two other occasions for regular CME seminars are the MPS lectures (which are held twice a year) and the Beijing-Hong Kong Medical Exchange (BHME). This year’s BHME will take part in Wenzhou, China, and the topic is ophthalmology. You can take this chance to visit the place famous for merchants.

Another source of regular seminars is the Certificate Course on Family Medicine organized by the HKMA and CUHK. This is the third year that the course has been held. You can always join in any time. This year, interesting topics include: Hepatitis B, Geriatric Giants, Influenza and Pneumococcal Vaccines, DM and Insulin, Convulsions, ADHD, Behavioural Changes, Problems of the Hands and Feet, Counselling in Primary Care, Drug Abuse, Diagnostic Imaging, Laboratory Tests, Colorectal Cancers, and Mindfulness.

Dr. CHENG Chi Man
Co-Chairman, CME Committee
Paraneoplastic syndromes (PNS) are symptoms associated with the occurrence or possible occurrence of a cancer. They may be caused by:
1. antibodies directed against tumours that cross-react with other body tissues (Figure 1); or
2. the systemic effects of substances secreted ectopically by a tumour (See note below).*

They are not caused by direct local destructive or mass effects of the tumour, metastasis, malnutrition, infections, side effects of treatment or the usual metabolic or endocrine effects of substances normally secreted eutopically by tissues from which the tumour originates [1,2].

The syndrome may develop before the tumour becomes overt, at the same time when the tumour manifests itself, or may herald the recurrence of a dormant tumour. Such cancers are usually indolent and metastasize slowly.

PNS may occur in different body systems. Examples are listed in Table 1.

Psychiatric and neurological PNS are often severe, rapidly progressive and disabling. They may precede the discovery of the cancer by a few months to a few years. In some patients, no tumours are found on long-term follow up [4-8].

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Table 1. PNS occurring in different body systems (estimated incidence 20%).

<table>
<thead>
<tr>
<th>Body system</th>
<th>PNS</th>
<th>Associated cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Acanthosis nigricans</td>
<td>Gastrointestinal cancer</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Cushing’s syndrome (ectopic production of ACTH)</td>
<td>Small-cell lung cancer (SCLC)</td>
</tr>
<tr>
<td>Haematological</td>
<td>Thrombocytosis</td>
<td>Breast, lung</td>
</tr>
<tr>
<td>Renal</td>
<td>Membranous glomerulonephritis</td>
<td>Lymphoma, colon, ovary</td>
</tr>
<tr>
<td>Rheumatological</td>
<td>Hypertrophic osteoarthropathy</td>
<td>Lung cancer</td>
</tr>
</tbody>
</table>

*Note: Point 2 above is often poorly explained in most papers. Some authors maintain that the distant effects of all chemicals secreted by the tumour are considered PNS. This would include Cushing’s syndrome caused by an adrenal tumour, or hypoglycaemia caused by beta-cell tumours of the pancreas. However, such direct effects of substances secreted eutopically should not be considered PNS. Other authors exclude the effects of all chemicals secreted by the tumour. However, Cushing’s syndrome caused by ectopic secretions, such as ACTH-like substances from lung cancers or thyrotoxicosis caused by thyroid hormone from a teratoma should be considered PNS [3].
Specific forms of psychiatric and neurological PNS are associated with certain cancers (Table 2).

**Myasthenia Gravis (MG):** A thymoma can be found in 10–15% of patients. Acetylcholine receptors, which are situated postsynaptically on the muscle fibre endplate, are blocked and destroyed by circulating antibodies. Patients present with ptosis, extraocular muscle weakness, dysarthria, dysphagia and respiratory muscle weakness. Rapid repetitive nerve stimulation (RNNS) produces a decremental response. Treatment with acetylcholinesterase inhibitors, e.g. pyridostigmine (Mestinon), is indicated.

**LEMS:** Seventy percent of cases are associated with SCLC. About 3% of patients with SCLC develop LEMS. Antibodies to voltage-gated calcium channels are found in the blood, with these channels being located presynaptically on the nerve ending of the motor endplate. The opening of the calcium channel is required for the release of acetylcholine into the synapse, but blocking of the channel results in failure of acetylcholine release. Patients present with lower-limb girdle weakness and ataxia. There may also be weakness of the upper-limb girdle. RNNS will give an incremental response. Treatment is by diaminopyridine, which overcomes the blockage of acetylcholine release (Figure 2 and Table 3).

**Limbic encephalitis:** The limbic system is a collection of structures and circuits in the brain. The main components include the cingulate gyrus, the parahippocampal gyrus, the hippocampus, amygdala and hypothalamus. It regulates emotion, memory and cognitive function (Figure 3). Limbic encephalitis is characterized by short-term memory loss, confusion, hallucinations, seizures, and mood disorders, including anxiety and depression. Disturbance of hypothalamic function may result in somnolence, hyperthermia, and endocrine abnormalities. Fifty percent of patients suffer from a neoplasm, usually SCLC. Some patients may have testicular tumours [9,10].

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Table 2. Psychiatric and neurological PNS (estimated incidence <1%) and associated cancers.

<table>
<thead>
<tr>
<th>PNS</th>
<th>Associated cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>MG</td>
<td>Thymoma</td>
</tr>
<tr>
<td>Lambert-Eaton myasthenic syndrome (LEMS)</td>
<td>SCLC</td>
</tr>
<tr>
<td>Limbic encephalitis</td>
<td>SCLC, testicular tumour</td>
</tr>
<tr>
<td>Encephalomyelitis</td>
<td>SCLC, others</td>
</tr>
<tr>
<td>Cerebellar degeneration</td>
<td>SCLC, ovary, breast, Hodgkin’s, others</td>
</tr>
<tr>
<td>Sensory neuronopathy</td>
<td>SCLC</td>
</tr>
<tr>
<td>Motor neuronopathy</td>
<td>Lymphoma</td>
</tr>
<tr>
<td>Opsomyoclonus</td>
<td>Neuroblastoma, breast, lung</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>SCLC, melanoma</td>
</tr>
<tr>
<td>Stiff person syndrome</td>
<td>Breast</td>
</tr>
</tbody>
</table>

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Figure 2. Normal neuromuscular junction function and impaired function in MG and LEMS.
Encephalomyelitis: The areas affected include the temporal lobes, the limbic system, brain stem, cerebellum, spinal cord, dorsal root ganglion and the autonomic system. Symptoms depend on the area most seriously affected; for example, a patient with predominant brain-stem encephalitis will present with diplopia, dysarthria, dysphagia, facial numbness and hearing loss. The most often encountered cancer is SCLC, but many other kinds of cancer are possible.

Cerebellar degeneration: Patients present with nausea, vomiting, dizziness and unsteady gait. The symptoms progress over weeks to months, and patients often become severely debilitated with dysphagia, dysarthria and ataxia. Fifty percent of cases have a cancer, usually SCLC, ovarian and breast cancers and Hodgkin’s lymphoma.

Sensory neuronopathy: The upper limbs are usually affected first. There is pain, paraesthesia, numbness and clumsiness, progressing to unsteady gait. There may be sensory loss in the face, chest and abdomen. The onset is subacute with rapid deterioration over weeks to months. Nerve conduction tests show marked reduction in sensory nerve action potential. Twenty percent of cases are associated with cancer, usually SCLC, gynaecological or Hodgkin’s lymphoma [11].

