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Editorial

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- Minimally Invasive Spine Surgery — *Dr. Daniel WK Ng*
- Carotid Stenosis — *Dr. George KC Wong, Dr. David TF Sun*

Dermatological Quiz

- Dermatological Quiz — *Dr. Ka-ho Lau*

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- News from FMSHK
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THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
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Congratulations to

Dr Margaret F.C. Chan

as the newly appointed
Director-General of the
World Health Organization

The Medical and Dental Directory of Hong Kong - new 8th Edition

Call for submission of individual data from all medical and dental practitioners

To all medical and dental practitioners,

We would like to take this opportunity to invite you to send us your most up-to-date information. You can complete the form and return to us by facsimile, through mail or via our Federation's Home Page: www.fmshk.org/directory . All respondents will be entitled to a free copy of the Medical & Dental Directory of Hong Kong 2007 upon submission of data. A voluntary contribution of HK$100 or above is welcome and upon submission, a data CD and Directory will be sent to your office (includes postage and package), otherwise you may have to pick up the Directory from the Federation office yourself if no contribution is made. In case we do not hear from you, basic information will be extracted from the available records of the Medical Council of Hong Kong and the Dental Council of Hong Kong. Thank you for your co-operation.

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The Federation of Medical Societies of Hong Kong

Editorial

Dr. Walter WK King
Editor-in-Chief, Hong Kong Medical Diary

Dr. Walter WK King

It gives me great pleasure to take this opportunity to thank all members of the Editorial Board of the Hong Kong Medical Diary (HKMD) for making 2006 a year of sound and solid growth for the HKMD with gains in both the quality of the scientific content (as evident in this issue on Neurosurgery edited by **Dr Fan Yiu-wah**) and the financial aspects of distributing 8500 copies of HKMD and over 3000 email alerts to many medical, dental and allied health practitioners in Hong Kong. The HKMD has become an important monthly multi-medium for the introduction, updating and exchange of information on new, refined or established medical technologies, treatment results, drug applications and medical advances.

The Issue Editors of each month play an especially important role in safe guarding the growth and development of the HKMD. I would especially like to thank the following 12 Issue Editors of 2006:

January 2006	**- Dr Ho Pak-leung**	**Clinical Microbiology and Infectious Diseases**
February 2006	**- Dr Alvin K.H Kwok**	**Ophthalmology**
March 2006	**- Dr Antonio A.T Chuh**	**Family Medicine**
April 2006	**- Dr Jason K.Y Fong**	**Neurology**
May 2006	**- Dr Tsang Wai-kay**	**Nephrology**
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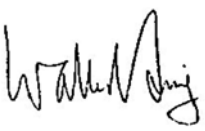
November 2006	**- Dr Mok Mo-yin**	**Rheumatology**
December 2006	**- Dr Fan Yiu-wah**	**Neurosurgery**

In appreciation of their significant work, the President of the Federation of Medical Societies of Hong Kong, Dr Dawson Fong and members of the Executive Committee are preparing a hard bound copy of the past 12 issues of the HKMD for the Issue Editors to keep as a momento of their great achievements.

There will be many more interesting manuscripts to be published in 2007. Let me thank in advance all the 2007 Editors for their forth coming hard work and diligence. Wishing you all a very Merry Christmas and a Prosperous New Year!



Dr Walter W.K King
Editor-in-Chief, Hong Kong Medical Diary

Management of Trigeminal Neuralgia

Dr. Yiu-wah Fan MBBS(HK), FRCS(SN)(Edin), FRACS, FCSHK, FHKAM(Surgery)

Specialist in Neurosurgery

Editor

Dr. Yiu-wah Fan

Symptoms and signs

Trigeminal neuralgia is a relatively common pain condition. The diagnosis is based on the typical history of episodic electric pain in the distribution of the trigeminal nerve. Touching the face or eating can sometimes provoke an attack. There is commonly a trigger area on the face or within the mouth that stimulation of it will provoke a neuralgic attack. Because of the pain and apprehension, many patients do not clean their face or shave their beard on the affected side. One can often make a spot diagnosis by noticing the dirt and beard distribution over the face after hearing the history.

Causes of trigeminal neuralgia

In most patients, the cause of the pain is thought to be related to the vascular compression syndrome. Peter Jannetta (Jannetta, McLaughlin, and Casey) theorised the vascular compression syndrome that a vessel loop compressing on the root entrance zone of the trigeminal nerve can cause trigeminal neuralgia. With chronic vessel compression, segmental demyelination (Love and Coakham) occurs in the trigeminal nerve. Incoming wave of sensory input from receptors in the face travelling through the trigeminal nerve partially reflects back at the area of demyelination. When the frequency of the incoming wave and the reflected wave come into resonance, a big volley of wave is generated and causes a sudden surge of pain. Although the vascular compression theory is popular, it cannot account for all phenomena associated with trigeminal neuralgia. Many patients with trigeminal neuralgia do not have a culprit vessel.

Apart form vascular compression, other pathologies such as tumour compression or intrinsic demyelination problems like multiple sclerosis can also produce trigeminal neuralgia.

Clinical workup

Although the diagnosis of trigeminal neuralgia is largely a clinical diagnosis, I believe some kind of imaging study is warranted in the present era. MRI is commonly used to examine the trigeminal nerve. The main use of imaging study is to exclude unusual pathologies, such as tumour. In the MRI, one needs to examine the whole course of the trigeminal nerve from the brainstem to the skull base. With modern MR technique, one often can identify the culprit vessel loop.(Akimoto et al.)

Medical treatment

In about 80% of patients, the pain can be readily managed with medication. The most useful drug is carbamazepine (Tegretol). Its effect is so specific that it can be used as a therapeutic test. If the pain can be relieved by carbamazepine, one can be quite certain that the diagnosis is trigeminal neuralgia. When one starts treatment with carbamazapine, one needs to build up the dose slowly. Among other side effects, the common problems of dizziness and unsteadiness in the beginning of treatment often scare patients off the medication and claim themselves intolerable to this most useful medication for trigeminal neuralgia. I often warn patients about this particular side effect upfront and teach them to titrate the dose themselves. I ask my patients to start with 100mg a day. If they feel well the next day, they add on another 100mg a day. If there is symptom of unsteadiness, they should stop increasing the dose or back down to the previous dose for a few days before stepping up again. I usually ask them to step up the dose till the pain is under control, or the dose has reached 600mg a day. Then I will review them again for the need of a higher dose or second medication. One can push carbamazapine to 1200mg a day.

There are two other useful medications for trigeminal neuralgia, viz. gabapentin (Neurontin) and pregabalin (Lyrica). If the patient fails or is intolerable to these medications, that will be the time to consider surgery for the condition.

Options of surgery

Surgery can be divided into two types, viz. restorative and destructive. The best surgical treatment is to remove the cause of pain. If it is due to vessel loop or tumour compression, surgical decompression should be the procedure of choice.

When no cause is identified, or the cause is difficult to deal with, one can interrupt the nerve so that it cannot conduct the pain signal to the brain.

There are three common surgical options for trigeminal neuralgia.
1. microvascular decompression
2. percutaneous ablative procedure
3. radiosurgery

Microvascular decompression and selective rhizotomy

In the treatment of pain conditions, the best option is to remove the cause of pain and preserve all sensations. This is the attractive point about microvascular decompression. In a young patient who presents with typical trigeminal neuralgia and MRI showing an arterial loop on the corresponding trigeminal nerve, the best option is to decompress the trigeminal nerve and cushion it off the culprit artery. The result of microvascular decompression is generally very good, with excellent pain control rate of about 70-90%. The risk of mortality associated with this operation is less than 1%. The commonest side effect is hearing loss related to retraction injury of VIII nerve. With the use of intra-operative evoked potential monitor, such complication can now be controlled at 1% (Barker et al.).

However, pre-operative MRI sometimes does not show an offending vessel. There is then an uncertainty about the efficacy of microvascular decompression. There may be nothing to decompress at the time of operation. For proponents of this operation, they will argue that veins and small arteries not easily discernable on MRI can also cause trigeminal neuralgia. They emphasise the need of careful exploration under the microscope. Having said that, I must admit that I have encountered cases without a culprit vessel on exploration.

I now conceptually look upon the operation as an exploratory procedure. I tell my patients that I start the operation with examination of the whole course of trigeminal nerve in the subarachnoid space. If I am convinced that a culprit vessel is compressing on the trigeminal nerve, usually with indentation on the nerve, I will cushion the artery off the nerve. Or if the culprit vessel is a vein, I will coagulate and remove a segment of the vein. If I am not convinced of a culprit vessel, I will do a selective rhizotomy at the root entrance zone. At this level, the trigeminal nerve has a constant topographic pattern. The ophthalmic division (V1) is located at the most superior and medial aspect. The mandibular division (V3) is located at the most inferior and lateral aspect. The maxillary division (V2) lies in the middle. For most patients, trigeminal neuralgia involves the V2 and V3 portions of the nerve. I normally divide the nerve by 50% starting from the lateral inferior aspect and therefore sparing the ophthalmic division.

Percutaneous ablative procedure

There are three common percutaneous techniques in lesioning the trigeminal nerve. All of them involve placement of a cannula through the foramen ovale to access the trigeminal ganglion. Among the three, viz.radiofrequency lesioning, chemical lesioning with glycerol, and balloon compression, I prefer the radiofrequency lesioning technique for its selective and controlled characteristics. It also offers the highest rates of complete pain relief (Lopez, Hamlyn, and Zakrzewska).

Selective rhizotomy of trigeminal nerve with radiofrequency

The procedure can be done as a day case. A cannula is placed through the foramen ovale based on anatomical landmarks and then confirmed with X-ray. The procedure requires intermittent short-lived deep sedation during placement of the needle and lesioning. The position of the needle is adjusted according to the patient's response during electrical stimulation. With fine-tuning of temperature and duration of lesioning, one can burn off the thinly myelinated pain fibre and preserve the thickly myelinated fibres that subserve touch sensation and motor function. Being an ablative procedure, the drawback of it is loss of sensation after the procedure. Most of the time, if the neuralgia is severe pre-operatively, patients will usually accept the hypaesthesia after the operation. In order not to over lesion a nerve, patients have to accept a possible recurrence after the procedure. However, it is not a problem to re-lesion the nerve when the pain recurs.The worst outcome from a lesioning procedure is the creation of analgesia dolorosa, a deafferentation syndrome without very effective treatment.

Radiosurgery

Radiosurgery has recently been established as an effective treatment for trigeminal neuralgia (Gorgulho and De Salles). The largest body of literature is related to gamma knife radiosurgery (Regis et al.). Linac based X-knife system (Richards et al.) and cyberknife (Lim et al.) have also been used to treat trigeminal neuralgia effectively.

A high dose of 70-90 Gy focused radiation is delivered to the subarachnoid portion of the trigeminal nerve. The success of radiosurgery demands a very accurate stereotactic system because the target is small. In a recent report by Regis (Regis et al.), the control rate is up to 83%, with a complication rate of 6% facial paresthesia and 4% hypesthesia.

Summary

Trigeminal neuralgia is one of the readily treatable pain condition. Most patients can be managed with medications (carbamazepine, gabapentin and pregabalin). However, if the pain is not controlled well with medication or patients cannot tolerate the side effect of medication, surgical management should be employs early. Microvascular decompression remains the best approach for treatment of trigeminal neuralgia especially if one can spot a culprit vessel loop on MRI. Percutaneous ablative procedure is very useful to control the pain if the pain is at the V2, V3 region. It is particularly attractive for old patients who cannot tolerate a craniotomy. Radiosurgery is a promising option. I believe it is a good choice for V1 neuralgia in patients who are too frail for MVD.

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THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
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A Go Go to Count Down

The Federation's 2006 Annual Dinner

7:00 pm, 31st December, 2006 (Sunday)
Run Run Shaw Hall
The Hong Kong Academy of Medicine Jockey Club Building
99 Wong Chuk Hang Road, Aberdeen, Hong Kong

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Please reserve _____ table(s)* / seat(s) for me at the Federation Annual Dinner 2006. A cheque for the amount of HK$ _____ (HK$900 per person) in favour of " **The Federation of Medical Societies of Hong Kong**" is enclosed.

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Spinal Dysraphism

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President, The Federation of Medical Societies of Hong Kong

Dr. Dawson Fong

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 December 2006.

Spinal dysraphism encompasses a spectrum of congenital conditions resulting in a defective neural arch through which meninges or neural elements may herniate. These conditions include spina bifida aperta, spina bifida occulta, meningocoele, myelomeningocoele, lipomyelomeningocoele, myeloschisis, and rachischisis - names given variably according to radiological or pathological findings. These variations can be grouped as open if the overlying skin is not intact, pending leakage of cerebrospinal fluid, and occult if the defect is well covered with full thickness skin. The two groups call for quite different approaches.

Prevalence

Prevalence of spinal dysraphism has been on a decline worldwide in the past 2 decades. Many factors are likely involved. Better nutrition of women, timely folate replacement, better antenatal care with high resolution ultrasound resulting in an available option for termination for mothers diagnosed with such a defective foetus and liberal assay of maternal serum alpha-fetoprotein are all contributing factors. In Tuen Mun Hospital, with an annual delivery of more than 5000, the incidence of open spinal dysraphism is about 0.2/1000 live births.

