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Neurosurgery

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Contents

Disclaimer

Chinese New Year Message from the President

Dr. Raymond SK LO

President
The Federation of Medical Societies of Hong Kong

Dr. Raymond SK LO

On behalf of Executive Committee and Foundation Directors of the Federation, I would like to wish colleagues of our member societies and readers of our Medical Diary a very happy and healthy Year of the Rabbit. We would like to wish you all a productive year ahead, especially in working together for our professional fraternity and in contribution to health services for our patients and the society at large.

Time flies and last year has been a busy year for the Federation and Foundation too. The charity concert for our Foundation project in helping bereaved children had been a resounding success, and our first outing with a group of bereaved children was most gratifying and rewarding. A continuing series of activities will be rolled out this year for bereaved children, with a play therapy programme commencing soon. Please watch out for our announcement. Meanwhile further information on the project regarding referrals, volunteering or donation will be available from our secretariat, and we would be most delighted if you will join us in this very worthy cause.

The Federation will maintain the momentum in linking up and supporting our member societies. We shall actively engage our members in promoting health knowledge and information to professionals and public through various channels, as well as providing assistance with secretarial services and organising courses, seminars or annual meetings. Our general interest talks for fellow professionals proved popular last year, and this year we shall start with an overseas education talk exclusively for our members, co-hosting with the British Medical Association (Hong Kong) and supported by the British Council. A variety of interesting events will follow.

Looking forward to your participation, and meeting you in our various functions!

Back : Dr.Yin-kwok NG, Dr.Maureen Mo-lin WONG, Dr.Nancy Shi-yin YUEN, Dr.Chi-wai MAN, Ms. Tina WT YAP, Dr. Gilberto Ka-kit LEUNG, Mr. Wilfred WONG
Front : Dr. Dawson To-sang FONG, Dr. Susanna Sze-ching LO, Prof. Godfrey Chi-fung CHAN, Dr. Raymond See-kit LO, Dr. Sai-kwing CHAN, Mr. Benjamin LEE

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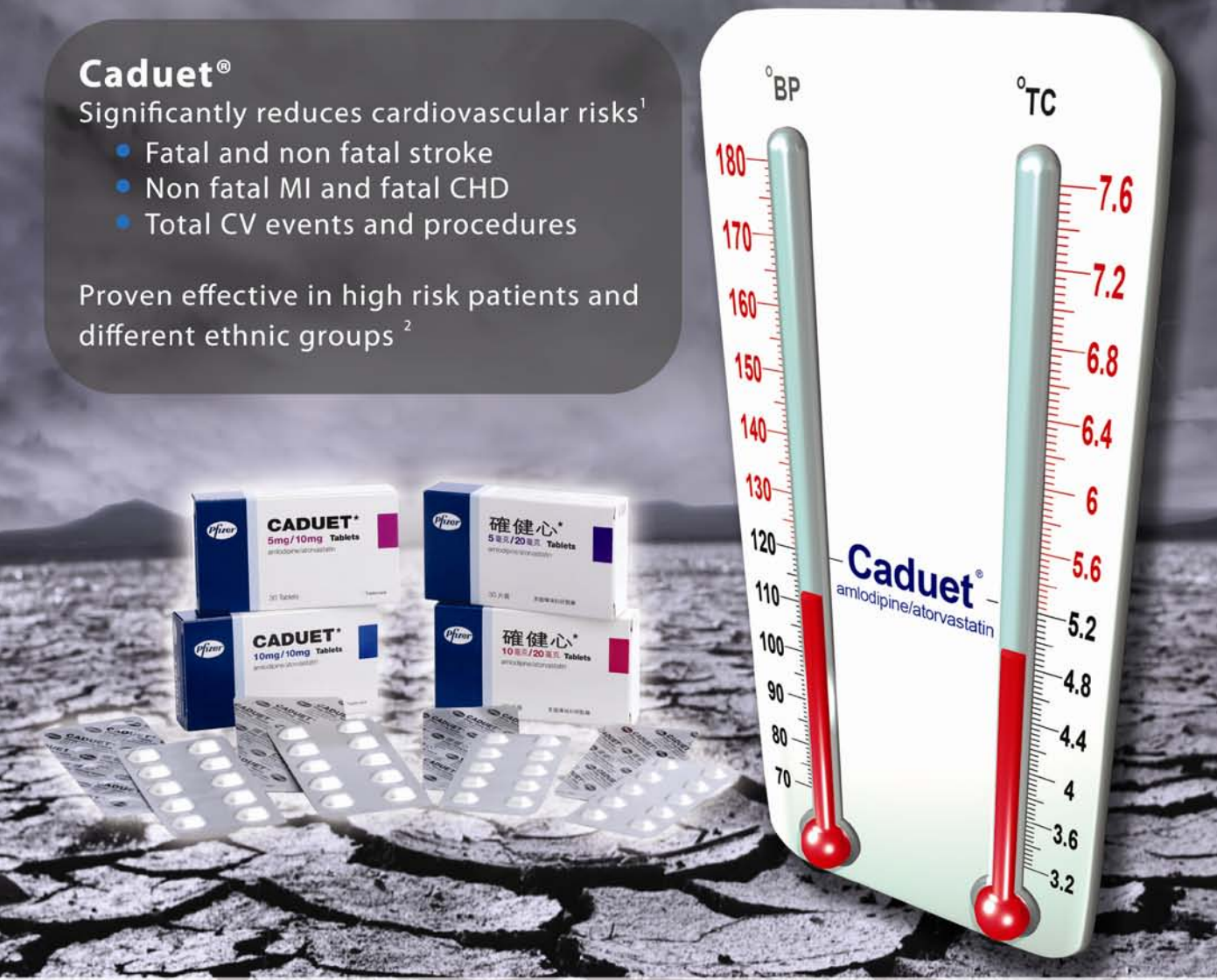