Motor neuronopathy: This is a rare syndrome. There is degeneration of the anterior horn cells, and patients present with lower motor neuron weakness of the upper and lower limbs. It is often associated with Hodgkin’s lymphoma.

Opsomyoclonus: Opsoclonus is the involuntary, arrhythmic, high-amplitude conjugate saccades of the eyes in horizontal and vertical directions. In the adult this is associated with myoclonus – jerky movements of the head, trunk or extremities. Patients suffer from SCLC or gynaecological cancers. Fifty percent of children with opsomyoclonus have an underlying neuroblastoma.

Retinopathy: In cancer-associated retinopathy (CAR), there is bilateral dysfunction of the cones and rods. Patients complain of photosensitivity and reduced visual acuity. There is decreased colour perception, ring scotoma, night blindness and prolonged dark adaptation. The underlying tumour is usually SCLC. In melanoma-associated retinopathy (MAR), rods are preferentially affected. Patients suffer from peripheral visual loss and night blindness. They may complain of sudden shimmering, flickering lights. The condition is associated with malignant melanoma.

Stiff person syndrome: The patients suffer from painful spasms due to co-contraction of agonist and antagonist muscles. The stiffness mainly affects axial muscles. Associated cancers are breast, colon, lung, thymoma and Hodgkin’s lymphoma.

Other syndromes include sensory motor peripheral neuropathy, autonomic neuropathy, myelitis and optic neuritis.

The above syndromes are associated with the emergence in the blood and cerebro-spinal fluid (CSF) of a number of onconeural antibodies, i.e. antibodies induced by cancer antigens that cross-react with healthy neural tissues. The well-characterized onconeural antibodies are listed in Table 4.
For patients who are smokers, aged over 50 and anti-Hu positive, the usual course of action is to proceed to chemotherapy even in the absence of a detectable tumour (Figure 4). Female patients positive for anti-Yo are associated with a very high risk of breast or gynaecological cancers. For such patients, if no cancer is found in the breast or reproductive organs, a radical gynaecological operation is indicated (Figure 5).

In a series of 200 patients from Barcelona, Spain, suffering from encephalomyelitis and positive for anti-Hu antibodies, a tumour was found in 83.5% of cases, 90% of which were SCLC. No tumours were found in 16.5%. This included patients who died of other causes or were lost to follow up. Very small tumours might also be missed in autopsies. Only 2% of cases did not develop a tumour after 5 years of follow up [12]. Similar results are obtained with other series [13].

A patient with encephalitis and anti-NMDA (N-methyl-D-aspartate) receptor antibodies was reported in the August 2010 issue of the Hong Kong Medical Journal. A 21-year-old female presented with fever and headache for 10 days, and was admitted to Ruttonjee Hospital in July 2008. Following admission, the patient became mentally confused and agitated. She developed tonic-clonic convolution, tardive dyskinesia, tremors, and choreoathetotic movements. After extensive workup for infective, autoimmune, metabolic and other causes of encephalitis, a 1.3-cm teratoma was found in the left ovary. The CSF tested positive for anti-NMDA antibodies. The teratoma was removed in October 2009. The patient suffered from residual orofacial dyskinesia, dysarthria, cognitive impairment and paraplegia. Most patients with anti-NMDA receptor encephalitis present to the psychiatrist initially [14].

In patients suspected to have paraneoplastic neurological disorders (PND), a comprehensive diagnostic workup should be performed to detect the underlying tumour. The specific clinical syndrome and the onconeural antibody detected may give some clues to the type of cancer to be looked for. If no tumour is found initially, PET-CT scanning is indicated.

In a recent series of 56 patients with clinically suspected PND reported by the Mayo Clinic, Rochester, USA, patients underwent PET-CT scanning after initial investigations, including endoscopies and routine CT scanning, were negative. Thirty-nine percent reported abnormal scans and 18% were histologically confirmed to suffer from cancer [15]. Other studies report a yield of about 10% [16]. If no cancer is found after all investigations are exhausted, a repeat of the workup is indicated in 6 months. The period of follow up lasts for about 4 years.

The Paraneplastic Neurological Syndrome Euronetwork (2004) proposed the following diagnostic criteria [17]:

**Definite PND:**
1. Classic syndrome with cancer diagnosed within 5 years of neurological symptom development.
2. Non-classic syndrome that resolves or significantly improves after cancer treatment.
3. Non-classic syndrome with cancer diagnosed within 5 years of neurological symptom development and positive anti-neuronal antibodies.
4. Neurological syndrome (classic or not) without cancer and with well-characterized anti-neuronal antibodies.

**Possible PND:**
2. Neurological syndrome (classic or not) without cancer and with partially characterized anti-neuronal antibodies.
3. Non-classic syndrome with cancer diagnosed within 2 years of neurological syndrome development, without anti-neuronal antibodies.

Treatment is directed primarily at the underlying tumour. This may alleviate symptoms of the paraneoplastic syndrome. Plasmapheresis, intravenous immunoglobulins and steroids may be useful in some patients. Others will require more potent immunosuppressants such as cyclophosphamide, tacrolimus, cyclosporine and rituzimab.

References

A 70-year-old gentleman with long-standing congestive heart failure

A 70-year-old gentleman has long-standing congestive heart failure due to coronary heart disease and old myocardial infarction. His left ventricular ejection fraction is about 28%. He is in NYHA functional class III and is in sinus rhythm of about 100/min.

QUESTIONS

1. The prevalence of aspirin resistance is about ___________.
   a. 1%
   b. 10%
   c. 25%

2. Which of the following statement(s) is/are true?
   The cause(s) of aspirin resistance is/are:
   a. Noncompliance
   b. Ibuprofen
   c. Chronic infection

3. Which of the following statement(s) is/are true?
   When compared with aspirin-sensitive patients, patients with aspirin resistance are at higher risk of:
   a. Death
   b. Acute coronary syndrome
   c. Cerebrovascular events

ANSWERS

1. c  2. a,b,c  3. a,b,c

Aspirin binds to the COX-1 enzyme and prevents the conversion of arachidonic acid to thromboxane A2, which results in platelet activation. However, recent advances in platelet biology have shown that considerable individual variability occurs.

Aspirin resistance is defined as the inability of aspirin to reduce platelet production of thromboxane A2, and thereby platelet activation and aggregation, which is a laboratory phenomenon. It has been generally defined as ≥20% platelet aggregation when using 0.5–1.0 mg/mL arachidonic acid as the agonist.

Depending on the population studied, the assay used and the definition applied, prevalence of aspirin resistance varies. A meta-analysis showed that among 2,930 patients with cardiovascular disease taking aspirin (ranging from 75–325 mg daily), 810 patients (28%) were classified as aspirin resistant [1].

Aspirin resistance has clinical consequences. Patients with aspirin resistance are more likely to have major cardiovascular events. The same meta-analysis reported an odds ratio of 3.85 for cardiovascular events, 5.99 for deaths, 4.06 for acute coronary syndromes, and 3.78 for cerebrovascular events for aspirin-resistant patients compared to aspirin-sensitive patients [1].