Embryology

The first 2 months of embryogenesis can be divided into 23 stages. Around day 18 at stage 8, the neural plate is formed, followed by neural folds and their subsequent fusion. Neuropore closure is to follow and completed by stage 12 around day 28. When caudal neuropore fails to close, open dysraphism ensues. From then until day 56, secondary neurulation sets in forming the spinal cord distal to the second sacral vertebra. Defective secondary neurulation results in occult dysraphism in which the caudal part of the spinal cord remains connected with the epidermis by tissues of mesenchymal origin - the ultimate cause for tethering later on in life. Since defect occurs so early in pregnancy, folate supplement, proven to be effective in preventing neural tube defects, has to be given in the anticipation of pregnancy.[5]

Pathology

Failure of primary neurulation leads to open dysraphism that poses an immediate threat of CSF leakage and consequently meningitis after birth. Neural placode is exposed in cases of myelomeningocoele. (Fig. 1) Severity of the neurological deficit depends on the degree of malformation of the placode as well the level of the defect - the higher the level the worse is the prognosis. A spectrum of abnormalities may be found in the central nervous system as well as other organs. Chiari malformation type II is nearly a constant associate. (Fig. 2)

In occult dysraphism, spinal cord remains anchored to the overlying intact skin by tissues that vary from adipose tissue to cartilage. Spinal cord lipoma is the most common entity and it can be divided into 3 groups - dorsal, terminal and transitional - results of different degrees of defective neurulation. (Fig. 6a, 7a) Cutaneous manifestation of intradural lipoma with an obvious fatty lump is noted in more than 70% of cases. Other cutaneous stigmata include capillary haemangioma, dermal dimple, bifurcating or eccentric intergluteal cleft, skin tags and hairy patch on the midline at the lumbosacral level. (Fig. 3)

A unique variant among occult lesions is split cord malformation, often noted as diastematomyelia. It is believed to be the result of a failure of midline integration of the newly formed notocordal cells leading to the formation of 2 hemicords. The midline ecto-endo-mesenchymal tract goes on to develop into a fibrous to bony septum and becomes the cause of tethering as the patient grows. (Fig. 4)

Symptomatology

For open dysraphism, symptoms are referable to leaking of CSF and an exposed spinal cord. If CSF leakage is not attended properly, meningitis may ensue adding significantly to its morbidity. The extent of sensorimotor deficits would depend on the level of the meningocoele - the higher the worse. It may be high at the cervical level in about 3.9% of spinal dysraphism.[9] In severe cases, sphincter dysfunction may present with rectal prolapse. Chiari malformation presentation varies from major brainstem and lower cranial nerve deficits to fairly asymptomatic and insidious.

With the advent of non-invasive investigations and clinical vigilance, diagnosis is generally made early even for occult lesions. At the time of diagnosis, these

patients are usually neurologically intact only to deteriorate later on. Reasons are manifold. As the child grows, disproportionate lengthening between the vertebral column and spinal cord puts the conus under increasing tension precipitating neurological dysfunction.[6] Activities that result in an abrupt flexion of the spine are also implicated as a cause of trauma to the spinal cord.[1] Repeated mechanical shock transmitted via the subcutaneous tissue up the spinal cord, minor as they may be individually, would in the long run pose another cause for neurological deterioration as the patient grows into adulthood.[2,8]

Common complaints include pain, sensorimotor deficits and sphincter dysfunction. Pain is usually at the lower back. It may be associated with posture that leads to stretching of the spinal cord or even on prolonged lying down.

In transitional lipoma, probably because of the extensive involvement, motor deficit is more common. Loss of motor neurons results in smaller calves or buttocks. If passed un-noticed, abnormal gait as the child starts to walk and run would alert parents of the problem.

Bladder dysfunction is even subtler for infants. Delayed toilet training is the usual story but tends to be ignored. The author has patients with sphincter problems all their life until the age of 12 when the diagnosis of lipoma was finally made.

Response to treatment differs among the three groups of symptoms. Most rewarding is pain which usually subsides soon after untethering procedures. Motor and gait improvement is also possible but takes time. Chronic bladder and sphincter problems, however, respond poorly to untethering. Long-term urological attention is likely.

Diagnosis

Decades ago, an open dysraphic lesion was diagnosed only when the baby was born and it invariably came as a surprise to the obstetrician, neurosurgeon and of course their parents. Nowadays with thorough antenatal care and investigation, diagnosis could be made early in pregnancy with ultrasound. Counselling could be done with parents and for lesions carrying good prognosis - small caudal lesions without other teratogenicity - the pregnancy is advised to continue and further study can be done with MR imaging. (Fig. 5) Precaution is then taken to assure an intact lesion at birth until surgical closure.

For occult lesions, suspicious stigmata along the midline noted after birth would usually lead to further investigations. Radiological finding of bifid spinal laminae due to the intervening tissues gives this group the name spina bifida occulta. MRI is now a standard investigation demonstrating clearly the nature, the level and extent of tethering.

Management

Proper management of children with spinal dysraphism calls for a multi-disciplinary approach. Clinicians have to be aware of how subtle the presenting signs and symptoms may be and follow with appropriate investigations and bring them to the attention of neurosurgeons. The pathology demands also the expertise of paediatric urologists as well as orthopaedic surgeons. Physiotherapist and occupational therapists also have an important role in their rehabilitation. They need to be followed up till their late teens to rule out possible retethering.

Open Dysraphsim

With the possibility of prenatal diagnosis of open dysraphism, attempts have been made in a few centres in the world to close the defect in utero with the belief that associated hydrocephalus and Chiari malformation could be avoided at birth. Success has been reported. Long-term results and benefit of such an approach are still to be seen.[3]

The aim of surgery in these cases is to free the placode from the surrounding abnormal skin and reduce it into the spinal canal which is closed in a watertight fashion. Epidermal tissue has to be meticulously trimmed from the placode to avoid the late complication of dermoid formation. The major early complication of this procedure is wound healing problem with CSF leakage and meningitis. This could be avoided with multiple layer closure or even double-breast closure utilising lumbosacral fascia on the sides. For huge defect, a proper skin closure may be difficult if not impossible without requiring rotational flap from the sides. Worst of these cases are those with kyphotic deformity of the spine. Expertise of orthopaedic and plastic surgeons may then be essential.

Equally important as the surgery itself is postoperative nursing care. The main wound has to be protected with light gauze and transparent waterproof dressing. It has been a standard protocol for the author to put the patient in the prone position for the first few days until good union is ascertained. In the prone position, excreta will flow forward leaving the dressing and wound unscathed diminishing the possibility of infection.

In unexpected cases in which the cyst is ruptured and CSF already leaking, antibiotic coverage and delayed closure is preferred.

Patients should be monitored with CT for hydrocephalus. Ventricular shunt could be done, usually at a separate setting from closure of the spinal defect.

Occult Dysraphism

Whether surgery is indicated for asymptomatic cases has been debatable.[4] But with a better understanding of the pathophysiology and natural course of the disease, the contention is gradually coming to an end. Total or near-total resection of a lipoma while the patient is just a few months old with meticulous microsurgical technique as advocated by Dachling Pang has been shown to lead to a better long-term outcome.[7] Surgery should therefore be offered to asymptomatic cases at the time of diagnosis. There is little doubt that without surgery, in the following years, symptoms would appear and surgery then would impose additional difficulties as it would then be a much bigger operation

with a task to remove a bigger piece of lipoma before the cord is well freed. Recovery from an advanced deficit is always more difficult and incomplete. After thorough neuroimaging and urological work up, surgery can be done between 3 to 6 months old. In a retrospective study done in Tuen Mun Hospital, better postoperative results are obtained for those done early as opposed to those diagnosed late and surgery done after 4 year old.[10]

The prime target is to free the whole spinal cord from any tethering within the dura. (Fig. 6,7) This usually means that the lipoma, which spans between the dorsal root entry zones on the sides, has to be completely excised along a silvery plain between the lipoma and the neuroplacode. Cutting along the precise plain with scissors is a far better way than using the surgical aspirator or lasers. At the end of excision, in transitional lipoma in particular, as the placode is flattened by the adherent lipoma, it has to be neurulised - the placode stitched together forming a cord like structure again. This is an effective way to avoid adhesion and thus retethering.

Proper meticulous watertight closure follows. Unlike open lesions, skin closure is seldom a problem. It is not necessary to pay special effort to excise extradural aberrant tissue except skin sinus and appendages. Cutaneous haemangiomas usually vanish with time. Similar nursing precaution is to be taken after surgery as in open cases.

For the rare incidence of split cord malformation (SCM), the principle is similar in that the midline septum in between the 2 hemicords has to be excised totally. For Type I SCM in which each hemicord has its own dural sheath, the 2 dural sheaths have to be opened and stitched as one alleviating the possibility of dural tethering later on.

Complication

Wound healing problems leading to CSF leakage and meningitis remain the most feared complication. However with appropriate precautions, such occurrence can be avoided totally. With effective multiple layer closure, subcutaneous pseudomeningocoele is noted in about 5% of cases. With time, most of them would subside spontaneously. Additional deficit is rare after surgery.[10]

Results

For symptomatic cases, pain is the most ready to improve irrespective of the pathology - be it just a fatty tight filum or a transitional lipoma.

Sensorimotor deficit comes next in terms of responsiveness. Acute recent deficit recovers better than those old burnt out deficits with orthopaedic deformities.

Bladder dysfunction also benefit apart from those atonic bladders with large residual volume which probably would require intermittent catheterisation indefinitely.

The general principle holds here in that the more advanced the disease is on presentation, the worse they would fair despite the best treatment.

Conclusion

Spinal dysraphism is one of the most common causes of disability in infants and children. It can be substantially reduced with folate and better dietary habits. Together with better antenatal care, the incidence is on the decline for the open type. However, occult lesions still require concerted efforts of clinicians in staying vigilant to make the right diagnosis, treatment instituted promptly with surgical expertise and followed with multi-disciplinary approach to get the best result.

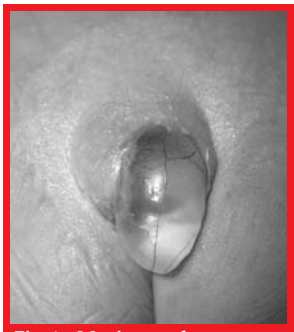

Fig 1a. Meningocoele

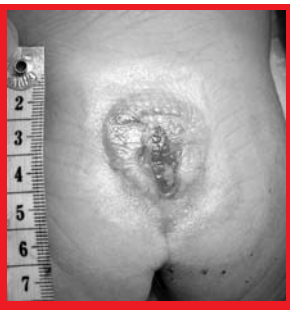

Fig 1b. Myelomeningocoele

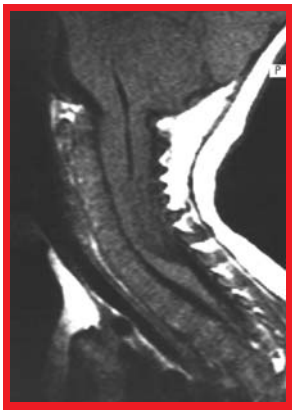

Fig 2. Chiari malformation type II in a child with spinal dysraphism

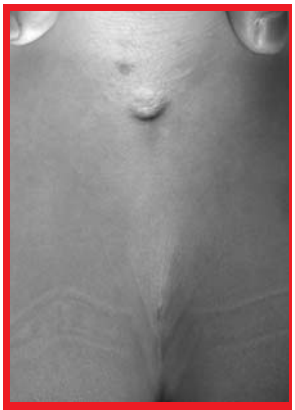

Fig 3. Cutaneous stigmata a Skin dimple cutaneous tethering

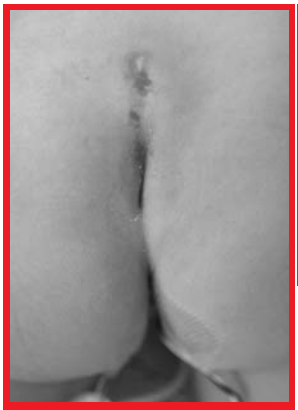

Fig 3b. Midline cleft with haemangioma

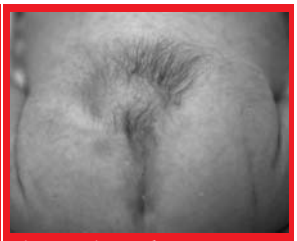

Fig 3c. Hairy patch

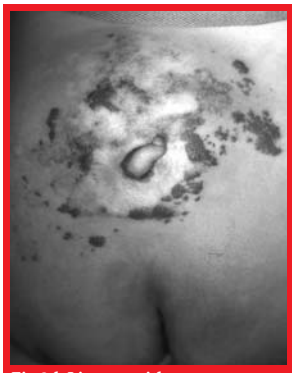

Fig 3d Lipoma with haemanagioma and skin tag

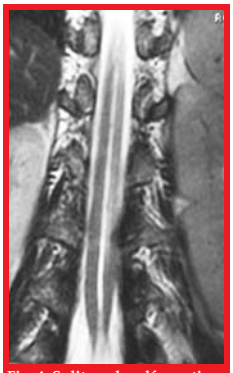

Fig 4. Split cord malformation in a 38 year old woman

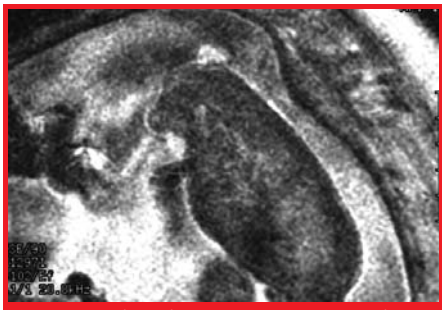

Fig 5. Maternal MR demonstrating a meningocoele at the sacral level

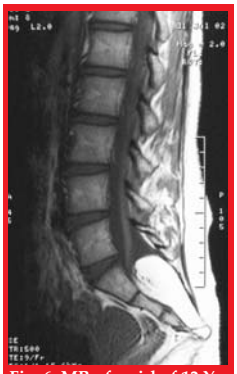

Fig 6. MR of a girl of 12 Y with terminal lipoma - Before operation

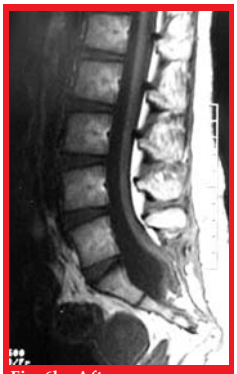

Fig 6b. After surgery

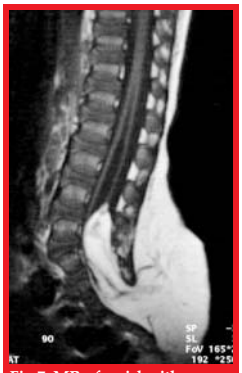

Fig 7. MR of a girl with transitional lipoma - Before surgery

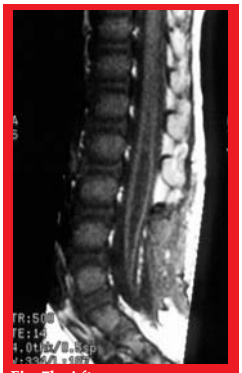

Fig 7b. After surgery

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Spinal Dysraphism" by Dr. Dawson Fong 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 sheet via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 December 2006. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