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Editorial

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Editor

Dr. Yiu-wah FAN

This issue of the Medical Diary is dedicated to a few incidental conditions, which we commonly encounter during 'rountine' MRI or CT scans of the brain.

As modern neuro-imaging is getting more widely available, we are referring more and more patients for brain scanning. Many patients actually ask for it as part of their health check. Undue anxiety sometimes arises after knowing the presence of a lesion in the brain. Many of these lesions are harmless while some require treatment.

We will start with an article outlining the different choices of common neuro-imaging modalities at the present time and then follow by articles covering unruptured intracranial aneurysms, cavernous haemangiomas, arterial stenosis, and incidental pituitary lesions. In the drug session, we will revise on the common medications we use during neuro-imaging and some of the precautions that we need to be aware of.

The Cover Shot

Dr. Kin-ming WONG
MBBS (HK), DFM (CUHK),
DOM (CUHK), DDME(CUHK)

Photo is a snap shot at Chinese New Year 2010 near the riverside of a branch of Songhua River (松花江), Jilin City (吉林市), Jilin Province (吉林 省). Outside temperature was -16C. It was very wonderful to have such a quiet and peaceful sunset. My brain was totally immersed in the golden atmosphere of winter.

Roche

In glioblastoma, at relapse or disease progression

- At 24 weeks more than 40% of Avastin patients are still alive without disease progression[1]
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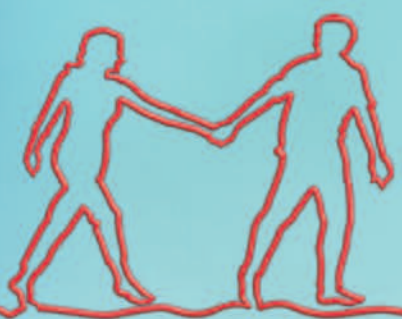
- There was a trend for patients taking corticosteroids at baseline to take stable or decreasing doses over time[1]



Around the world 28 countries have now recognised the significant clinical benefits Avastin offers to people with relapsed glioblastoma by granting approval