The mechanisms that are behind incomplete platelet response to aspirin include:
   a. Noncompliance.
   b. Inadequate dosing with various aspirin formulas, e.g. enteric-coated preparations.
   c. Drug interactions, e.g. ibuprofen.
   d. Enhanced platelet turnover, e.g. post coronary artery bypass graft surgery, chronic infection or inflammation.
   e. Genetic factors, e.g. genetic polymorphisms of COX-1.

Recent developments in the evaluation of platelet inhibition and the introduction of the point-of-care platelet function machinery make assessing the degree of platelet response more accessible and easier to perform.

Identification of aspirin resistance has clinical implications. Drug compliance and drug interactions should be checked. Proper forms of aspirin should be prescribed. Any concomitant infection or inflammation should be treated.

The management of patients who develop adverse vascular events despite being on aspirin remains challenging. More studies are needed to determine if laboratory assays to detect aspirin resistance should guide therapy.

The choice of increasing aspirin dosage or adding alternative anti-platelet agents should be based on individual cases, e.g. adding extended-release dipyridamole or substituting aspirin with clopidogrel would be a logical alternative to patients who develop stroke despite using aspirin. Another example is adding clopidogrel to patients who develop acute coronary syndrome while already taking daily aspirin.

Reference

A 46-year-old gentleman with itchy nodules on the scrotum and inner thigh

A 46-year-old gentleman was seen in the sexually transmitted diseases (STD) clinic because of enlarging papules over the inner thigh and scrotum. He had been well until twelve months earlier, when he noticed diffuse, itchy, non-tender nodules on his medial thigh. Four weeks later, he noticed several new nodules on the scrotum. He recalled venereal exposure with a prostitute weeks before his first visit to the clinic.

Q&A
Please answer ALL questions
1. What is the diagnosis?
2. What are the differential diagnoses?
3. What are the investigations?
4. What are the treatments?

DECEMBER ANSWERS

QUESTIONS
1. What is the diagnosis?
2. What are the differential diagnoses?
3. What are the usual presenting symptoms?
4. What are the associated factors?
5. What are the treatments?

ANSWERS
1. The clinical diagnosis is knuckle pads. They are benign, asymptomatic, well-defined, smooth, firm, skin-coloured lesions measuring 0.5–3 cm in diameter, over dorsal aspects of the metacarpophalangeal and interphalangeal joints. It is a common condition.
2. Differential diagnoses include fibromas, picker’s nodules, xanthomas, warts, Herberden’s nodule and rheumatoid nodules.
3. Knuckle pads are usually asymptomatic. Occasionally there may be pain or difficulty with hand functioning. Patients usually seek medical advice because of cosmetic issues.
4. They may be related to repetitive trauma associated with sporting activities or occupational activities. Patients with Dupuytren’s contracture are more likely to have knuckle pads.
5. There are no established, generally successful treatments. Patients should avoid repetitive trauma to the lesion. Wearing gloves or changing occupation may be considered. Application of topical steroids or keratolytics should be tried. Intralesional steroid injections may reduce the size of the lesions. However, knuckle pads persist indefinitely, with little change in most cases.
A 25-year-old male with complaints of bloody diarrhoea

A 25-year-old man presented to the office with complaints of bloody diarrhoea. He had intermittent episodes of diarrhoea with large amounts of mucus and some blood for several weeks. The patient described the blood as being small amounts streaked on the stool and pink mucus. He stated that he had the diarrhoea for several days and then was asymptomatic for a day or two. The frequency was 1–2 episodes per day, usually after eating. The man initially thought the bleeding might be from a haemorrhoid, but he decided to come in for evaluation when the bleeding and diarrhoea continued. He had not been incontinent, and he denied nausea and vomiting. He also said he had not felt weak or light-headed.

The patient denied travel, exposure to well water, fever, joint pain, recent antibiotic therapy, and chronic medical problems. His only prior problem with diarrhoea occurred several years earlier. At that time, he had diarrhoea and vomiting for 3 days after exposure to a friend with similar symptoms. He had no family history of chronic bowel diseases.

On examination, the patient was afebrile. His blood pressure was 126/72 mmHg, and his pulse was 68 bpm. He did not have symptoms of orthostatic hypotension. The man weighed 73 kg, and his height was 178 cm. He appeared comfortable. Results of the examination were normal except for mild tenderness in the lower left quadrant of his abdomen and mild tenderness at the anal verge on rectal examination.

Laboratory findings included a guaiac-positive stool, normal electrolyte levels, and no evidence of abnormalities consistent with a diagnosis of anaemia. Results were pending regarding serum iron, total iron binding capacity, serum albumin, liver enzymes, and stool studies.

Infectious causes of diarrhoea should be considered, particularly with a relatively acute history of the condition. Viral gastroenteritis is unlikely, as it tends to be of shorter duration and is not usually associated with bleeding (unless haemorrhoids or an anal fissure exists). Salmonella, Shigella, Campylobacter, Clostridium difficile, Yersinia, and toxigenic Escherichia coli (0157:H7) are bacterial causes that should be considered. Salmonella infection may occur after ingestion of contaminated chicken or eggs. Clostridium infection often follows antibiotic treatment but not always. Amebiasis, giardiasis, gonococcal or chlamydial proctitis, and infection by Cytomegalovirus, herpes simplex virus, or Mycobacterium should also be considered. Salmonella infection may often occur after ingestion of contaminated chicken or eggs. Clostridium infection often follows antibiotic treatment but not always.

Noninfectious causes of diarrhoea include celiac sprue, side effects of medications (e.g. salicylates, nonsteroidal anti-inflammatory drugs), irritable bowel syndrome, malignancy, ischaemic bowel disease, diverticulitis, endometriosis, radiation enteritis (in patients with prior radiation therapy for abdominal malignancy), collagenous colitis, Crohn’s colitis, lymphocytic colitis, and ulcerative colitis (UC). As is the case for infectious causes, further studies are needed to differentiate between these potential causes of the diarrhoea.

In this patient, the results of stool studies did not identify an infectious aetiology, and sigmoidoscopy and biopsy results showed rectal and sigmoidal inflammation consistent with UC. The results of the remaining blood tests were normal.

Abnormal findings are most severe in the rectum and then extend proximally in varying degrees. Approximately 40–50% of affected patients have only rectal and rectosigmoid disease; another 30–40% of patients have disease that extends beyond the sigmoid region but does not affect the entire colon. About 20% of patients have involvement of the entire colon. The disease does not skip areas, although rarely, inflammation of the appendix or cecum may be associated with subtotal disease.

Early in the inflammatory process, the colonic mucosa is hyperaemic and oedematous, resulting in a granular appearance. As the inflammation worsens, haemorrhage and small ulcers develop. These ulcers can progress to deep ulcers that may be irregular and may extend linearly. Pseudopolyps may develop in the colon as a result of irregular epithelial regeneration. In advanced disease, the mucosa becomes atrophic, with shortening of the colon and loss of haustra.

Histologically, the inflammation occurs in the mucosa, with infiltration of plasma cells, neutrophils, eosinophils, lymphocytes, macrophages, and mast cells. Early in the disease, particularly in mild cases, the mucosal appearance will return to normal during remissions. As the disease becomes chronic, changes in the structure of the mucosal crypts and irregularities in the mucosal surface occur. Chronic inflammatory changes can also be found.