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

1. **With the advent of sophisticated neuroimaging modalities, the incidence of open spinal dysraphism in neonates is on an increase.**
2. **Folate as a vitamin supplement during pregnancy is not good enough to prevent dysraphism in the embryo.**
3. **The more rostral a dysraphic defect is, the more extensive is expected of the morbidity.**
4. **Children with cutaneous stigmata along the midline need to be investigated with spinal MR irrespective of whether there is demonstrable neurological deficit.**

5. Severity and extent of occult dysraphic defect is directly proportional to the neurological deficit at birth.

6. Hydrocephalus is commonly associated with occult dysraphism.

7. For a baby born with myelomeningocoele, the defect should be well dressed to prevent CSF leak.

8. Surgery can be delayed until the baby is older and stronger.

9. As the patient with an occult lesion grows and gets taller, new symptoms would arise from repeated mechanical damage as well as a worsening tethering on the spinal cord.

10. There is no difference when to operate on an occult dysraphism in terms of technical difficulty and patient outcome.

ANSWER SHEET FOR DECEMBER 2006

Please return the completed answer sheet to the Federation Secretariat on or before 31 December 2006 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Spinal Dysraphism

Dr. Dawson Fong MBBS(HK), FRCS(Edin), FCSHK, FHKAM(Surgery)
Chief of Service and Consultant Neurosurgeon, Department of Neurosurgery, NT West Cluster

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

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

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Others Membership No. (please indicate): _____

Contact Tel No.:_____

Answers to November 2006 issue

Early Diagnosis of Spondyloarthropathies

1. b 2. c 3. a 4. b 5. d 6. a 7. c 8. c 9. d 10. b

Chronic Pain: Neurosurgical Perspectives

Dr. Kwan-ngai Hung MBBS(HK), FRCS(Edin), FCSHK, FHKAM(Surgery)

Consultant Neurosurgeon, Chief of Service, Department of Neurosurgery, Queen Mary Hospital

Dr. Kwan-ngai Hung

Surgery is usually used as a last resort in pain management, after failure of medical treatment. Neurosurgical procedures involve in pain management can be divided into two groups, ablative and augmentative.

Ablative procedures

These procedures aim to reduce the source of pain production or disconnect the pain stimulation along the sensory pathways. Excision of neuromas is a common procedure to reduce pain after peripheral nerve injury. Spinal cord destructive procedures, namely cordotomy had been previous used as the treatment for patients with terminal malignancy, with complications including urinary retention, hemiparesis, and unmasking of contralateral pain.[1] Other complications includes injury to the spinorecticular tract in the cervical cord, which affect breathing. The nonpermanent nature of the pain relief (which may be lost after 1-2 years) or the possibility of inducing deafferentation pain (if the lesion affects the nociceptive pathways) makes destructive procedures not suitable for noncancerous chronic pain.[2] Another ablative procedure includes hypophysectomy, which significantly reduces pain in terminal cancer patients. Previously, it was done via transsphenoidal route under general anaesthesia. Currently radiosurgery can be used for hypophysectomy to reduce the risk of open operation and general anaesthesia.

Augmentative procedures

With the advance in computer and electronics technology, mini stimulator is now available for implantation. The use of stimulation of dorsal column of spinal cord was first started in the 1960s,[3] it was not popular because of absence of appropriate hardware for chronic stimulation. The proposed explanation of the mechanism is based on the gate control theory of pain, which states that stimulation of large A fibres can reduce input signals from small C fibres which reduce the subsequent pain transmission to the central nervous system.[4] Nowadays, dorsal column stimulation is widely used for neuropathic pain (due to abnormal firing of the neurons despite the irritating source may be absent) of spinal origin that is refractory to drug treatment. Patients selected for implantation procedures will have a trial implantation under local or general anaesthesia with either percutaneous fluoroscopic guidance or open laminectomy to implant the trial lead. The trial lead will be externalised and connected to a temporary stimulator. Various combinations of lead contact, polarity, stimulation current, frequency and pulse width can be adjusted to obtain a good cover of the painful area. The test will last for a few days. If there is satisfactory response, the lead and the pacemaker will be implanted subcutaneously. Otherwise the lead will be removed. Other indications for dorsal column stimulation include pain reduction in angina pectoris and ischaemic limbs are still controversial. [5]

For nocioceptive pain, which is due to continuous pain stimulation from an irritating source, narcotics are the drugs of choice. In the presence of the blood brain barrier, the amount of drugs that reaches the central nervous system (CNS) is much reduced. Direct admission of narcotics to the CNS decreases the dose required and hence reduces systemic side effects. However, it requires a meticulous control of the rate of delivery, as overdose will result in respiratory suppression and mortality. Currently the availability of programmable pumps allows continuous infusion with flow rate control. The same apparatus can be used for baclofen infusion to reduce spasticity and pain related to muscle spasm.

Deep brain stimulation had been used for pain reduction and various targets had been proposed. Periventricular gray (PVG) and periaqueductal gray PAG) were the targets for nociceptive pain, while ventropostero-lateral nucleus (VPL) and ventralis posteromedialis (VPM) in the thalamus is used for bodily pain and facial pain respectively. For patients with mixed pain, implantation of various targets simultaneously was tried with promising results. [6]

Motor cortex stimulation (MCS) was first reported in 1991.[7] It was used mainly for thalamic pain, secondary to stroke. It was also useful in central facial pain, with outcomes as high as 83% pain reduction in 77% of patients. [8]

In summary, with the advance in computer and electronics technology, augmentative procedures have replaced ablative procedures in the management of patients with chronic pain.

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Neuro-endovascular Therapy of Carotid-Cavernous Fistula

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Department of Neurosurgery, Queen Elizabeth Hospital

Dr. Kin-ming Cheng

Introduction

Carotid-cavernous fistula (CCF) is an abnormal arteriovenous communication in the cavernous sinus. The cavernous sinus is a venous cavern between layers of the dura of the skull base (Fig 1). It receives venous drainage of the eye and the brain via the ophthalmic veins and the superficial middle cerebral vein. There are also venous communications with the opposite cavernous sinus, the clival venous plexus and the transverse sinus. CCF was first described by Travers (1809) who recognised pulsating exophthalmos to be due to an arterio-venous shunt and treated it with common carotid ligation.[1] Although surgical ligation often produced initial good results, collateralisation from the arterial branches of the external and internal carotid artery produced high recurrence rate. Later, Brooks (1931) surgically opened the internal carotid artery in the neck and floated a strip of muscle into the fistula and Gardner (1932) described internal carotid artery ligation as a method of treatment.[1] It was Serbinenko (1974) who described the use of detachable balloon technique to obliterate the fistula and leave the carotid artery patent.[2] In the modern era, neuroendovascular therapy offers a safer and more effective treatment for CCF. [3, 4, 5]

Aetiology

Carotid-cavernous fistula can be classified into traumatic and spontaneous types. The traumatic type is caused by severe head injury after high velocity traffic accident, major skull base fracture or penetrating wound through the orbit. The spontaneous type can be congenital, secondary to rupture of carotid aneurysm into the cavernous sinus or acquired with multiple arterial branches of the carotid arteries that shunt into the cavernous sinus. The later type is the commonest and is usually called dural CCF. In Hong Kong, traumatic cause is uncommon and most are spontaneous in origin. In Queen Elizabeth Hospital (1997-2005), a total of 80 patients with CCF were treated. Seventy-six (95%) cases are spontaneous and only 4 cases (4%) are of the traumatic type.

Pathophysiology

The abnormal arteriovenous shunt creates a high venous pressure in the cavernous sinus that impairs venous drainage of the ophthalmic veins and causes eye congestion and increase in intraocular pressure (Fig 2). The increase in cavernous sinus pressure also causes pressure effects on the third, fourth and sixth cranial nerves that resulted in ophalmoplegia. In addition, CCF can create back pressure in the cerebral veins that can lead to intracerebral haemorrhage (Fig 3).

Clinical features

Carotid-cavernous fistula usually presents with chemosis, proptosis, diplopia and visual impairment of the eye. Rarely, CCF can cause cerebral dysfunction (hemiplegia, dysphasia, etc). The clinical presentation of traumatic CCF is acute but some of the cases can present one week after the injury. Spontaneous CCF is usually insidious in onset. Physical signs include eye bruit, pulsatile exophthalmos, cranial nerve palsy (3rd, 4th, 6th), and impaired vision.

Investigations

Carotid-cavernous fistula can be diagnosed with imaging studies. CT scan with contrast may show skull base fractures, a dilated superior ophthalmic vein and venous engorgement in the region of the cavernous sinus. MRI and MR angiography are also useful tools for screening and follow-up of the patients. Cerebral angiography is a more invasive form of imaging study which provides the best diagnostic study for CCF. Selective internal and external carotid angiograms are essential to define the anatomy and develop the treatment plan.

Treatment

In the old days, CCF was treated with surgical operations with significant morbidity and mortality, In the modern era, neuroendovascular therapy offers a safe and effective treatment for patients with CCF and has replaced open surgery as the treatment of choice.

Most of the endovascular procedures for CCF can be done under local anaesthesia. The procedure starts with arterial or venous puncture in the groin region. Catheters and co-axial microcatheters are introduced via the femoral route to the intracranial circulation. The endovascular approaches can be transarterial or transvenous. The embolic materials include polyvinyl alcohol particles, histoacryl glue, platinum coils and Guglielmi detachable coils. Although technically more difficult to perform, the transvenous approach is safer and more effective than the transarterial approach and is recommended in most cases of dural CCF. The

transvenous embolisation routes includes the inferior petrosal sinus, inter-carvernous sinus, superior ophthalmic vein (Fig 4, 5, 6) and rarely, cortical vein, superior petrosal sinus and pterygoid venous plexus. Clinical cure can be achieved in more than 90% of the patients and the procedure-related morbidity and mortality is very low.[5, 6]

Conclusion

Carotid-cavernous fistula is a well-known disease entity that can be encountered in daily clinical practice. CCF can cause visual and neurological deficits when the condition is left untreated. Diagnosis of CCF can be made by clinical examination and imaging studies. Neuroendovascular therapy is safe and effective and is the treatment of choice in CCF.

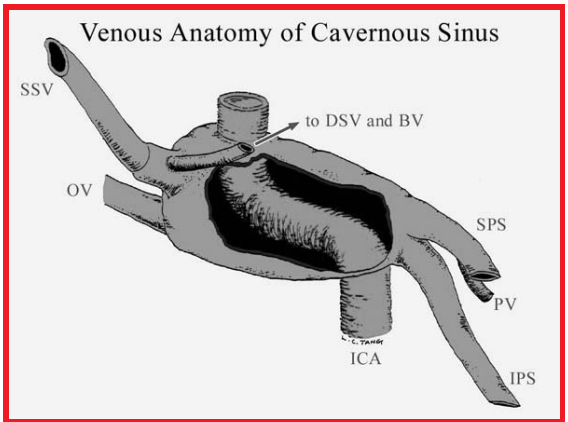

Figure 1. Anatomical diagram showing the venous anatomy of cavernous sinus. SSV = superficial sylvian vein, OV = ophthalmic vein, DSV = deep sylvian vein, BV = basal vein of Rosenphal, ICA = internal carotid artery, SPS = superior petrosal sinus, PV = petrosal vein, IPS = inferior petrosal sinus.

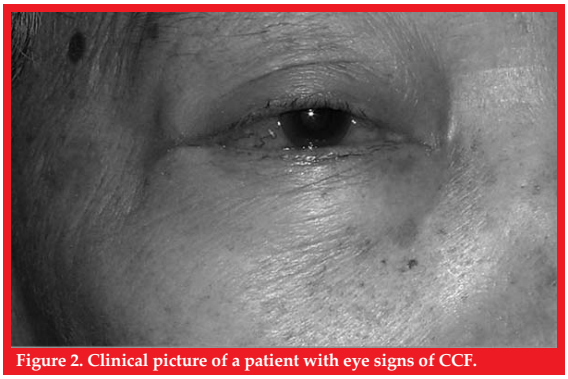

Figure 2. Clinical picture of a patient with eye signs of CCF.