Abbreviated Prescribing information - Avastin Roche injection 100mg/4ml (bevacizumab)
Indications: ***mCRC***: In combination with fluoropyrimidine-based chemotherapy. ***mBC 1st line***: In combination with paclitaxel or docetaxel. ***NSCLC 1st line***: In addition to platinum-based chemotherapy. ***RCC 1st line***: In combination with interferon alfa-2a. ***Glioblastoma***: As single agent for patients with progressive disease following prior therapy.
Dosage & administration: ***mCRC***: 5mg/kg or 10mg/kg every 2 weeks or 7.5mg/kg or 15mg/kg every 3 weeks. ***mBC:*** 10mg/kg every 2 weeks or 15mg/kg every 3 weeks. ***NSCLC***: 7.5mg/kg or 15mg/kg every 3 weeks in addition to platinum-based chemotherapy for up to 6 cycles, then as monotherapy. **RCC/** ***Glioblastoma:*** 10mg/kg every 2 weeks. Administration times: *initial dose:* 90 minute IV infusion. Infusion time for second & subsequent doses can be reduced if previous dose was well tolerated.
Contraindications: Hypersensitivity to bevacizumab, Chinese hamster ovary cell products, other recombinant human or humanised antibodies or any excipients.
Warnings & Precautions: GI perforation; fistulae; haemorrhage, especially tumour-associated haemorrhage and risk of CNS haemorrhage in patients with untreated CNS metastases has not been evaluated; patients should be monitored for signs and symptoms of CNS bleeding and discontinue Avastin Roche in cases of intracranial bleeding; congenital bleeding diathesis; acquired coagulopathy or during anticoagulant therapy; serious/fatal pulmonary haemorrhage/haemoptysis (NSCLC only): avoid in patients with recent pulmonary haemorrhage/haemoptysis (>1/2 teaspoon of red blood); control pre-existing hypertension; monitor blood pressure; Reversible Posterior Leukoencephalopathy Syndrome (RPLS); history of arterial thromboembolism; venous thromboembolism, including pulmonary embolism; clinically significant cardiovascular disease such as pre-existing coronary artery disease or CHF; neutropenia; wound healing complications (do not initiate for at least 28 days following major surgery or until surgical wound is fully healed); proteinuria; infusion/hypersensitivity reactions (close observation during and following the administration); avoid in pregnancy and use appropriate contraception for at least 6 months after last dose; do not breast feed for at least 6 months after last dose; use with caution in elderly; not recommend in paediatrics, patients with renal or hepatic impairment. MAHA reported when use in combination with sunitinib.
Undesirable effects: For full listings please refer to the Avastin Roche package insert. Most serious: GI perforation, haemorrhage, including pulmonary haemorrhage/haemoptysis, arterial thromboembolism. Other serious: fistulae, tumour-associated haemorrhage, hypertension, venous thromboembolism, CHF, proteinuria, hypersensitivity reactions. Most frequent: fatigue or asthenia, diarrhoea & abdominal pain. Laboratory abnormalities and Post Marketing - refer to package insert.
Date of preparation: November 2010
Full prescribing information should be viewed prior to prescribing.

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Management of Unruptured Intracranial Cerebral Aneurysms

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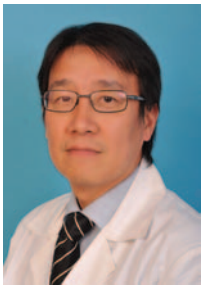

Dr. Yiu-wah FAN Dr. Wai-man LUI

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 28 February 2011.

Findings of incidental unruptured intracranial cerebral aneurysms on MRI or CT brain examinations are getting more common. Epidemiology studies suggest that as many as 5% of people harbour a cerebral aneurysm by the age 75.[8] Management of such aneurysms is becoming an important subject in our daily medical practice.

Treat or Observe?

The truly incidental aneurysms are those asymptomatic aneurysms in patients without any history of subarachnoid haemorrhage (SAH). This is a topic of major controversy.