The exact cause of UC is unknown. Various hypotheses have been put forth on the basis of clinical and pathological findings associated with UC. Possible causes include infection, autoimmune responses, immune responses to bowel flora, abnormal epithelial cells, allergy to food, and emotional/psychosomatic disease. No specific infecting organism has been identified, although it appears that pathogenic bacteria may have some role in the disease process. In addition, evidence exists of a genetic component to the disease. First-degree relatives and monozygotic twins of affected persons have a higher risk of UC.
Because UC may occur concurrently with autoimmune diseases such as thyroiditis, it is possible that an autoimmune process is responsible for the disease. This theory is supported by findings of auto-antibodies to colonic epithelial cells and responses to treatment with drugs that suppress the immune system.

Although it has been suggested that milk allergy may contribute to the disease process, this association has not been proved. Psychosomatic causes also have not been proved; however, patients with chronic disease may have psychiatric symptoms, such as depression and anxiety, because of the effects of the disease (e.g. chronic diarrhoea, chronic pain, faecal incontinence).

An interesting negative association is cigarette smoking. Although smokers are at greater risk for Crohn’s disease, they appear to be less at risk for UC. The reason for this is unclear, but it is known that smoking can decrease mucosal permeability in the colon, and this may be a factor.

The most common symptoms are diarrhoea, passage of mucus, rectal bleeding, and abdominal pain. The severity of the symptoms usually correlates with the severity of the disease, although some patients may be asymptomatic. The symptoms typically have a slow, insidious onset, but they also may develop acutely. Sometimes the symptoms develop after an episode of infectious bacterial gastroenteritis.

The diarrhoea is often associated with bleeding. Postprandial diarrhoea is common, as are urgency and incontinence. Nocturnal diarrhoea may also be present. The diarrhoea is caused by impaired colonic peristalsis and impaired sodium and chloride transport, resulting in reduced salt and water absorption. In more severe episodes, the diarrhoea is accompanied by severe abdominal cramping. Systemic symptoms may also develop. Symptoms of anaemia (fatigue, orthostasis), nausea, anorexia, fever, and ulcers in the oral mucosa may be present. Other symptomatic inflammatory processes may be found, such as arthropathy (5–10% of patients), sacroilitis (12–15%), ankylosing spondylitis (1–2%), erythema nodosum (2–4%), uveitis or episcleritis (5–8%), and primary sclerosing cholangitis (3%).

The diagnosis of UC is usually made on the basis of the endoscopic appearance and histological findings combined with a history that is compatible with UC. Other laboratory tests, such as stool studies, are done to rule out other aetiologies. Findings that are compatible with the diagnosis of UC include stool with white blood cells, red blood cells, and eosinophils; iron deficiency and a hypochromic, microcytic anaemia; leukocytosis; hypoalbuminemia; electrolyte abnormalities (e.g. hypokalaemia); and elevated liver enzymes.

Plain radiographs of the abdomen can be very useful in milder disease, but they should be avoided in severe disease—particularly if the bowel is dilated—because of the high risk of perforation or the precipitation of acute dilatation (acute toxic megacolon). Gentle sigmoidoscopy is generally preferred, as it offers additional diagnostic information by means of direct visualization and the opportunity for biopsy.

The evaluation and history allow categorization into mild, moderate, or severe disease. Mild disease presents with fewer than four stools daily (with or without blood), no systemic signs of toxicity, and a normal erythrocyte sedimentation rate. Moderate disease is characterized by more than four stools daily but no significant signs of systemic toxicity. Severe disease presents with more than six stools daily, haematochezia, and systemic signs of toxicity (e.g. fever, anaemia, elevated erythrocyte sedimentation rate).

Treatment can be tailored to the severity of the disease. Using the American College of Gastroenterology guidelines, patients with mild or moderate rectal or rectosigmoid disease are treated with an oral or topical aminosalicylate (e.g. sulfasalazine or mesalamine drugs) or a topical steroid (corticosteroid suppositories or enemas). If there is no response to the aminosalicylates, oral prednisone is added. For patients with more diffuse disease that is mild or moderate, oral aminosalicylates are usually effective, but oral corticosteroids are added if there is inadequate response.

Intravenous steroids are used in patients with severe disease that does not respond to oral corticosteroids. Patients who do not respond to corticosteroids in the acute phase or who have severe disease may respond to immunosuppressive therapy. Cyclosporine is often beneficial in controlling the symptoms and can be used as first-line treatment in patients with severe disease. Cyclosporine combined with corticosteroids is more effective than either agent alone. Azathioprine and 6-mercaptopurine are additional treatment options in patients with moderate to severe disease, but they have a slower onset of action.

Miscellaneous agents used for treatment include infliximab, antibiotics (specifically, tobramycin), nicotinamide, and probiotics. Infliximab, a chimeric monoclonal antibody against tumour necrosis factor-alpha, is being studied for use in UC. Infliximab has been very successful in the treatment of Crohn’s disease, but the results of small studies in which infliximab was used for patients with UC have been mixed. Tobramycin has been shown to improve remission rates in patients concurrently treated with corticosteroids, but it is not routinely used. Transdermal nicotine patches have provided relief in some patients with mild to moderate disease; the benefit appears to be greatest for ex-smokers. The use of probiotics, such as non-pathogenic forms of E. coli, Bifidobacterium, and Saccharomyces boulardii, have shown some initial promise, but further study is needed to determine effectiveness.

Surgery is indicated for patients with severe disease that does not respond to corticosteroids or immunosuppressive agents. The specific indications for surgical intervention...
(urgent colectomy) include a severe episode that does not respond to therapy, complications including perforation or acute dilation of the colon, chronic severe disease, and histological evidence of dysplasia or malignancy.

Once the acute symptoms are controlled, maintenance therapy can be started. Maintenance with aminosalicylates, such as sulfasalazine or the newer mesalamine drugs, is usually effective. Therapy must be continued indefinitely to prevent recurrence of the symptoms. Because corticosteroids do not prevent relapse and have significant long-term adverse effects they are not indicated for maintenance therapy. About 80% of patients have intermittent recurrences, and about 10% have chronic, persistent disease despite appropriate maintenance therapy.

Acute severe haemorrhage, electrolyte abnormalities, nutritional deficits, colon perforation, acute dilation of the colon (toxic megacolon), and dysplasia may develop. Acute dilation of the colon can be precipitated by the use of narcotics and anticholinergic agents as well as colonoscopy. Even with treatment, mortality with severe disease can be as high as 3%.

The risk of carcinoma increases over time, but the risk is greater if the disease developed early in life. The risk severity corresponds with the duration of the disease and an increased risk is noted in patients who have had the disease for at least 10 years.

Further reading

Hair follicles go through three stages of growth. About 90% of the time, hair follicles are in the anagen phase, which is a slow-growth period. Mitotic activity in the root produces the keratinized cells of the shaft and the cortex. The growth period for scalp hair ranges from 2 to 6 years and results in the growth of about 15 cm a year.

The next stage is the catagen phase, which represents cell death in the keratinocytes that create the hair matrix. Fewer than 1% of scalp hairs are in the catagen phase at any given time. Cell division stops and the hair follicle begins to involute.