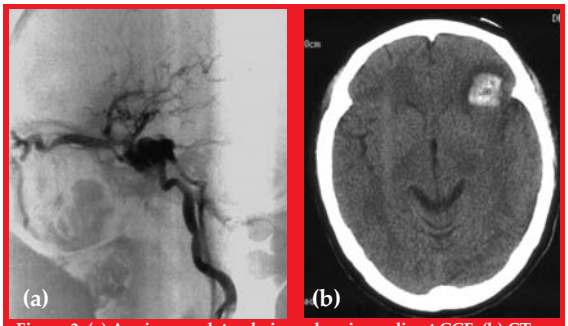

Figure 3. (a) Angiogram, lateral view, showing a direct CCF. (b) CT scan, axial view, showing intracerebral haemorrhage

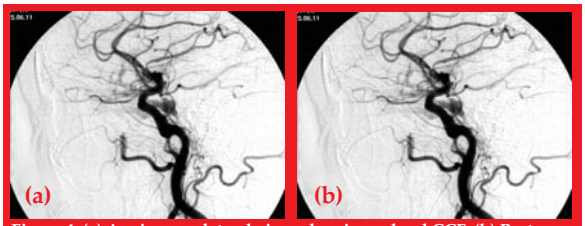

Figure 4. (a) Angiogram, lateral view, showing a dural CCF. (b) Post-embolisation angiogram, lateral view, showing complete fistula occlusion with coils via inferior petrosal sinus approach.

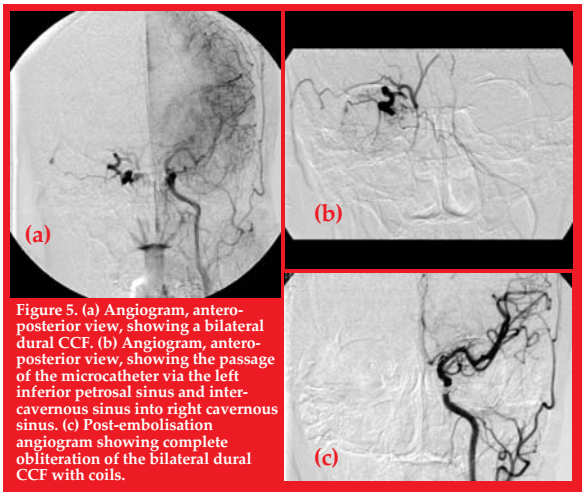

Figure 5. (a) Angiogram, antero-posterior view, showing a bilateral dural CCF. (b) Angiogram, antero-posterior view, showing the passage of the microcatheter via the left inferior petrosal sinus and inter-cavernous sinus into right cavernous sinus. (c) Post-embolisation angiogram showing complete obliteration of the bilateral dural CCF with coils.

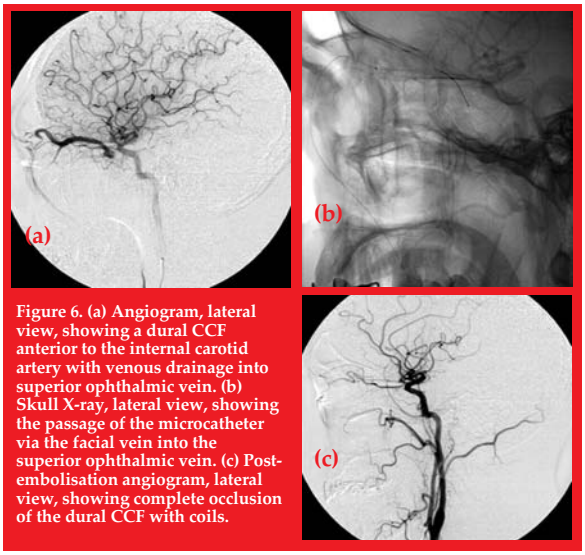

Figure 6. (a) Angiogram, lateral view, showing a dural CCF anterior to the internal carotid artery with venous drainage into superior ophthalmic vein. (b) Skull X-ray, lateral view, showing the passage of the microcatheter via the facial vein into the superior ophthalmic vein. (c) Post-embolisation angiogram, lateral view, showing complete occlusion of the dural CCF with coils.

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Post Concussional Syndrome

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Introduction

Post Concussional Syndrome is a common disease entity encountered in head injury patients. Sometimes the injury impact can be very trivial, and patient is thought of being malingering. It has a wide spectrum of presentations. A correct diagnosis is important as it has significant impact in medico-legal aspect especially in work compensation.

History

The understanding of Post Concussional Syndrome evolved from a pure "neurosis", or "malingering" to a organic disorder. The first one to describe the syndrome was by Boyer in 1822.

John Erichsen, Professor of Surgery at London University, described "Railway spine" for the railway injured worker in 1866. He stated that this minor injury could result in severe disabilities with "Molecular disarrangement or anaemia of the spinal cord"

Miller in 1961 studied 200 patients (involved in compensation). At least 47 of them showed features of "compensation neurosis", which have features of arriving late, accompanied by relative who take the active role, unshakable conviction of unfitness for work and absolute refusal to admit any degree of symptomatic improvement.

But Guthkelch (1980) studied 398 patients involved in compensation. Only 6.8% got "accident neurosis", with "Bizarre and inconsistent complaints, exaggeration of length of initial unconsciousness, and attention seeking behaviours".

Fee and Rutherford (1988) found 39% of their patients were symptomatic at the time of settlement and 34% were still symptomatic one year after.

Definition

The word concussion is derived from the Latin concussus, which means "to shake violently."

Previously "loss of consciousness" was thought to be necessary.

The Congress of Neurological Surgeons in 1966 gave what still stands as our best succinct definition of concussion: "an immediate and transient impairment of neural function such as an alteration of consciousness, disturbance of vision, equilibrium, and other similar symptoms"

Concussion has been categorised into three types or grades for clinical purposes. Several classification schemes have been proposed, with three currently in widespread use.

Table 1. Grading for brain concussion

	Mild Grade 1	Moderate Grade 2	Severe Grade 3
Cantu	No LOC or PTA < 30 min	LOC < 5 min or PTA of 30 min-24 hr	LOC > 5 min or PTA > 24 hr
Torg	No LOC. No amnesia	LOC few minute, PTA or retrograde amnesia	LOC (coma), confusion with amnesia
Colorado	No LOC Confusion without amnesia	No LOC Confusion with amnesia	LOC

LOC: loss of consciousness PTA: post traumatic amnesia

Table 2. ICD 9 classification (International Classification of Disease)

850.0	No LOC	Mental confusion or disorientation
850.1	Brief LOC	LOC< 1 hour
850.2	Moderate LOC	LOC 1-24 hours
850.3	Prolonged LOC with recovery	LOC > 24 hrs with complete recovery
850.4	Prolonged LOC	Incomplete recovery
850.5	LOC of unknown duration	
850.9	Unspecified	

Post Concussional Syndrome is a collection of symptoms and signs including headache, dizziness, vertigo, tinnitus, hearing loss ,blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, diminished taste and smell, irritability, anxiety, depression, personality change, fatigue, sleep disturbances, decreased libido, decreased appetite, memory dysfunction, impaired concentration and attention, slowing of reaction time and information processing. 70-85 % head injury admissions belong to

minor ones. 15 to 30% of them will have post-traumatic symptoms.

Headache (46%), dizziness (14%), amnesia (13%) and weakness (10%) are the most common presentations, which usually will improve with time.

Pathology

Concussed neurons can have increase in extracellar K^+, increase in glucose utilisation, possibly mediated by glutamate, to correct the K^+ concentration.

There is increase in metabolic rate but decrease in cerebral flow, which is shown to be present within several days after injury

Transient cerebral ischaemia, oedaema, widespread neuronal depolarisation from release of acetylcholine, and the shearing of neurons and nerve fibres causing alterations of mental status

Other mediators: Arachidonic acid cascade, catecholamine, cytokines, excitatory amino acid, Ca^{2+}, O^2 free radical, PAF, etc

There may be even white matter changes in the brainstem, representing axonal tearing secondary to rapid acceleration or deceleration, or to head impact movements.

The brainstem is thought to be especially involved in patients who have sustained a loss of consciousness. Actually diffuse axonal injury can also occur in patients with mild head injury.

Other studies showed trauma induced Hippocampus necrosis which can occur even in mild head injuries.

Investigation

CT and MRI are the main radiological tests. CT brain is usually normal. 9.4% of patients with GCS 15 gets positive findings. 0.3% needs surgery. MRI may show evidence of diffuse axonal injury in 15 to 30% of patients with normal head CT findings after mild head injury. These lesions may represent the pathologic substrate underlying the Post Concussion Syndrome.

Other tests, including Electroencephalography (EEG), Brainstem Auditory Evoked Potential (BAEP) and even Single Photon Emission Computerized Tomography (SPECT) are used in helping to make the diagnosis, but the significance of the tests was controversial.

Neuropsychology

Besides imaging tests, cognitive function assessment is important in assessment of Post Concussional Syndrome.

Areas of interest are: 1. Premorbid state 2.Memory 3.Intelligence 4.Language 5.Visual-spatial perception 6.Attention-concentration 7.Information-processing 8.Executive functioning

Some of the deficits that can be detected include: reduction in information processing speed, reduction in attention, prolonged reaction time and impaired memory for new information.

Symptoms and cognitive impairment usually show marked improvement within 6 months, up to one year, but residual deficits can still persist.

If there are prolonged deficits, without any improvement, other causes which maintain the syndrome must be considered.

It is important to assess pre-morbid conditions which may prolong the symptoms:

- Psychiatric conditions: depression ,personality, anxiety, recent life event
- Lower socioeconomic state, unemployment, younger age, female, lower education
- Alcoholism, drug abuse.
- Multiple trauma
- Environment: self-prediction of head injury symptoms

Management

Take a careful history and physical examination including the incidence, mechanism of injury, any loss of consciousness, post traumatic amnesia (PTA), using GOAT (table 3), any witness, current symptoms and make detailed records, both in-hospital and outpatient. The longer the PTA, the more significant of the head injury.

Table 3 The Galveston Orientation and Amnesia Test (GOAT)

Description	Error points
1. What is your name? (2) When were you born? (4) Where do you live? (4)	
2. Where are you now? (5) (City) Hospital(5) (unnecessary to state name of hospital)	
3. On what date were you admitted to this hospital?(5) How did you get here? (5)	
4. What is the first event you can remember after the injury? (5) Can you describe in detail (e.g. date , time, companions) the first event you can recall after the injury? (5)	
5. Can you describe the last event you recall before the accident? (5) Can you describe in detail (e.g. date , time, companions) the last event you can recall before the injury? (5)	
6. What time is it now? (1 for each half hour removed from current time to a maximum of 5 points)	
7. What day of the week is it? (1 for each day removed from the correct one)	
8. What day of the month is it? (1 for each day removed from the correct one to a maximum of 5 points)	
9. What is the month? (5 for each month removed from the correct one to a maximum of 15 points)	
10. What is the year? (10 for each year removed from the correct one to a maximum of 30 points) Total error points Total score (100 minus total error points)	

Necessary investigations should be done to rule out organic lesions, observe the time course of the symptoms. Social history is an important part of assessment.

We have to refer patients to psychologists early for cognitive function assessment, get a baseline, with serial follow up tests, and to psychiatrists if malingering or psychiatric illness is suspected.

We have to know the validity of all the investigations, and limitations of the psychological assessments.

Treatment is symptomatic (medication to reduce headache and dizziness), psychotherapy, and education.

Conclusion

Post Concussional Syndrome is common especially in minor head injury patients. It is a self limiting disease and usually it will improve gradually. After ruling out organic lesions by imaging, a detailed psychological assessment is necessary to get the baseline and progress, and to find out factors that may prolong the recovery. It is inappropriate to label patient as "malingering" or "asking for compensation" before all these are done.

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Minimally Invasive Spine Surgery

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Introduction

Problems of the spine encompass a wide range of disorders including those of degenerative, developmental, neoplastic, and traumatic nature. Spine surgery usually gives the impression of being a major surgery with prolonged recovery. However, in recent years surgery of the spine, as with other branches of surgery, has evolved along the lines of minimal invasiveness and problems that were dealt with by open surgery are now being treated with minimally invasive techniques. In this article, several minimally invasive techniques in the treatment of spine diseases will be discussed.

Biomechanics of the spine

It is important to understand the basis of spinal stability to appreciate the importance of preserving as much anatomical structure as possible. Spinal stability has always been difficult to define but for our purpose we can adopt the definition from White and Panjabi[1] defined as the ability of the spinal column to withstand physiological stress. Different concepts have been proposed to describe the biomechanical basis of spinal stability. The two column theory can provide a useful model for our discussion.

The vertebral body, intervertebral disc, and connecting ligaments comprise the anterior column while the posterior elements including the facets, the lamina and the posterior ligamentous complex constitute the posterior column. Generally speaking, the anterior column supports axial load of the body while the posterior column serves as a tension band. The two columns complement each other in terms of function, i.e. one column cannot function adequately without the other.

Posterior approach

The traditional posterior approach to the spinal canal is laminectomy. Laminectomy involves the removal of the bony lamina and spinous processes, ligamentum flavum, as well as the interspinous and supraspinous ligament. The removal of these structures damages the tension band mechanism and significantly weakens the posterior column. This can lead to instability and post-laminectomy deformity has been reported[2,3].