Aneurysm represents a weakening in the vessel. It is a degenerative disease with a tendency to enlarge with time. The probability of rupture is related to the size of the aneurysm. Aneurysm rupture is a devastating event. Approximately 15 percent of patients die prior to reaching the hospital and, of those who make it in time, only one-third will have a "good result" after treatment. Therefore, salvage treatment is not effective.

It is logical to think that one should treat the aneurysms before they rupture. For some time, neurosurgeons are eager to treat incidental aneurysms hoping to prevent subsequent ruptures and try to beat the natural history.

The enthusiasm was dampened in 1998 when the International Study of Unruptured Intracranial Aneurysms (ISUIA)[7] was published. It was a retrospective study based in North America and Europe and is the biggest study on the subject at the present time. It quoted a rupture rate of 0.05% per year for aneurysms smaller then 10mm and, 0.5% per year for aneurysms larger than 10mm. This is much less than what we have formerly believed. Using these figures to compare the treatment risk of about 10% (mortality and morbidity combined), one would find it difficult to recommend prophylactic treatment for any aneurysm less than 10mm.

The study was, however, heavily criticised for recruitment and selection bias, and it prompted a prospective study. In the 2003 prospective ISUIA study, the 5-year cumulative rupture rates for patients who did not have a history of subarachnoid haemorrhage with aneurysms located in the internal carotid artery, anterior communicating artery, anterior cerebral artery, or middle cerebral artery were 0%, 2.6%, 14.5%, and 40% for aneurysms less than 7 mm, 7–12 mm, 13–24 mm, and 25 mm or greater, respectively, compared with rates of 2.5%, 14.5%, 18.4%, and 50%, respectively, for the same size categories involving the posterior circulation and posterior communicating artery aneurysms.[9] This 7mm size is now regarded as the cutoff for treatment by most neurosurgeons.

Although the ISUIA-II study settled some controversies, what remained puzzled is why we are seeing many small aneurysms, less than 7mm, rupture with SAH and yet the ISUIA study was telling us that they have a very low risk of rupture. From a recent study, 71.8% of ruptured aneurysms were smaller than 7 mm, and 87.9% were smaller than 10mm. [3]

Among many of us, the belief is that small aneurysms can also rupture and it is better to secure them if technically feasible.

When one makes the decision for treating an incidental aneurysm, one does not only take in consideration the size alone. The morphology of the aneurysm is also important. The presence of a daughter sac in the aneurysm, representing a weak point in the aneurysm, will prompt for intervention. Patients with a history of previous SAH, an aneurysm at the posterior circulation, a family history of SAH and patients with autosomal dominant polycystic kidney disease (ADPKD) all pose higher risks of ruptures.

Making the decision of whether to treat or not is a complex issue and we will consider the following:

Factors favouring treatment for incidental aneurysms

1. size >7mm
2. posterior circulation <including PcoA> aneurysm
3. presence of daughter sac (representing a weak point in the aneurysm)
4. long life expectancy, (young age)
5. history of SAH in the past
6. family history of SAH
7. patient with ADPKD

In the ISUIA study[9], the treatment risk (combined morbidity and mortality) at 1 year was 12.6% for

clipping and 9.8% for coiling. Although a simple comparison of treatment risk and natural history can be calculated, one has to take into consideration the psychological factor. No matter what the statistics show, no one can guarantee a risk-free choice. After explaining the pros and cons, we have to take care of the patient's psychology and help them make decision. If the patient cannot accept the psychological burden of having a 'time bomb' in his /her head, treating a small aneurysm may be justified.

Screening for Aneurysms

It is generally believed that routine screening for aneurysms in the general population is not indicated. If it is to be carried out, it should be targeted to specific subpopulations at increased risks.

Specific genetic syndromes that are associated with an increased risk of SAH include the Ehlers-Danlos syndrome type IV, Marfan syndrome and autosomal dominant polycystic kidney disease (ADPKD). Among them, ADPKD is most indicated for screening.[2]

If two or more members of the family are affected with aneurysms, screening is recommended for the first-degree relatives. Screening with MRA or CTA is recommended to start in their twenties and then every 5 to 10 years thereafter.