The telogen phase is a resting phase, and it is during this phase that normal hair loss occurs. The telogen-phase hairs are lost at a rate of about 100 hairs a day. About 10% of hair follicles are in this phase at a given time, and these are randomly distributed. Any interruptions of this cycle will result in excessive hair loss.

The first thing to determine is whether the hair loss has been gradual or of sudden onset. A sudden onset of hair loss suggests disruption of the telogen growth phase.

A history of new medications, serious illness, or thyroid disorders should be investigated, as these can disrupt the telogen phase.

The pattern of the hair loss can be very helpful in establishing the differential diagnosis.

Androgenic alopecia is the most common cause of hair loss in men and women. In men, the initial hair-loss pattern results in thinning on the temples and crown. Women will have central and frontal scalp thinning without the loss of hair at the temples. In women with androgenic alopecia, it may be familial, or it may be the result of hyperandrogenism; during the examination, other signs of excess androgens should be noted.

A diffuse pattern of loss suggests a systemic cause of loss, particularly if body hair is also affected. Prolonged fever, hypothyroidism, toxins, hormonal changes, and severe illness, injury, or psychological stress can cause diffuse hair loss.

Patchy or focal hair loss can result from a number of local factors. Trauma from habitual hair pulling (trichotillomania) or excessive tension on the hair (as with tight hair braids) may cause focal hair loss. In this case, the base of the area will have multiple broken-off hairs. Local infection with tinea or cellulitis can cause local hair loss also.

Alopecia areata is a condition characterized by patches of complete hair loss. This may evolve into alopecia universalis, in which all body hair, including eyelashes and eyebrows, is lost. These conditions can be familial and are more common in patients with Down syndrome. In addition, both forms may be due to autoimmune activity, but the exact cause is unknown.

The “hair pull” test is done by grasping about 40 hairs. The hair is then pulled with slow, constant traction—just enough to pull the scalp up slightly. The hair is gradually released by sliding the fingers up the shafts without releasing the traction. Under normal circumstances, fewer than six hairs should come out.

Examine the bulb of the hair under a microscope. If the bulb is small, white, and oval-shaped, the follicle is in telogen phase. Normally, fewer than 10% of the hair follicles in the sample would be telogen hairs. In the growth (anagen) phase, the bulb is larger, cylindrical, and pigmented (unless the hair is white). Hair that is thinner near the shaft just distal to the bulb (called “exclamation mark” hair because it resembles the appearance of the symbol) is characteristic of alopecia areata.

If the shaft of the hair is abnormal, the hair will fragment, and the bulb will not pull free with the hair follicle. The shaft can be further evaluated by clipping a middle segment of hair and examining it on a wet slide under the microscope. Chemical damage or structural defects will be evident in the shaft. If the diagnosis is still in doubt, a scalp biopsy may be useful.

For patients with diffuse hair loss, blood tests for metabolic diseases such as hypothyroidism and autoimmune diseases such as lupus are indicated.

In most cases, if the loss is caused by infection, treating the infection will result in eventual regrowth of the hair. The growth cycle resumes once the local inflammation resolves. Hair loss is permanent in areas of scarring that might have resulted from the infection.

If the hair loss is the result of a systemic disease or illness, treating the underlying disorder is necessary to prevent further loss. In most cases, as the illness resolves or is controlled, hair growth resumes.

Androgenic alopecia can be treated with antiandrogens (Minoxidil and finasteride). Hair transplant surgery, hair weaves, or wigs may be useful for some patients.

Treatment for alopecia areata and alopecia universalis can be difficult and should be managed by a specialist. Topical immunotherapy (e.g. with diphencyprone) or therapy with intralesional corticosteroids, antralin, ultraviolet light,
This clinic will be closed from 

for Lunar New Year.

In an emergency, please contact:

如有緊急查詢，請致電：
or psoralen are helpful in local disease, but they are not curative. Fortunately, most patients have regrowth of their hair within a year, with or without treatment. Up to 10% have permanent hair loss.

Trichotillomania is a psychiatric disorder that may be related to the anxiety disorders or obsessive-compulsive disorders. A distinction must be made between obsessive hair pulling and habitual hair pulling, as the approach to treatment may be different. Some patients respond to behaviour therapy and some to treatment with clomipramine.

Further reading

Please indicate whether the following statements are true or false.
1. The anagen phase is a rapid-growth period.
2. Fewer than 1% of scalp hairs are in the catagen phase at a given time.
3. Telogen-phase hairs are lost at a rate of about 100 hairs a day.
4. In the anagen phase, the hair bulb is large, cylindrical, and pigmented.

1. b        2. d        3. a        4. b

Characteristic features of Japanese women’s hair with aging and with progressing hair loss

Background: There have been few studies of the features of hair with aging and hair loss in Japanese women. Objective: Features of Japanese women’s hair with aging and with progressing hair loss were investigated. Methods: Japanese women with hair loss (n = 46) or with no or less hair loss (n = 113), aged 14–68 years, were studied. Severity of hair loss was rated by visual comparison with six standard photographs. Hair density, hair growth rate, and hair diameter were analyzed by phototrichogram. Follicular units were deduced by a non-invasive method using tree-view analysis on scalp imaging. Results: Hair loss in Japanese women is commonly characterized by a diffuse central pattern occurring after approximately 40 years of age. Hair density declines with age after the 40s. The reduction resulted from an increase in the number of one-haired follicular units and a reduction of three- and more-haired follicular units. Both the ratio and the growth rate of anagen hair also declined with age after the 40s. Mean hair diameter and the ratio of thick hairs increased with age from about 10 to 40 years, and decreased with progressing hair loss. There were few vellus-like hairs in women with hair loss, in comparison with male-pattern baldness. Conclusion: In Japanese middle-aged women, hair density declined with age without the appearance of hair loss. Hair loss appeared after approximately 40 years of age. The major causes might be reduction of hair density and the ratio of thick hairs, but not an increase of vellus-like hairs.
An extremely common dermatological problem worldwide, the prevalence of atopic dermatitis (AD) has been increasing over recent decades, especially among young children in developed countries, suggesting that genetic factors cannot be the only component in the development of the disease, which has a multifactorial pathophysiology [1,2].

Pathophysiology

The rise in the number of cases of AD coincides with an increasing exposure to environmental agents that impair epidermal barrier function, including soaps, detergents and house dust mites that, together with an immunological element or bacterial infection, are thought to be the major components underlying the pathophysiology of AD [3].

There is increasing evidence that loss of epidermal barrier integrity is an important factor in the development of AD [4]. Genes have been identified that regulate important components of epidermal barrier functions, abnormalities of which can cause ichthyosis, or extreme drying of the skin with keratinization, which commonly occurs with AD [5]. Impairment of epidermal barrier integrity is thought to allow allergens to penetrate the skin, resulting in immune system sensitization.

Regarding the immunopathogenesis of AD, the skin can be considered an organ with an independent immune system. This becomes abnormal in patients with AD, who have a tendency to mount exaggerated hypersensitivity reactions to common allergens [6], at the same time failing to mount an effective immune defence mechanism against infection by bacteria and viruses.

Patients with AD are often carriers of infectious bacteria such as Staphylococcus aureus due to their impaired immune defence systems and poor epidermal barrier function. Such infections produce allergenic proteins that can elicit a strong inflammatory response, thereby exacerbating the symptoms of AD [7].