Apart from the aforementioned structures, the paraspinal muscles also play an important role in stabilisation of the spine. Stripping of the paraspinal muscles in performing laminectomy may lead to muscle atrophy[4] and can contribute to instability. Also the wide exposure of the spinal canal from a laminectomy may contribute to epidural scarring.

Lumbar disc disease

Lumbar disc herniations typically present with back and leg pain which tends to be worse with physical activities. Tension signs can be positive on physical examination. MRI will reveal the diagnosis.

Different approaches have been developed for treatment of disc herniations in the lumbar spine. The standard procedure of laminectomy or fenestration and diskectomy via a midline approach involves destruction of the posterior elements as described above. Therefore performing microdiskectomy with the aid of a microscope can help to minimise tissue trauma and laminectomy is not required to remove disc fragments. By using a paramedian approach[5] the midline ligamentous structure can be preserved making deformity less likely. Less trauma to the paraspinal muscles also minimise postoperative muscle atrophy. Other minimally invasive techniques using endoscopic technology has also been developed [6,7].

Microsurgical decompression of lumbar spinal stenosis

Patients with degenerative spinal stenosis usually present with back, buttock, thigh, and calf pain. Neurogenic claudication is another feature with this entity. Again the diagnosis is made with MRI.

Using a similar paramedian approach, spinal stenosis caused by hypertrophy of the ligamentum flavum and facet joints can be decompressed using minimally invasive microscopic techniques[8] Fig. 1. Undercutting of the base of the spinous process together with tilting of the operative table allows access to the contralateral spinal canal and adequate decompression without disturbing much of the posterior structures. Hypertrophied ligamentum flavum and facet joints can be resected with rongeurs and high speed drill.

Minimally invasive approach to extramedullary spinal tumours

Extramedullary spinal tumours often present with

neurological deficits as a result of neural compression. Alternatively it can present with pain. The commonest extramedullary tumour in the spine is neurilemmoma followed by meningioma.

A relatively new development is the development of minimally invasive approach to treatment of intradural extramedullary spinal tumours by using the minimally invasive technique[9] Fig. 2. Traditionally the laminectomy is required for exposure for treatment of these tumours which requires opening of the dura. Using the same principle as outlined above, these tumours can be approached via a paramedian approach with undercutting of the spinous process. One major concern is the possibility of CSF leakage after dural opening. Our experience shows that primary closure of the dura is possible with this technique and where this is not possible with dural defects, synthetic onlay dural substitute can be used. No new neurological deficit or postoperative CSF leak has been observed so far with this technique. Whether this technique can be used with intramedullary tumours or large extramedullary tumours remains to be seen. However, this technique can be converted to the traditional approach if needed without much difficulty.

Minimally invasive lumbar spine instrumentation

The standard approach for fixation of the lumbar spine with instrumentation involves exposure of the posterior elements of the lumbar spine. Techniques have been developed to allow percutaneous placement of pedicle screws and rods to provide rigid fixation from posteriorly[10]. Tissue trauma associated with this approach is considerably less than the conventional approach.

Microscopic approach to the anterior aspect of the lumbar spine has also been developed to allow interbody fusion to be done along natural tissue plane with minimal access[11]. Interbody fusion can also be accomplished by Posterior Lumbar Interbody Fusion (PLIF)[12], and Transforaminal Lumbar Interbody Fusion (TLIF)[13] where anterior approach is not necessary. In PLIF and TLIF the disc space is approached posteriorly after resecting the medial and lateral part of the facet joints respectively. Interbody fusion cages can then be inserted. Combined with posterior instrumentation this will provide fixation of both anterior and posterior columns of the spine.

Conclusion

The development of minimally invasive spine surgery marks a new era in the treatment of spinal problems. With more preservation of normal structures, functional outcome can hopefully be improved. It is, however, important to note that traditional approach forms the basis of spine surgery and has been proven reliable and remains so. It is perhaps more important to realise the limitations in the problems that can be treated by minimally invasive techniques. Careful preoperative planning is imperative and familiarity with spinal anatomy is a must before attempting minimally invasive technique. One should also note the learning curve associated with this technique and be prepared to convert to traditional approach when necessary.

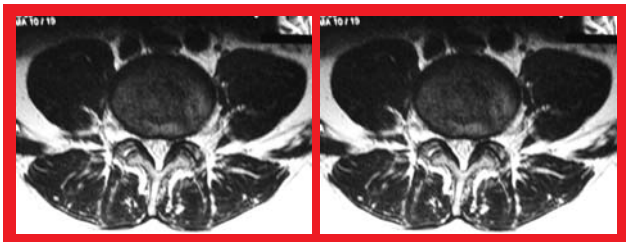

Fig. 1 Decompression of spinal stenosis by microsurgical method. Note the preservation of midline structures and the minimal effect on paraspinal musculature in postoperative image.

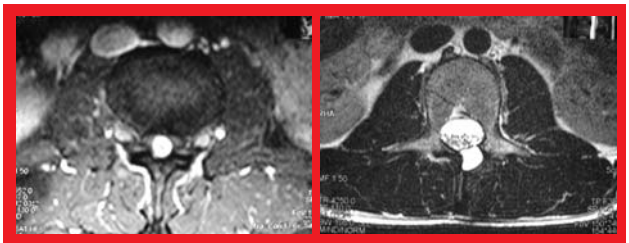

Fig. 2 Minimally invasive approach to extramedullary intradural tumours. Note in the postoperative image the interval of access.

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Carotid Stenosis

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Introduction

Carotid artery stenoses, particularly involving the origin of the internal carotid artery, are most commonly due to atherosclerosis. One of the common aetiological factors in Hong Kong is radiation therapy to the head and neck region, especially in nasopharyngeal carcinoma patients. Atherosclerosis is considered as an inflammatory disease. The process of atherogenesis consists of migration and proliferation of smooth muscle, arterial wall thickening, accumulation of foam cells, formation of fibrous plaque and fibrous cap and haemorrhage and thrombosis within the plaque. Endothelial dysfunction and platelet activation play a key role in the initiation of the process. Eventually, ulceration of plaque may occur and lead to thrombosis and distal embolism. The presence of an ulcerated plaque is associated with a stroke rate of 7.5% per year. The nature of the atherosclerotic plaque and the mechanisms by which it results in symptoms must be kept in mind when assessing the approach to carotid artery stenosis.

Stroke is the third leading cause of death in Hong Kong and accounts for more than 20 deaths per 100 000 population per year.[1] Carotid stenosis accounts for about 20% of ischaemic strokes and should be managed efficiently to minimise the incidence of stroke. Unfortunately, only about 15% of strokes are preceded by transient ischaemic attacks (TIAs).[2,3] Until recently, North American guidelines recommended that assessment and investigation be completed within one week of a TIA and British guidelines recommended assessment within 2 weeks.[4,5] A screening study carried out between 1995 to 1997 in Hong Kong showed that severe carotid stenosis was detected in 37.7% of patients with cerebrovascular disease, 24.5% of patients with peripheral vascular disease and 11.1% of patients with coronary artery disease.[1] Thus, the prevalence of carotid stenosis in Chinese population is not low in the patient group with atherosclerotic disease in other vascular beds. It had been suggested that screening of carotid stenosis might be considered in patients with ischaemic heart disease and irradiated nasopharyngeal carcinoma patients.

The prevalence of carotid artery stenosis in the general population is too low to justify widespread screening for this condition.[6] About 35% of patients with a carotid bruit have moderate or severe carotid stenosis. Patients found to have carotid bruit should be further evaluated.[2] However, unlike cardiac murmur, carotid bruit may be absent in patients with severe carotid stenosis and thus all symptomatic patients should be investigated radiologically irrespective of the presence or absence of carotid bruit.

Investigation

Symptoms of stroke or TIA referable to carotid territory include weakness or paralysis on the contralateral side, numbness or loss of sensation on the contralateral side, dysarthria, dysphasia, and amaurosis fugax on the ipsilateral side. If carotid stenosis is also present, it would be classified as symptomatic carotid stenosis. Patients presenting with motor weakness or speech deficit are at higher risk of subsequent strokes than patients presenting with sensory deficits or amaurosis fugax.

Carotid duplex ultrasound is the most frequent employed modality in the initial evaluation of patients with suspected carotid artery disease. Duplex ultrasound can provide an estimation of the degree of stenosis, structure and composition of plaque. With ultrasound finding of carotid stenosis of 60% or more, patient should undergo further diagnostic evaluation with non-invasive techniques such as contrast-enhanced MR angiography or CT angiography.

In terms of non-invasive investigation, a recent review suggested that contrast-enhanced MR angiography may be more sensitive (0.94, 95% CI 0.88-0.97) and specific (0.93, 95% CI 0.89-0.96) for 70-99% stenosis than Doppler ultrasound, MR angiography and CT angiography.[7] Intra-arterial angiography remains the gold standard when in doubt.

Management of symptomatic carotid stenosis

Aggressive medical therapy had been shown to reduce atherosclerotic carotid artery stenosis and prevent symptoms.[8] Antiplatelet therapy had been shown to reduce risk of fatal strokes by 16% and non-fatal strokes by 28%.[9] Lipid lowering therapy reduce risk of strokes by 25%.[10,11] Angiotensin-converting enzyme inhibitors decrease stroke rates by 32% and were shown to have slowed progression of atherosclerosis in general.[12,13,14,15] Effective management of hypertension decreased stroke rates by 28% to 40%.[16,17] Smoking cessation had been shown to decrease women's risk of strokes by 48%.[18,19]

Among patients with TIA or stroke and documented

carotid stenosis, a number of randomised trials have compared endarterectomy plus medical therapy with medical therapy alone. For patients with symptomatic atherosclerotic carotid stenosis greater than 70%, as defined by using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria, the value of carotid endarterectomy (CEA) has been clearly established from the results of 3 major prospective randomised trials: the NASCET, the European Carotid Surgery Trial (ECST), and the Veterans Affair Cooperative Study Program.[20,21,22,23,24] Among symptomatic patients with TIAs or minor strokes and high-grade carotid stenosis, each trial showed impressive and absolute risk reductions for those randomised to surgery. For those with symptomatic carotid stenosis in the moderate category (50% to 69% stenosis), the result from NASCET and ECST demonstrated significant though less impressive benefits for CEA in this moderate group compared with medical therapy. For patients with carotid stenosis less than 50%, these trials showed that there was no significant benefit of surgery

Various comorbid features altered the benefit-to-risk ratio for CEA for moderate carotid stenosis. Benefits were greater among those with more severe stenosis, those more than 75 years of age, men, patients with recent stroke and patients with hemispheric symptoms.[25] Other radiographic features found to predict better outcomes after CEA included the presence of intracranial stenosis, the absence of leukoaraiosis, and the presence of collaterals.[26,27] Benefit from surgery was greatest within 2 weeks after last ischaemic event and fell with increasing delay.[28]

We had managed over 90 carotid endarterectomies in our unit since 1996.[29] In 2002, we started to use regional anesthesia when performing carotid endarterectomies unless the patients could not tolerate, in which cases, general anaesthesia will be used. Data suggest that regional anaesthesia might reduce the need of intraoperative shunting, duration of hospital stay, perioperative stroke and myocardial infarction.[30] We perform superficial cervical plexus block along the posterior border of the sternocleidomastoid muscle with a mixture of lignocaine and ropivacaine and supplemented by light sedation with dexmedetomidine infusion. Intraoperatively, we monitor the patients by (1) clinical neurological status, (2) measuring the middle cerebral artery flow velocity by transcranial Doppler, and (3) monitoring EEG with spectral edge frequency, particularly observing any significant change before and after vascular clamping.[31] We use a transverse skin incision with extension to the back of the jaw to have better cosmetic effect. After exposing the common carotid artery, internal carotid artery and external carotid artery, a trial cross clamping of the vessels will be performed. If there was no adverse effects as shown by the monitoring, intraarterial shunt is not used. An arteriotomy is then made and the atheroma is removed from the intima. We routinely repair the arteriotomy site with a vascular patch to enhance the diameter of the vessel. The median of days of hospital stay for patients having undergone carotid endarterectomy in our unit is 3 days. The stroke risk after CEA in our cohort was 2.2% over a mean follow-up period of five years. (Figure 1a and 1b)

Data on carotid balloon angioplasty and stenting (CAS) for symptomatic patients with internal carotid artery stenosis in stroke prevention consists of a number of published case series and few randomised multicentre comparisons of CEA and CAS.[32] The Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) trial randomised 334 patients to endarterectomy under general anaesthesia or stenting with the use of an embolic-protection device under local anaesthesia, testing the hypothesis that stenting was not inferior to endarterectomy.[33] Only 30% of the study population was symptomatic. Qualified CAS operators had a periprocedural stroke, death or myocardial infarction complication rate of 4%. The primary end point of the study (the cumulative incidence of death, stroke or myocardial infarction within 30 days after intervention, or death or ipsilateral stroke between 31 days and 1 year) occurred in 20 stent patients and 32 endarterectomy patients (30-day risk, 5.8% versus 12.6%; P=0.004 for noninferiority). Most of the benefits was detected in the lower risk of myocardial infarction for the stent compared with the high-surgical risk general anaesthesia endarterectomy cases. At present, CAS has been used in selected patients instead of carotid endarterectomy in the presence of severe vascular or cardiac comorbidities or specific conditions. They may include contralateral laryngeal nerve palsy, radiation therapy to the neck, previous CEA with recurrent restenosis, high cervical internal carotid / below the clavicle common carotid lesions, severe tandem lesions, severe pulmonary disease, congestive heart failure (New York Heart Association class III/IV), known severe left ventricular dysfunction, recent myocardial infarction (>24 hours and < 4week), unstable angina and contralateral occlusion. This definition, however, is not evidence based and is not universally shared.[34] Randomised trials comparing the efficacy of CAS versus CEA in preventing strokes are ongoing in United States, Europe and Australia and 2-year outcome data should come out in 1-2 years. 30-day strokes and mortality rates of the European trials did not support the hypothesis of non-inferiority as compared to carotid endarterectomy.