Treatment

The above pathophysiological mechanisms underlying AD should all be considered when choosing a suitable treatment. It is common for patients to present to a dermatologist after having been prescribed topical steroids by their general practitioner, with no consideration having been paid to potential disruption of epidermal barrier function or the possibility of bacterial infection. Topical steroids alone may suppress inflammation, but are relatively ineffective if AD is due to impaired epidermal barrier functions, necessitating the use of emollients and moisturizers [8].

However, patients with AD are also more prone to develop allergic reactions to potential allergens and may go on to develop contact dermatitis (CD) when exposed to certain preservatives in skin care products, also known as CD to chronic medicament usage [9]. Therefore, it is important that such products contain only hypoallergenic constituents.

Lipids, including phospholipids, are known to be very important in terms of providing effective barrier functions [10]. Topical skin-care creams are now available that contain phospholipid components as a means of improving epidermal barrier functions, with recent clinical data supporting the anti-irritative and anti-inflammatory effects of these products [11].

The different types of moisturizers include those of the oily type that contain no preservatives but are too oily, while others contain phospholipids and other chemical agents that bind water within the skin.

In addition to phospholipids and water-binding agents, moisturizers containing N-palmitoylethanolamine (PEA) have also been shown to reduce epidermal inflammation and irritation [12], at the same time being less oily and more tolerable. However, despite claims of hypoallergenicity, lipid-based moisturizers also need to contain at least some degree of preservative, due to the risk of bacterial proliferation.

A recent trend has been the addition of an antiseptic agent to routine skin care procedures to prevent bacterial overgrowth. In the US, some dermatologists even recommend the addition of bleach to bathwater, the idea being to reduce the growth of bacterial flora, especially that of S. aureus, thereby preventing production of protein allergens and the resulting so-called ‘super-antigen’ response by the immune system. However, local Hong Kong patients find bleach unacceptable, although various skin-care products are now available that contain other antiseptics.

Other non-steroidal medications that are frequently used for the treatment of AD include tacrolimus and pimecrolimus, particularly as an adjunctive therapy in patients who are prone to develop atrophy of the skin due to the use of topical steroids. However, there are safety issues
with these non-steroidal drugs, which prompted a 2006 US Food and Drug Administration black-box warning, based on animal study data showing an increased prevalence of skin cancer with these drugs [13].

Patients using moisturizers or emollients should be advised to apply them to affected areas only, after washing with hypoallergenic soap and gently patting themselves dry, in order to moisturize the skin more effectively, particularly in the winter months when more potent moisturizers tend to be needed. Application of antiseptic additives two to three times weekly followed by showering is also recommended.

References

Please indicate one answer to each question

1. Patients with AD are often carriers of infectious bacteria such as ________________________ due to their impaired immune defence systems and poor epidermal barrier function.
   a. Staphylococcus epidermidis
   b. Staphylococcus aureus
   c. Staphylococcus warneri
   d. Streptococcus pyogenes

2. Topical steroids alone may suppress inflammation, but are relatively ineffective if AD is due to impaired epidermal barrier functions, necessitating the use of emollients and moisturizers.
   a. True  b. False

3. Moisturizers containing __________________________ have also been shown to reduce epidermal inflammation and irritation.
   a. N-acyl-ethanolamine (NAE)
   b. N-oleoyl-ethanolamine (OEA)
   c. N-palmitoyl-ethanolamine (PEA)
   d. N-stearoyl-ethanolamine (SEA)

4. In the ___________ months, more potent moisturizers tend to be needed.
   a. Spring  b. Summer  c. Autumn  d. Winter
Please return completed answer sheet to the HKMA Secretariat (Fax: 2865 0943) on or before 15 February, 2011 for documentation. However, if you choose to do the exercises online, you do not need to return this answer sheet by fax.

Please answer ALL questions and write the answers in the space provided.

Both the Cardiology and Dermatology courses must be completed to earn 0.5 CME point. The other courses attract 1 CME point each.

**SPOTLIGHT**

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

**CARDIOLOGY**

| 1 | 2 | 3 |

**GASTROINTESTINAL MEDICINE**

| 1 | 2 | 3 | 4 |

**GENERAL MEDICINE**

| 1 | 2 | 3 | 4 |

**DERMATOLOGY REVIEW**

| 1 | 2 | 3 | 4 |

**DERMATOLOGY**

1. ____________________________________________________________________________________________________________________

____________________________________________________________________________________________________________________

2. ____________________________________________________________________________________________________________________

____________________________________________________________________________________________________________________

3. ____________________________________________________________________________________________________________________

____________________________________________________________________________________________________________________

4. ____________________________________________________________________________________________________________________

____________________________________________________________________________________________________________________

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**Name 姓名:** ________________________________

HKMA Membership No. or HKMA CME No. 香港醫學會會員編號或持續進修編號:

HK ID No. 香港身份證號碼: □□□ □ □ □ □ □ □ XXX (x)

Signature 簽名: ______________________________

Contact Tel No. 聯絡電話: ____________________

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THIS MONTH GO ONLINE
AND COMPLETE UP TO 3 OTHER
MONTHLY COURSES FOR AN EXTRA
3 CME POINTS

[www.hkmacme.org](http://www.hkmacme.org)
I would like to register for the following CME lecture(s)

<table>
<thead>
<tr>
<th>HKMA Structured CME Programme with HKS&amp;H</th>
<th>HKMA Member</th>
<th>CME Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>17 February, 2011 HKMA Structured CME Programme with HKS&amp;H Year 2010 Session III – Pain Management</td>
<td>HK$50 ☐</td>
<td>HK$80 ☐</td>
</tr>
</tbody>
</table>

I enclose herewith a cheque of HK$____ to cover the CME payment.

Name: ____________________________ Tel No.: __________ Fax No./Signature: __________

HKMA Membership No./CME No.: ____________________________

Please register for participation. First come, first served. 名額有限 請早登記

Please be informed that Confirmation Letter of Registration is required. If you have not received any replies, please do not hesitate to contact us at 2527 8452.

REGISTRATION:

Please fill in and return the Registration Form together with a cheque of adequate amount made payable to "The Hong Kong Medical Association" to 5/F Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong. Each lecture will carry 1 CME point under the MCHK/HKMA CME Programme (unless otherwise stated).

Accreditation from other colleges is pending. (The Secretariat fax no.: 2865 0943)

Please note that Light lunch will begin at 1:15 p.m. unless otherwise stated.

CME EVENT 講課簡介

17 February, 2011 (Thursday)
HKMA Structured CME Programme with HKS&H Session II: Pain Management

Dr. Li Ching Fan, Carina
MB BS (HK), FANZCA, FHKCA, FHKAM (Anaesthesiology), Dip. Pain Mgt (HKCA), Specialist in Anaesthesiology

This symposium is co-organized with Hong Kong Sanatorium & Hospital

VENUE & TIME 地點及時間

The HKMA Dr. Li Shu Pui Professional Education Centre 2/F, Chinese Club Building
21-22 Connaught Road Central, HK
Lecture: 2:00–3:00 p.m.
(Light lunch will begin at 1:15 p.m.)