Our protocol for carotid angioplasty and stenting is described as below. With respect to preprocedural therapy, adequately dosed dual antiplatelet therapy is essential. Patients would receive a combination of clopidogrel 75 mg and aspirin 160 mg for 5 days before CAS. The procedure is carried out under local anaesthesia. Continuous monitoring of pulse oximetry, blood pressure and heart rhythm is essential. Usually, the procedure is performed through a 7F or 8F right femoral arterial sheath. The role of initial diagnostic angiography is limited to the lesion side as determined by preprocedural noninvasive imaging. We obtain angiographic runs with an evaluation of lesion severity, carotid bifurcation, anatomy of common carotid artery and ipsilateral intracranial anatomy. The diagnostic catheter would then be exchanged for a guiding catheter. The tip of the guiding catheter is positioned in the distal common carotid artery. Heparin bolus of 4000 units will be administered after guiding catheter placement. Next the lesion is crossed with a 0.014-inch guidewire, usually that of an embolic protection device. The embolic protection device is deployed in the distal cervical internal carotid artery. Intravenous atropine 0.5 mg may be applied before stenting and balloon angioplasty, especially in elderly patients with heavily calcified

plaque. With the rapid exchange monorail system, stents such as Wallstent would be positioned to cover the lesion with adequate anchorage and deployed. Subsequent balloon angioplasty would be performed for optimal stent expansion. Femoral sheath would be removed when the activated clotting time has fallen below 150 seconds. Patient will be discharged with instruction to take clopidogrel 75mg daily for 4 to 6 weeks, except for patients treated for lesions related to prior neck irradiation, in whom clopidogrel treatment may be extended to 1 year. In the absence of contraindication, aspirin 160mg daily is prescribed indefinitely. We would arrange early computed tomographic angiography and ultrasound duplex study as a reference for later follow up evaluation. We had carried out 10 CAS in the last year and there was no post-procedural ipsilateral stroke or mortality. (Figure 2a and 2b)

Management of asymptomatic carotid stenosis

Patients with asymptomatic carotid stenosis treated medically only have a small risk of future stroke of about 2% per annum. If CEA can be performed safely with a perioperative stroke and death rate of no more than 3%, randomised trials showed a significant benefit of surgery over 5 year follow-up, with an overall reduction in the risk of stroke from about 11% over 5 years down to 6%.[35,36] An alternative paradigm was employed in our unit. The workup in our unit for asymptomatic carotid stenosis would include tests for cerebrovascular reactivity (which indicates cerebrovascular reserve). Patients with poor cerebrovascular reactivity would be of higher risk of subsequent stroke and would be offered carotid intervention.[37] Upon a mean follow up of 67 months, none of the 16 patients with asymptomatic carotid stenosis and normal cerebrovascular reactivity developed stroke.

A variety of different imaging techniques have been developed for the indirect or non-invasive assessment of cerebral haemodynamics in patients with carotid artery disease. One method would be to measure flow velocity in the middle cerebral artery (MCA) by transcranial Doppler ultrasound (TCD).[38] It has been demonstrated that during carbon dioxide inhalation there is little change in MCA diameter and therefore any change is directly proportional to the change in flow. Both carbon dioxide and acetazolamide have been used to measure the vasodilatory reserve.[39,40] TCD-based techniques are cheap and simple and are tolerated by almost all patients. Impaired reactivity determined using this method correlated with evidence of ischaemia on magnetic resonance spectroscopy, as determined by the presence of lactate and a reduction in the neuronal marker N-acetyl aspartate.[41] It also correlated with vasodilatation, detected as an increased CBV (cerebral blood volume) to CBF (cerebral blood flow) ratio, estimated by PET.[42] Other options include stable Xenon-CT (also employed in our unit), SPECT, PET, MRI to have quantitative or relative measurement of CBF and CBV.[43]

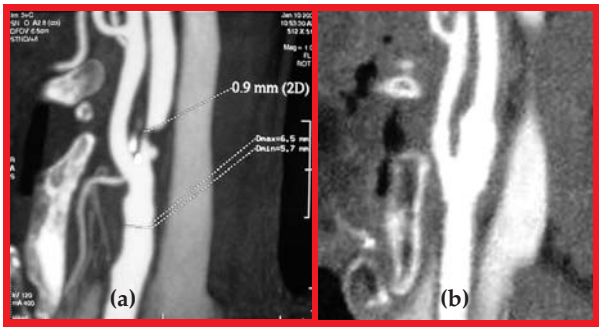

Figure 1 (a) CT angiogram showed typical proximal severe internal carotid stenosis; (b) CT angiogram showed satisfactory revascularisation after CEA.

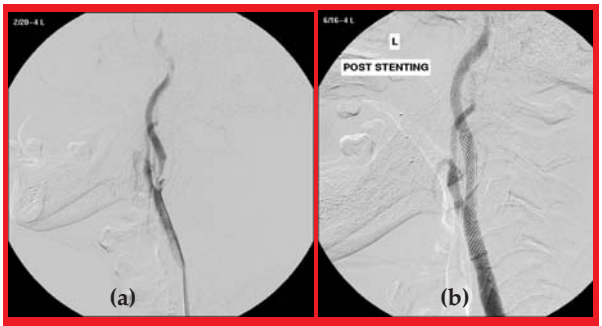

Figure 2 (a) DSA showed typical severe internal carotid stenosis with ulcerated plaque; (b) Post-CAS DSA showed satisfactory revascularisation.

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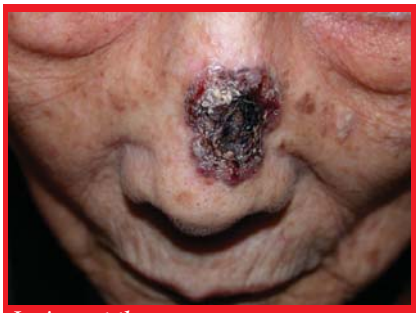
Dermatological Quiz

Dr. Ka-ho Lau MBBS(HK), FRCP(Glasg), FHKCP, FHKAM(Med)

Yaumatei Dermatology Clinic, Social Hygiene Service

Dr. Ka-ho Lau



Lesions at the nose

This 70-year-old female home resident was wheelchair bound and had multiple medical problems including dementia. She was noticed to have this pigment mole at her nasal bridge. The duration of the lesion was not clear to the attending elderly home staff. The mole seemed to have slowly increased in size with occasional bleeding.

Questions:

1. **What is your diagnosis or differential diagnoses?**
2. **How will you confirm the clinical diagnosis?**
3. **How will you manage this elderly woman?**

(See P. 26 for answers)

Soccer Five Tournament 2006

Match results for 29 October 2006

Score

HKMA	1 : 0	Jacobson
Schwarz Pharma	3 : 2	HKDA
GSK	1 : 2	HKOS
Janssen	1 : 2	Treasure Mountain

Quarter final match on 29 October 2006
There were 4 exciting matches between medical societies and pharmaceutical companies on 29 October 2006. All teams played well with support from their friends.

Schwarz Pharma team from back row left to right: Yeung Chuen Kwan, Roger Leung, Lam Ho Yin, Cedric Cheng, Paul Cheng, Rocky Leung and Dicky Lam, front row from left to right: Lewis Wong and Stephen Wong

Match results for 12 November 2006

Score for Semi-final match

HKMA	3 : 5	Schwarz Pharma
HKOS	3 : 4	Treasure Mountain

Score for 2nd Runner up

HKMA	3 : 0	HKOS

Score for Final match

Treasure Mountain	3 : 0	Schwarz Pharma

Quarter final match on 29 October 2006
All finalists displayed excellent team work and powerful play during the final matches. Our congratulations go to Treasure Mountain, Champions of the Federation Soccer Five Tournament 2006, Schwarz Pharma (1st Runner up), HKMA (2nd Runner up) and HKOS (3rd Runner up). The winner of the Top Scorer 2006 award was Stephen Wong, Schwarz Pharma who scored a total of 9 goals throughout the tournament.

Treasure Mountain team, Champions of the Federation Cup 2006.

Mr Anthony Chan, Vice President of HKAPI presenting the trophy and prize CD to the Schwarz Pharma team, 1st Runner up of the Federation Cup 2006.

HKOS team, 3rd Runner up of the Federation Cup 2006.

Dr Dawson Fong, President of FMSHK presenting the Top Scorer award to Mr Stephen Wong, Schwarz Pharma.

Mr Anthony Chan, Vice President of HKAPI presenting the trophy and prize CD to the HKMA team, 2nd Runner up of the Federation Cup 2006.

Federation News

At the 21st Annual General Meeting on November 9, 2006, new office-bearers for the year November 2006 - November 2007 were elected as follows:

President:	**Dr. Dawson T.S. FONG**
1st Vice-President:	**Dr. Chi-kuen CHAN**
2nd Vice-President:	**Dr. Susanna S.C. LO**
Hon. Treasurer:	**Mr. Nelson L.C. LAM**
Hon. Secretary:	**Dr. Raymond S.K. LO**
Deputy Hon. Secretary:	**Dr. Chun-on MOK**
Executive Committee Members:	**Dr. Godfrey C.F. CHAN**
	Dr. Sai-kwing CHAN
	Dr. James C.S. CHIM
	Dr. Chung-ping HO
	Dr. Wilson W.S. HO
	Dr. Kwan-ngai HUNG
	Dr. Walter W.K. KING
	Dr. Albert M.P. LEE
	Dr. Dominic F.H. LI
	Dr. Chi-wai MAN
	Ms. Manbo B.L. MAN
	Mr. Peter Y.Y. TO

Presentation of souvenir to Dr. Richard LO, the former 1st Vice-President of the FMSHK

From left to right: Dr. Richard LO and Dr. Dawson FONG (President, FMSHK)

Group photo - Dr. Dawson FONG (President, FMSHK) with Council Members and staff of the FMSHK

Society News

News from Member Societies

Hong Kong Continence Society Ltd.
New office-bearers for the year are as follows: President: Dr. Edward Man-fuk LEUNG, Vice-President: Dr. Cecilia CHEON, Hon. Secretary: Ms. Regina Kam-tin IP, Hon. Treasurer: Dr. Cheuk-kwan TAM, Council Representative: Dr. John FENN.

The Hong Kong Society of Rheumatology
New office-bearers for the year are as follows: President: Dr. Gavin Ka-wing LEE, Hon. Secretary: Dr. Temy Mo-yin MOK, Council Representative: Dr. Gavin Ka-wing LEE.

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

Answer to Dermatological Quiz

1. The diagnosis is basal cell carcinoma (BCC) in its pigmented form. The plaque, measuring about 2.5cm x 2cm at the nasal bridge of this fragile elderly woman, has an irregular well defined edge and darkly pigmented centre with scaly crust. Careful examination of the irregular edge shows characteristic pearly erythematous papules with some fine overlying telangiectatic vessels. The irregular well defined translucent glistening rolled pearly advancing edge is a very useful clue to the diagnosis of basal cell carcinoma. Among the different types of skin cancers, BCC is the commonest form encountered and there are different variants, namely nodulo-ulcerative (more commonly known as rodent ulcer), cystic, morphoeic, superficial multicentric, and pigmented forms. In Caucasians, nodulo-ulcerative variant is the most common form (~58.5% of patients) while pigmented BCC is rare (only occurred in 3.1% of patients). However, in Hong Kong, a study conducted in the Social Hygiene Service showed that pigmented BCC accounted for 58.1% of BCC and is the commonest form of presentation in local Chinese. Differential diagnoses in our patients should include malignant melanoma, pigmented Bowen's disease, squamous cell carcinoma and seborrhoeic warts.

2. Skin biopsy shows small, darkly blue staining, basophilic basaloid cells which invade the dermis. Mitoses are rare, and the cells do not look anaplastic. At the periphery, the masses of tumour cells tend to be arranged in characteristic palisades. A connective tissue stroma surrounds the tumour masses and is arranged in parallel strands. Mucinous, mucopolysaccharide components of the stroma are lost during tissue fixation, giving the histologic appearance that the tumour masses have shrunk with surrounding clefts.

3. Excision with a narrow margin of normal skin is the treatment of choice for younger patients. Cryotherapy, or curettage and cautery are sometimes useful for small superficial lesions. Radiotherapy is also effective and is especially suitable for the elderly, such as this patient, who is not a good candidate for complicated radical surgical excision which itself bears morbidity.

Dr. Ka-ho Lau
MBBS(HK), FRCP(Glasg), FHKCP, FHKAM(Med)
Yaumatei Dermatology Clinic, Social Hygiene Service

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
					★ 健康服務助理員訓練課程 (TC-HCA-0306) ★Certificate Course on Quality Management (TC-CQM-0106) ★9th International Symposium on Thrombolysis and Acute Stroke Therapy (TAST 2006) & 19th Annual Scientific Meeting of The Hong Kong Neurological Society **1**	★9th International Symposium on Thrombolysis and Acute Stroke Therapy (TAST 2006) & 19th Annual Scientific Meeting of The Hong Kong Neurological Society **2**
★HKMA Structured CME Programme Year 06/07 (IX) - Emergency Medicine & Anaesthesia ★9th International Symposium on Thrombolysis and Acute Stroke Therapy (TAST 2006) & 19th Annual Scientific Meeting of The Hong Kong Neurological Society **3**	★Certificate Course for Development of Advanced Nursing Practice (TC-ANP-0106) ★家務助理陪月服務訓練課程 (TC-PNC-0206) ★醫療護理常用英語初階 (SUSMH 025 0 B) **4**	**5**	★ 健康服務助理員訓練課程 (TC-HCA-0306) ★ 醫療護理常用英語初階 (SUSMH 025 0 B) **6**	★家務助理陪月服務訓練課程 (TC-PNC-0206) ★HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2006 (XII) - Health Check ★Advanced Wound Care Management (TC-AWCM-0106-CNSG) ★HKMA Council Meeting **7**	★ 健康服務助理員訓練課程 (TC-HCA-0306) ★Certificate Course on Quality Management (TC-CQM-0106) ★13th Annual Scientific Meeting : Skull Base and Cavernous Sinus Surgery **8**	★13th Annual Scientific Meeting : Skull Base and Cavernous Sinus Surgery ★HKMA Refresher Course for Health Care Providers 2006/2007 (IV) - Update on Hepatitis for Primary Care Providers ★Hong Kong Ophthalmological Symposium 2006 -Theme: Glaucoma **9**
★Pre-hospital Trauma Life Support (PHTLS) Provider Course ★HKMA Structured CME Programme Year 06/07 (IX) - Ophthalmology ★Hong Kong Ophthalmological Symposium 2006 -Theme: Glaucoma **10**	★Certificate Course for Development of Advanced Nursing Practice (TC-ANP-0106) ★家務助理陪月服務訓練課程 (TC-PNC-0206) ★醫療護理常用英語初階 (SUSMH 025 0 B) **11**	★HKMA Newsletter Editorial Meeting **12**	★ 健康服務助理員訓練課程 (TC-HCA-0306) ★ 醫療護理常用英語初階 (SUSMH 025 0 B) ★Hong Kong Neurosurgical Society Monthly Academic Meeting - Review on Management of Pituitary Adenoma **13**	★HKMA Lecture Series with Kennedys Part III: The Requirement of Consent ★家務助理陪月服務訓練課程 (TC-PNC-0206) **14**	★ 健康服務助理員訓練課程 (TC-HCA-0306) ★Certificate Course on Quality Management (TC-CQM-0106) ★Fourth International Huaxia Congress of Endocrinology **15**	★Fourth International Huaxia Congress of Endocrinology ★Advanced Certificate / Advanced Diploma in Health Informatics (Information Seminar) **16**
★Pre-hospital Trauma Life Support (PHTLS) Provider Course ★Fourth International Huaxia Congress of Endocrinology ★2nd Certificate Course in Recent Medical Advances for General Practitioners **17**	★Certificate Course for Development of Advanced Nursing Practice (TC-ANP-0106) ★家務助理陪月服務訓練課程 (TC-PNC-0206) ★Fourth International Huaxia Congress of Endocrinology ★醫療護理常用英語進階(SUSMH 026 0 B) **18**	**19**	★ 健康服務助理員訓練課程 (TC-HCA-0306) ★ 醫療護理常用英語進階 (SUSMH 026 0 B) **20**	**21**	★ 健康服務助理員訓練課程 (TC-HCA-0306) **22**	**23**
24			★ 健康服務助理員訓練課程 (TC-HCA-0306) ★ 醫療護理常用英語進階 (SUSMH 026 0 B)		★ 健康服務助理員訓練課程 (TC-HCA-0306) ★Certificate Course on Quality Management (TC-CQM-0106)	
★The Federation's 2006 Annual Dinner - A Go Go to Count Down ★HKMA Annual Dinner **31**	**25**	**26**	**27**	**28**	**29**	**30**

Medical Diary of December

Date / Time	Function	Enquiry / Remarks
1 (,6,8,13,15,20,22,27,29) **FRI** (8,15,29) (2,3)	健康服務助理員訓練課程 (TC-HCA-0306) Organised by: College of Nursing, Hong Kong **Certificate Course on Quality Management (TC-CQM-0106)** Organised by: College of Nursing, Hong Kong **9th International Symposium on Thrombolysis and Acute Stroke Therapy (TAST 2006) & 19th Annual Scientific Meeting of The Hong Kong Neurological Society** Organised by: The Hong Kong Neurological Society, Chinese Society of Neurology & The Hong Kong Polytechnic Universtiy (Rehabilitation Science Department) Speaker: Various #Jockey Club Auditorium, The Hong Kong Polytechnic University, Hung Hom, Kowloon	Sugar Tel: 2572 9255 Fax: 2838 6280 Sugar Tel: 2572 9255 Fax: 2838 6280 Conference Secretariat Email: tast2006@icc.com.hk Website: www.tast 2006.com
3 2:00pm **SUN**	**HKMA Structured CME Programme Year 06/07 (IX) - Emergency Medicine & Anaesthesia** Organised by: The Hong Kong Medical Association and Queen Elizabeth Hospital Chairman: Dr. T.C. SHIH Speaker: Various # Queen Elizabeth Hospital, Lecture Theatre, Block M, G/F, Queen Elizabeth Hospital, Kowloon	Miss Nina HUNG Tel: 2861 1979 (Registration fee is required) 3 CME Points
4 **MON** (11,18) (7,11,14) 6:30 pm - 9:30 pm (6,11,13)	**Certificate Course for Development of Advanced Nursing Practice (TC-ANP-0106)** Organised by: College of Nursing, Hong Kong 家務助理陪月服務訓練課程 (TC-PNC-0206) Organised by: College of Nursing, Hong Kong 醫療護理常用英語初階 (SUSMH 025 0 B) Organised by: College of Nursing, Hong Kong	Sugar Tel: 2572 9255 Fax: 2838 6280 Sugar Tel: 2572 9255 Fax: 2838 6280 Sugar Tel: 2572 9255 Fax: 2838 6280
7 2:00 pm **THU** 6:00 pm - 8:00 pm 8:00 pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2006 (XII) - Health Check** Organised by: The Hong Kong Medical Association and Hong Kong Sanatorium & Hospital Chairman: Dr. T.C. SHIH Speaker: Dr. M.Y. LO Goh # HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong **Advanced Wound Care Management (TC-AWCM-0106-CNSG)** Organised by: College of Nursing, Hong Kong Speaker: Various **HKMA Council Meeting** Organised by: The Hong Kong Medical Association # HKMA Headquarter Office, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Miss Nina HUNG Tel: 2861 1979 1 CME Point (Registration fee is required) Sugar Tel: 2572 9255 Fax: 2838 6280 Ms. Christine WONG Tel: 2527 8285
8 9:00am - 5:00pm **FRI** (9)	**13th Annual Scientific Meeting : Skull Base and Cavernous Sinus Surgery** Organised by: Hong Kong Neurological Society Speaker: Various # Langham Hotel, 8 Peking Road, Tsimshatsui, Kowloon, Hong Kong	Dr YC PO Tel: 2990 3788 Fax: 2990 3789
9 2:30 pm **SAT** (10)	**HKMA Refresher Course for Health Care Providers 2006/2007 (IV) - Update on Hepatitis for Primary Care Providers** Organised by: The Hong Kong Medical Association and Our Lady of Maryknoll Hospital Speaker: Dr. M.C. WONG # Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon, Hong Kong **Hong Kong Ophthalmological Symposium 2006 -Theme: Glaucoma** Organised by: College of Ophthalmologists of Hong Kong Chairman: Prof. Clement CY THAM Speaker: Prof. Robert STEGMANN # Hong Kong Convention & Exhibition Centre, Wanchai, Hong Kong	Ms. Clare TSANG Tel: 2354 2440 2 CME Points Ms. Vicki WONG Tel: 2761 9128 Fax: 2715 0089
10 **SUN** (17) 2:00 pm	**Pre-hospital Trauma Life Support (PHTLS) Provider Course** Organised by: Department of Surgery, University of Hong Kong and Hong Kong Chapter of the American College of Surgeons **HKMA Structured CME Programme Year 06/07 (IX) - Ophthalmology** Organised by: The Hong Kong Medical Association and Kwong Wah Hospital Chairman: Dr. T.C. SHIH Speaker: Dr. W.N. CHAN # Kwong Wah Hospital, Lecture Theatre, 10/F, Yu Chun Keung Medical Memorial Centre, Kwong Wah Hospital, Kowloon	Course Secretariat Tel: 2530 8016 Miss Nina HUNG Tel: 2861 1979 (Registration fee is required) 1 CME Point
12 8:00 pm **TUE**	**HKMA Newsletter Editorial Meeting** Organised by: The Hong Kong Medical Association Chairman: Dr. H.H. TSE # HKMA Headquarter Office, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Tammy TAM Tel: 2527 8941
13 7:30 pm **WEN**	**Hong Kong Neurosurgical Society Monthly Academic Meeting - Review on Management of Pituitary Adenoma** Organised by: Hong Kong Neurosurgical Society Chairman: Dr. PO Yin Chung Speaker: Dr. KWAN Chuk Lun Marco # Seminar Room, G/F, Block A, Queen Elizabeth Hospital, Kowloon	Dr. Y.C. PO Tel: 2990 3788 Fax: 2990 3789 2 CME Points
14 8:00 pm **THU**	**HKMA Lecture Series with Kennedys Part III: The Requirement of Consent** Organised by: The Hong Kong Medical Association and Kennedys Chairman: Dr. K CHOI Speaker: Mr. J WALLACE # HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong	Miss Nina HUNG Tel: 2861 1979 1 CME Point
15 **FRI** (16,17,18)	**Fourth International Huaxia Congress of Endocrinology** Hong Kong Convention & Exhibition Centre, Wanchai, Hong Kong	Conference Secretariat Tel: 2734 3312 Email: huaxia2006@pctourshk.com
16 2:30 pm - 4:00pm **SAT**	**Advanced Certificate / Advanced Diploma in Health Informatics (Information Seminar)** Oganized by: HKU Cyberport Institute of Hong Kong # Room S11, 3/F, Admiralty Centre, 18 Harcourt Road, Hong Kong	Ms Alice Wong Tel: 2587 2373 Website: http://cyber-i.hku.hk/courses/C163-101-00/index.html
17 **SUN**	**2nd Certificate Course in Recent Medical Advances for General Practitioners** Oganized by: the Family Medicine Unit, the University of Hong Kong and the Family Medicine Division, Hong Kong Sanatorium and Hospital Speakers: Various	Hospital Administration Department Tel: 2835 8800 Fax: 2835 8008 E-mail:hospadm@hksh.com, Website: http://www.hksh.com/CME.pdf
18 6:30 pm - 9:30 pm **MON** (20,27)	醫療護理常用英語進階 (SUSMH 026 0 B) Organised by: College of Nursing, Hong Kong	Sugar Tel: 2572 9255 Fax: 2838 6280
31 8:00 pm **SUN** 8:00 pm	**The Federation's 2006 Annual Dinner - A Go Go to Count Down** Organised by: The Federation of Medical Societies of Hong Kong Chairman: Dr. Albert LEE # Run Run Shaw Hall, The Hong Kong Academy of Medicine Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong **HKMA Annual Dinner** Organised by: The Hong Kong Medical Association Chairman: Dr. Y.S. CHAN # Conrad Hotel, Pacific Place, 88 Queensway, Hong Kong	Ms. Karen Chu Tel: 2821 3515 Fax: 2865 0345 Secretariat Tel: 2527 8285