香港中環干諾道中二十一至二十二號華商會所大廈二樓
香港醫學會李樹培醫生專業教育中心
講課：下午二時至三時正
（茶點於下午一時十五分開始）
## HKMA Certificate Course on Family Medicine

**Jointly organized by**
- Hong Kong Medical Association
- School of Public Health and Primary Care, CUHK
- Queen Elizabeth Hospital

**DATE** | **TOPIC** | **Speaker(s)**
--- | --- | ---
13 February, 2011 | **Geriatric Giants** | Dr. TAM Kui Fu, Stanley  
Specialist in Geriatric Medicine, Department of Medicine, Queen Elizabeth Hospital  
Dr. WONG Wai Hong  
Specialist in Geriatric Medicine, Private Practice

13 March, 2011 | **Influenza and Pneumococcal Vaccination** | Dr. CHAN Yee Shing, Alvin  
Vice-President, The Hong Kong Medical Association  
Dr. TSE Hung Hing  
Immediate Past President, The Hong Kong Medical Association

**Venue** | Lecture Theatre, G/F, Block M, QEH  
**Time** | 2:00–5:00 p.m.  
**CME points** | 3  
**Fee** | HK$50 per lecture for HKMA Members  
HK$80 per lecture for CME Participants  
Light snacks will be provided

**Certification Requirement:** 75% attendance

### Registration Form

Please fill in and return the Registration Form together with a cheque of adequate amount made payable to “The Hong Kong Medical Association” to 5/F Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong **before the date of the function.**

**First come, first served. 名額有限 請早登記**

### HKMA Certificate Course on Family Medicine at QEH — Registration Form

**I would like to register for the following lecture(s):**

<table>
<thead>
<tr>
<th>QE</th>
<th>Date</th>
<th>Topic</th>
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</thead>
<tbody>
<tr>
<td>13 February, 2011</td>
<td>Family Medicine</td>
<td></td>
</tr>
<tr>
<td>13 March, 2011</td>
<td>Family Medicine</td>
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</tbody>
</table>

I enclose herewith a cheque of **HK**

**Name:**  
**Tel No.:**  
**Fax No.:**

**HKMA Membership No./CME No.:**

**Signature:**

Data collected will be used and processed for the purposes related to the MCHKHKMA CME Programme only. All registration fees are not refundable or transferable. Personal data will be subject to the Hong Kong Medical Association’s Privacy Policy. For more details, please visit www.hkmacme.org.
### CME Calendar

**January 2011**

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Event Description</th>
</tr>
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<tbody>
<tr>
<td>15 (Sat)</td>
<td>10:00am–12:30 pm</td>
<td>Hospital Authority: Head Office – Nephrology/Co-ordinating Committee in Surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Surgical Outcomes Monitoring &amp; Improvement Programme (SOMIP) Forum</td>
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<tr>
<td>16 (Sun)</td>
<td>8:45 am–4:15 pm</td>
<td>Hong Kong Doctors Union</td>
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<tr>
<td></td>
<td></td>
<td>The 198th Hong Kong Doctors Union Sunday Afternoon Symposium</td>
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<tr>
<td>16 (Sun)</td>
<td>3:00–6:30 pm</td>
<td>Hong Kong College of Cardiology</td>
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<tr>
<td></td>
<td></td>
<td>The 199th Hong Kong Doctors Union Sunday Afternoon Symposium</td>
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<tr>
<td>17 (Mon)</td>
<td>7:00–10:00 pm</td>
<td>The University of Hong Kong: Faculty of Social Sciences – Hong Kong University Family Institute</td>
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<tr>
<td></td>
<td></td>
<td>Supervision Group in Marriage and Family Therapy (2010–2011)</td>
</tr>
<tr>
<td>19 (Wed)</td>
<td>8:30 am–5:30 pm</td>
<td>Hospital Authority: Caritas Medical Centre</td>
</tr>
<tr>
<td></td>
<td></td>
<td>American Heart Association Advanced Cardiovascular Life Support Provider Renewal Course (Fee: HK$800)</td>
</tr>
<tr>
<td>19 (Wed)</td>
<td>1:00–3:00 pm</td>
<td>Hong Kong Doctors Union</td>
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<td>Tsuen Wan Study Group</td>
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<td>Acute and Chronic Visual Loss, Ocular Trauma</td>
</tr>
<tr>
<td>19 (Wed)</td>
<td>4:15–5:15 pm</td>
<td>The University of Hong Kong: Dept of Obstetrics &amp; Gynaecology</td>
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<tr>
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<td>Tumour Board Meeting – Clinical–Pathological Conference on Gynaecological Oncology Cases</td>
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<tr>
<td>19 (Wed)</td>
<td>6:30–10:00 pm</td>
<td>Hong Kong Community Psychological Medicine Association</td>
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<td></td>
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<td>GAD &amp; Psychosomatic Disorders in Primary Care</td>
</tr>
<tr>
<td>20 (Thu)</td>
<td>1:00–4:00 pm</td>
<td>Hong Kong Medical Association: Best Drugs Action Committee/ New Territories West Community Network</td>
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<tr>
<td></td>
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<td>Certificate Course on Management of Drug Abuse Patients for Family Doctors: 1) Ketamine Uropathy 2) Psychological Treatment of Drug Abuse</td>
</tr>
<tr>
<td>21 (Fri)</td>
<td>9:00 am–5:00 pm</td>
<td>The University of Hong Kong: Dept of Surgery</td>
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<td>Hong Kong Surgical Forum – Winter 2011</td>
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<td></td>
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<td>Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital Forum Secretary – Tel: 2255 4885</td>
</tr>
<tr>
<td>21 (Fri)</td>
<td>10:00 am–12:30 pm</td>
<td>The University of Hong Kong: Carol Yu Centre for Infection/ Dept of Microbiology</td>
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<tr>
<td></td>
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<td>Infectious Diseases Rounds for Year 2011</td>
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<tr>
<td></td>
<td></td>
<td>Conference Room, Room 405, Clinical Pathology Building, Queen Mary Hospital</td>
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<tr>
<td></td>
<td></td>
<td>Ms. Karis Lam – Tel: 2255 3243</td>
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<tr>
<td>22 &amp; 30</td>
<td>1:00–4:00 pm</td>
<td>The University of Hong Kong: Dept of Surgery</td>
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<tr>
<td>(Sat &amp; Sun)</td>
<td></td>
<td>Pre-Hospital Trauma Life Support (PHTLS) Provider Course</td>
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<td>St. John Tower, 2 McDonnell Road, Hong Kong</td>
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<td></td>
<td></td>
<td>Senior Training Officer – Tel: 2250 8020</td>
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<tr>
<td>23 (Sun)</td>
<td>1:00–5:00 pm</td>
<td>Association of Licentiates of Medical Council of Hong Kong</td>
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<tr>
<td></td>
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<td>1) Update in Asthma Management</td>
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<td>2) Anxious Mother, Anxious Child</td>
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<tr>
<td>24 (Mon)</td>
<td>10:00 am–5:00 pm</td>
<td>The University of Hong Kong: Hong Kong University Family Institute</td>
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<tr>
<td></td>
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<td>One-Day Workshop on “Mentalization-Based Work with Families”</td>
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<td>Hong Kong University Family Institute, 5/F, Tszan Yuk Hospital</td>
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<td></td>
<td></td>
<td>Ms. Lam Koon Che, Rachel – Tel: 2859 5300</td>
</tr>
<tr>
<td>25 (Tue)</td>
<td>6:00–7:30 pm</td>
<td>The University of Hong Kong: Dept of Obstetrics &amp; Gynaecology</td>
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<td></td>
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<td>Tung Wah Group of Hospitals: Medical Division</td>
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<td>The Clinical Care of Metabolic Syndrome: Western and Chinese Medicine Means</td>
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<td></td>
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<td>Lecture Theatre, 1/F, TWGHs Yu Chun Keung Memorial Medical Centre, Kwong Wah Hospital</td>
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<tr>
<td></td>
<td></td>
<td>Ms. Darlene Lau – Tel: 2857 7729</td>
</tr>
<tr>
<td>26 (Wed)</td>
<td>4:15–5:15 pm</td>
<td>The University of Hong Kong: Dept of Obstetrics &amp; Gynaecology</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tumour Board Meeting – Clinical–Pathological Conference on Gynaecological Oncology Cases</td>
</tr>
</tbody>
</table>