Meetings

12-13/01/2007	**Hong Kong Surgical Forum, Winter 2007** Organised by: American College of Surgeons, Hong Kong Chapter & Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong # Underground Lecture Theatre, New Clinical Building, Li Ka Shing Faculty of Medicine, University of Hong Kong Medical Centre, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: http://www.hku.hk/surgery
13/1/2007	**Live Surgery - A New Specialty** Organised by: American College of Surgeons, Hong Kong Chapter & Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong Speaker: Prof. Henri BISMUTH Venue: Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885 / 2855 4886 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: www.hku.hk/surgery
25-27/01/2007	**International Colorectal Disease Symposium (ICDS) 2007** Organised by: Hong Kong Society for Coloproctology & Pamela Youde Nethersole Eastern Hospital (Department of Surgery) Chairman: Mr. Michael K.W. LI Speaker: Various # 2/F New Wing, Hong Kong Convention & Exhibition Centre Enquiry: Ms. Olivia HO Tel: 2595 6362 Fax: 2515 3195
2-4/02/2007	**Cardio Rhythm 2007** Organised by: Hong Kong College of Cardiology & Chinese Society of Pacing and Electrophysiology # Hong Kong Convention & Exhibition Centre Enquiry: Secretariat, CMP Medica Pacific Limited Tel: 2559 5888 Fax: 2559 6910 Email: info@cardiorhythm.com Website: www.cardiorhythm.com
10-11/02/2007	**Cancer Imaging 2007 - Joint Meeting of the International Cancer Imaging Society & Hong Kong College of Radiologists** Organised by: International Cancer Imaging Society & Hong Kong College of Radiologists Chairman: Ms. Lilian LEONG Speaker: Various Venue: Hong Kong Academy of Medicine Jockey Club Building Enquiry: Mrs. Maureen WATTS Tel: 44 (0) 208 661 3420 Fax: 44 (0) 208 661 3901 E-mail: Maureen.Watts@icr.ac.uk or Ms. Diane LEE Tel: 2871 8788 Fax: 2554 0739 E-mail: enquiries@hkcr.org
13-17/06/2007	**The 21st Congress of International Association of Paediatric Dentistry IAPD** Organised by: Hong Kong Society of Paediatric Dentistry # Hong Kong Convention & Exhibition Centre Enquiry: Mr. Daniel CHOK Tel: 2871 8896 Fax: 2871 8898 Email: info@iapd2007.com Website: http://www.iapd2007.com
7-8/07/2007	**Head and Neck Course 2007 - Surgery for Nasopharyngeal Cancer** Organised by: Department of Surgery, Li Ka Shing Faculty of Medicine, University of Hong Kong Medical Centre, Queen Mary Hospital # Underground Lecture Theatre, New Clinical Building, Li Ka Shing Faculty of Medicine, University of Hong Kong Medical Centre, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Course Secretariat Tel: 2855 4885 Fax: 2819 3416 Email: hnsrg@hkucc.hku.hk Website: http://www.hku.hk/surgery
12/07/2007	**The 1st Nursing Forum** Organised by: American College of Surgeons, Hong Kong Chapter & Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong # Underground Theatre, New Clinical Building, Li Ka Shing Faculty of Medicine, University of Hong Kong Medical Centre, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885/ 2855 4886 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: http://www.hku.hk/surgery
12-14/07/2007	**The 50th Hong Kong Surgical Forum** Organised by: American College of Surgeons, Hong Kong Chapter & Department of Surgery, Li Ka Shing Faculty of Medicine, The University of Hong Kong # Underground Lecture Theatre, New Clinical Building, Queen Mary Hospital, Pokfulam, Hong Kong Enquiry: Forum Secretary Tel: 2855 4885/ 2855 4886 Fax: 2819 3416 Email: hksf@hkucc.hku.hk Website: http://www.hku.hk/surgery

Courses

3,5,10,12,17,19,24,26, 31/01/2007 2,7,9,14,16,28/02/2007 2,7,9,14,16,21,23,28,3 0/03/2007	健康服務助理員訓練課程 (TC-HCA-0306) Organised by: College of Nursing, Hong Kong Enquiry: Sugar Tel: 2572 9255 Fax: 2838 6280
3,10,17,24,31/01/2007 7/02/2007	**Certificate Course on Medical Genetics (C111)** Organised by: The Federation of Medical Societies of Hong Kong & Hong Kong Society of Medical Genetics Speakers: Various # Lecture Hall, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong Enquiry: Karen CHU Tel: 2821 3515 Fax: 2865 0345
4,11,18,25/01/2007	**Certificate Course on Drugs Dispensing in Office Clinics (C114)** Organised by: The Federation of Medical Societies of Hong Kong Speakers: Mr Wilson WONG & Dr Ben FONG # Conference Room II, Shatin Hospital, 33 A Kung Kok Street, Ma On Shan, Shatin, NT Enquiry: Karen CHU Tel: 2821 3515 Fax: 2865 0345
9,16/01/2007	**Certificate Course on Drugs Safety in Old Age Homes (C112)** Organised by: The Federation of Medical Societies of Hong Kong Speakers: Mr Henry CHAN & Ms Rebecca POON # Lecture Hall, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong Enquiry: Karen CHU Tel: 2821 3515 Fax: 2865 0345
21/1/2007, 11/2/2007 18/3/2007, 22/4/2007 20/5/2007, 17/6/2007	**2nd Certificate Course in Recent Medical Advances for General Practitioners** Jointly organized by the Family Medicine Unit, the University of Hong Kong and the Family Medicine Division, Hong Kong Sanatorium and Hospital Speakers: Various, Enquiry: Hospital Administration Department Tel: 2835 8800, Fax: 2835 8008, E-mail:hospadm@hksh.com, Website: http://www.hksh.com/CME.pdf

CERTIFICATE COURSE FOR MEDICAL AND HEALTH PROFESSIONALS

Certificate Course on Medical Genetics

醫學遺傳學證書課程

(Course no. C111)

Jointly organised by

The Federation of Medical Societies of Hong Kong
香港醫學組織聯會

Hong Kong Society of Medical Genetics
香港醫學遺傳學會

Objective: The field of medical genetics has seen rapid advances in the last ten to twenty years, especially so with the knowledge brought about by the Human Genome Project. This course aims to provide the participants an overview and update in this field, so that they can appreciate how the practice of medicine is changing with the genetic advances.

Date	Topic	Lecturer
3 January 2007	Medical Genetics in Hong Kong - An Overview 醫學遺傳學在香港的概覽	Dr. Stephen Lam 林德深醫生
10 January 2007	Genetic Counseling 遺傳輔導	Dr. Ivan Lo 盧輝文醫生
17 January 2007	Chromosomal Disorders in Hong Kong 香港的染色體疾病	Mr. Chan Wing-kwong 陳永光高級化驗師
24 January 2007	Application of Molecular Genetics in Patient Care 分子遺傳學的臨床應用	Dr. Brian Chung 鍾侃言醫生
31 January 2007	Prenatal Diagnosis & Therapy 產前診斷及治療	Dr. Leung Kwok-yin 梁國賢醫生
7 February 2007	Treatment Strategies in Genetic Diseases 遺傳病的療法	Dr. Larry Baum 包立怡博士 Dr. Richard Choy 蔡光偉博士

Date : 3 January 2007 to 7 February 2007 (Every Wednesday)
Time : 7:00 p.m. - 8:30 p.m.
Venue : Lecture Hall, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong
Course Fee : HK$960 (6 Sessions)
Language : English
Certificate : Awarded to participants with a minimum attendance of 70%
Enquiry : The Secretariat of the Federation of Medical Societies of Hong Kong
Tel. : 2527 8898 Fax: 2865 0345 Email: info@fmshk.org

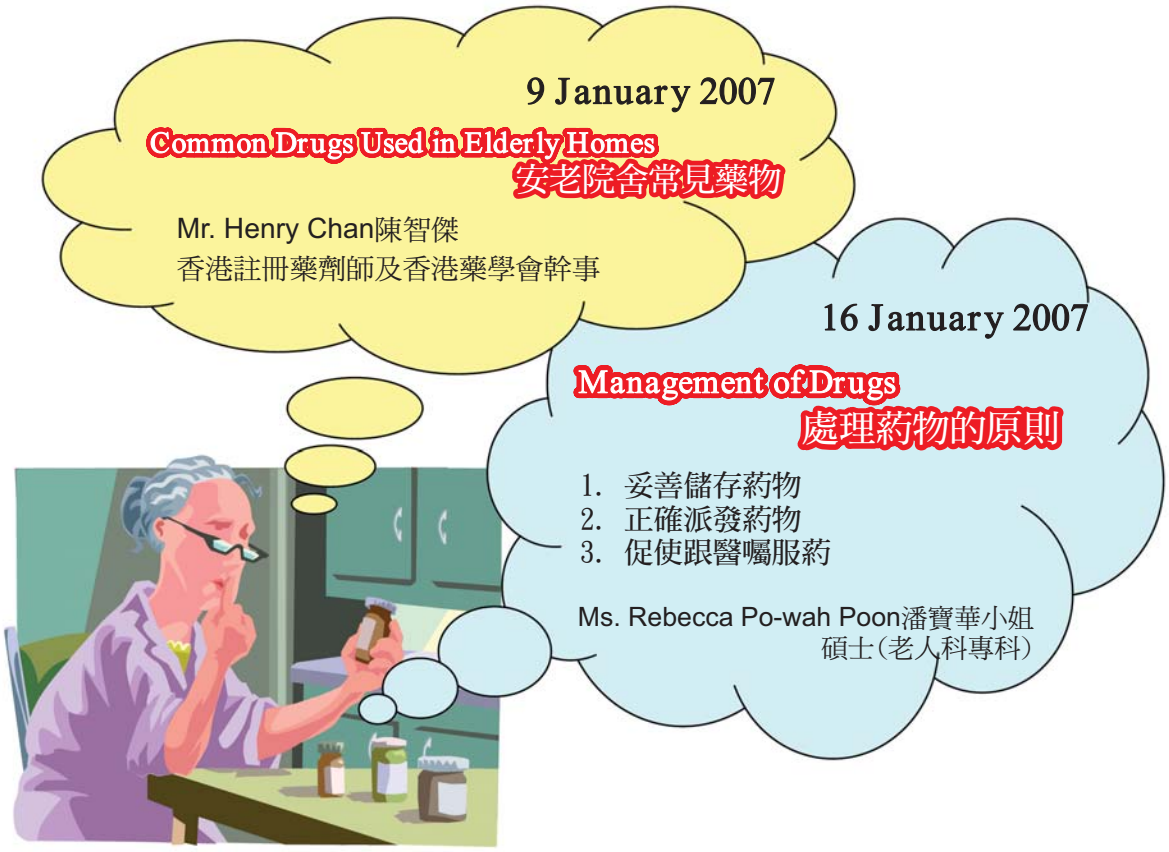
CME/CPE Accreditation applied for
For downloading the application form, please refer to our website:
http://www.fmshk.org

THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
香 港 醫 學 組 織 聯 會

Certificate Course on Drugs Safety in Old Aged Home

(Course No. C112)

Objective: To enhance safety of drugs handling in old aged home



Date	: 9 & 16 January 2007
Time	: 7:00 p.m. - 8:30 p.m.
Venue	: Lecture Hall, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong
Course Fee	: HK$350 (2 Sessions)
Language	: Cantonese (Supplemented with English)
Certificate	: Awarded to participants with a minimum attendance of 70%
Enquiry	: The Secretariat of the Federation of Medical Societies of Hong Kong
Tel.	: 2527 8898 Fax: 2865 0345 Email: info@fmshk.org

CME/CPE Accreditation applied for
For downloading the application form, please refer to our website:
http://www.fmshk.org

THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
香 港 醫 學 組 織 聯 會

CERTIFICATE COURSE FOR MEDICAL AND HEALTHCARE PROFESSIONALS

Certificate Course on Drug Dispensing in Office Clinics
診 所 藥 物 處 理 及 配 藥 基 本 課 程

(Course No. C114)

Objective: *To enhance safety and efficacy of clinic assistants in drug dispensing in office clinics.*

2007年1月4日

Drug Classification in Hong Kong – A Simple Approach Common Drugs Identifications in Clinic
香港藥物分類簡介及診所藥物識別

Mr Wilson Wong 黃永杰先生
卓健醫療體檢中心有限公司藥劑部高級經理

2007年1月11日

Drugs Stock Keeping and Recording
診所藥品庫存及記錄

Mr Wilson Wong 黃永杰先生
卓健醫療體檢中心有限公司藥劑部高級經理

2007年1月18日

Good Dispensing Guidelines
良好配藥守則

Dr Ben Fong 方玉輝醫生
香港中文大學大學保健處處長

2007年1月25日

Clinic Dispensing Management
診所配藥管理

Mr Wilson Wong 黃永杰先生
卓健醫療體檢中心有限公司藥劑部高級經理

日期 ：2007年1月4日至2007年1月25日
時間 ：下午2時至3時30分
地點 ：馬鞍山亞公角道33號，沙田醫院一樓會議室(II)
收費 ：每位港幣$500元(4堂)
語言 ：粵語
備註 ：如出席率達70%，可獲發證書

索取報名表格及查詢，請與香港醫學組織聯會秘書處聯絡

香港灣仔軒尼詩道十五號溫莎公爵社會服務大廈四樓
電話: 2527 8898　傳真: 2865 0345　電郵: info@fmshk.org
或瀏覽網址: www.fmshk.org 下載報名表格

THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
香 港 醫 學 組 織 聯 會

Duke of Windsor Social Service Building, 4/F, 15 Hennessy Road, Hong Kong
Tel: (852) 2527 8898 Fax: (852) 2865 0345 Homepage: www.fmshk.org E-mail: info@fmshk.org

Application Form for Certificate Course

Name of Applicant:(Prof/Dr./Mr./Ms./Mrs.)*_____ (English) _____ (Chinese)
*Please delete as appropriate (in block letters)

Correspondence Address: _____

_____

Tel. No. : _____ Fax No.: _____ Age: _____ Sex: _____

Email Address: _____ Occupation: _____

Course Title: (please tick)
☐ Certificate Course on Drugs Dispensing in Office Clinic (C113)
☐ Certificate Course on Medical Genetics (C111)
☐ Certificate Course on Drugs Safety in Old Age Home (C112)

Education : (please tick) ☐Secondary ☐Undergraduate ☐Postgraduate ☐Others _____

Fee enclosed (please tick):

☐Cheque No: _____ made payable to **The Federation of Medical Societies of Hong Kong**

☐Cash HK$ _____

_____ _____
Signature Date

Note:
1. The application form together with the appropriate fee should be sent to the Secretariat of the Federation of Medical Societies of Hong Kong, 4/F Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong.
2. Fees are not refundable, except in the event of a course being oversubscribed or cancelled.
3. The Federation of Medical Societies of Hong Kong reserves the right to cancel the course should too few participants enroll for the course.
4. No classes will be held when typhoon signal No. 8 or above or black rainstorm warning is still hoisted after 12:00 noon. Please contact the Secretariat at 2527 8898 to enquire matters regarding cancellation of class due to typhoon or black rainstorm.

For office use:

Registration confirmed on : _____ **Registration Number :** _____

Cheque Issuing Bank : _____ **Cheque Number:** _____

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