For full details and registration, please visit the HKMA CME Bulletin online at www.hkmacme.org.
<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Event</th>
<th>Venue</th>
<th>Contact</th>
</tr>
</thead>
<tbody>
<tr>
<td>27 (Thu)</td>
<td>1:00–4:00 pm</td>
<td>Hong Kong Medical Association: Beat Drugs Action Committee/New Territories West Community Network</td>
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<tr>
<td></td>
<td></td>
<td>Certificate Course on Management of Drug Abuse Patients for Family Doctors: 1) Latest Drug Scene in Hong Kong 2) Service Delivery by Local Substance Abuse Clinic</td>
<td>Auditorium, Pok Oi Hospital</td>
<td>Ms. Queenie Lam – Tel: 2527 8285</td>
</tr>
<tr>
<td>28 (Fri)</td>
<td>8:00 am–12:00 pm</td>
<td>The Institute of Clinical Simulation</td>
<td>The Institute of Clinical Simulation, 3/F, North District Hospital, Ms. Katherine Ip – Tel: 2683 8307</td>
<td></td>
</tr>
<tr>
<td>28 (Fri)</td>
<td>10:00 am–12:00 pm</td>
<td>The University of Hong Kong: Carol Yu Centre for Infection/Dept of Microbiology</td>
<td>Infectious Diseases Rounds for Year 2011 Conference Room, Room 405, Clinical Pathology Building, Queen Mary Hospital Ms. Karis Lam – Tel: 2255 3243</td>
<td></td>
</tr>
<tr>
<td>28 (Fri)</td>
<td>12:30–3:30 pm</td>
<td>Hong Kong Medical Association: Shatin Doctors Network</td>
<td>Contemporary BPH Management</td>
<td>Jasmine Room 1, Level 2, Royal Park Hotel Tel: 2649 4468</td>
</tr>
<tr>
<td>28 (Fri)</td>
<td>1:00–2:00 pm</td>
<td>Hospital Authority: Castle Peak Hospital – Psychiatry Dept</td>
<td>CBT Skill Training – Other Cognitive/Behavioural Techniques (activity scheduling, etc)</td>
<td>Seminar Room 4, Block F, Castle Peak Hospital Ms. Cherry Man – Tel: 2456 7855</td>
</tr>
<tr>
<td>28 (Fri)</td>
<td>1:00–3:15 pm</td>
<td>Hong Kong Medical Association: Hong Kong East Community Network</td>
<td>Update on Osteoporosis</td>
<td>The Hong Kong Medical Association Wanchai Premises, 5/F, Duke of Windsor Social Services Building, 15 Hennessy Road, Wanchai Ms. Carolice Tang – Tel: 2527 8265</td>
</tr>
<tr>
<td>31 (Mon)</td>
<td>7:00–10:00 pm</td>
<td>The University of Hong Kong: Faculty of Social Sciences – Hong Kong University Family Institute</td>
<td>Supervision Group in Marriage and Family Therapy (2010–2011)</td>
<td>Hong Kong University Family Institute, 5/F, Tszan Yuk Hospital Ms. Lam Koon Che, Rachel – Tel: 2859 5300</td>
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<tr>
<td>10 (Thu)</td>
<td>5:30–7:00 pm</td>
<td>The University of Hong Kong: Dept of Obstetrics &amp; Gynaecology</td>
<td>Tutorials for Training in Prenatal Diagnosis and Counselling: Central Nervous System/Face and Profile</td>
<td>Seminar Room K509, Queen Mary Hospital Ms. Annie Chow – Tel: 2255 3401</td>
</tr>
<tr>
<td>11 (Fri)</td>
<td>8:00 am–4:30 pm</td>
<td>The Institute of Clinical Simulation</td>
<td>Safe Sedation Course</td>
<td>The Institute of Clinical Simulation, 3/F, North District Hospital Ms. Katherine Ip – Tel: 2683 8307</td>
</tr>
<tr>
<td>11 (Fri)</td>
<td>10:00 am–12:00 pm</td>
<td>The University of Hong Kong: Carol Yu Centre for Infection/Dept of Microbiology</td>
<td>Infectious Diseases Rounds for Year 2011</td>
<td>Conference Room, Room 405, Clinical Pathology Building, Queen Mary Hospital Ms. Karis Lam – Tel: 2255 3243</td>
</tr>
<tr>
<td>11 (Fri)</td>
<td>3:00–6:00 pm</td>
<td>Hong Kong College of Psychiatrists</td>
<td>Qualitative Study</td>
<td>Lecture Theatre, LG/F, Block J, Kwai Chung Hospital Tel: 2456 7111</td>
</tr>
<tr>
<td>12 (Sat)</td>
<td>2:30–4:30 pm</td>
<td>Hong Kong Medical Association</td>
<td>Refresher Course for Health Care Providers 2010/2011 – Growth Problems in Children</td>
<td>Hospital Authority: Our Lady of Maryknoll Hospital Ms. Clara Tsang – Tel: 2354 2431</td>
</tr>
<tr>
<td>13 (Sun)</td>
<td>2:00–5:00 pm</td>
<td>Hong Kong Medical Association</td>
<td>The Chinese University of Hong Kong: School of Public Health &amp; Primary Care</td>
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<td>14 (Mon)</td>
<td>7:00–10:00 pm</td>
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<td>Supervision Group in Marriage and Family Therapy (2010–2011)</td>
<td>Hong Kong University Family Institute, 5/F, Tszan Yuk Hospital Ms. Lam Koon Che, Rachel – Tel: 2859 5300</td>
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**February 2011**

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<thead>
<tr>
<th>Date</th>
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<th>Venue</th>
<th>Contact</th>
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<tr>
<td>8 (Tue)</td>
<td>5:30–6:30 pm</td>
<td>The University of Hong Kong: Dept of Obstetrics &amp; Gynaecology</td>
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<td>Seminar Room K509, Queen Mary Hospital Ms. Annie Chow – Tel: 2255 3401</td>
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<tr>
<td>10 (Thu)</td>
<td>5:30–7:00 pm</td>
<td>Hospital Authority: Tuen Mun Hospital – Family Medicine Dept</td>
<td>Dermatology Workshop</td>
<td>Room 614, 6/F, Ambulatory Care Centre, Tuen Mun Hospital Ms. Cowin Tang – Tel: 2456 6601</td>
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