Patients with treated aneurysms may develop new aneurysms with time.[4] Rinne and Hernesniemi suggested in their study of 1150 aneurysm patients, that individuals who present with SAH before the age of 40 may be particularly susceptible to aneurysm formation and may benefit from screening at 5-year intervals.[6]

If we choose not to do surgery, what can we do to help?

It has been shown that cigarette smoking, and hypertension are associated with aneurysm formation. Controlling these risk factors theoretically will help the situation.

How often we should do follow up angiograms?

It is common practice to follow up patients with UIA with angiogram. There is really no guideline on this subject. Depending on the size of the lesion, and the intention of the patient, if it is approaching the 7 mm cutoff and the patient is ready to have it treated, then we will do the follow-up angiograms more frequently. A yearly follow up is our usual practice.

We use MRA for follow-up. MRA can be done without radiation and contrast. Bear in mind that we may need to do many follow up angiograms, especially if the patient is young. Although MRA is not as accurate as DSA for small aneurysms (< 5 mm), it does not really matter if we are using 7 mm as the cutoff for treatment.

Shall we treat immediately if we see a small growth of aneurysm on follow-up? Or shall we wait until it is bigger than 7 mm?

Although we tend to believe most aneurysms grow in a linear fashion, they sometime can enlarge rapidly. From the statistical point of view, there may not be any difference in bleeding risks for a 4 mm aneurysm and a 6 mm aneurysm. We look upon a growing aneurysm as an unstable aneurysm and we tend to treat it when there is documented growth on follow up angiograms, especially if the growth is irregular.

During observation of an unruptured incidental aneurysm, is it safe to use antiplatelet and anticoagulant?

It is understandable that SAH will be more devastating during aneurysm rupture if the patient is on medications that hinder haemostasis. As vascular diseases are prevalent, there is an increasing need for such medications. When confronted with such a problem, we continue to prescribe antiplatelet and anticoagulant if there is a clear indication for them. We tend to believe that these medications do not actually increase the chance of ruptures, but they will make things worse if a rupture occurs. Having said that, the weighting for treating the aneurysm will be higher in such circumstances.

What treatment? Clip or coil / stent?

Once the decision has been made for treatment, we have a choice of open clipping and endovascular treatment (coiling and stenting).

With the rapid development of endovascular technology, and support of the International Subarachnoid Aneurysm Trial (ISAT)[5] (which showed less complication with coiling than open clipping in patients with ruptured aneurysms), both patients and doctors are gradually siding towards endovascular treatment. However, one should be careful in assessing the end point of treating unruptured aneurysms, in which long-term secure control is the aim, especially in young patients. Clipping has a long track record for good secure control whereas endovascular treatment is often associated with recurrences and requirement for retreatment.

If the patient is younger than 50 year old and the aneurysm is technically easy to clip, there is a point of going for open clipping. Otherwise, endovascular management seems to be the treatment of choice especially for posterior-circulation aneurysms.

In the ISUIA study, the combined morbidity and mortality rates at 1 year were 12.6% for clipping and 9.8% for coiling. These are general figures including treatment for large difficult aneurysms, which we know are associated with a high morbidity. With further development in technology, we expect to see lower

management mortality and morbidity in the future. Recent reports, especially with endovascular treatment, have demonstrated a very low risk of treatment (1.5%).[1] Having said that, one very important factor in keeping the treatment risk low is to adopt a conservative attitude during surgery for unruptured aneurysms. At the time of operation, if there is any difficulty, one should reconsider the need to press on when the risk becomes substantial.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Management of Unruptured Intracranial Cerebral Aneurysms" by Dr. Yiu-wah FAN and Dr. Wai-man LUI and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded 1 CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 28 February 2011. 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. **Most intracranial aneurysms are congenital in nature and are present at birth.**
2. **The prevalence of unruptured intracranial aneurysms in the general population is up to 5%.**
3. **The immediate mortality rate for ruptured intracranial aneurysms is 50%.**
4. **Posterior communicating artery aneurysms have a lesser tendency to rupture compared with anterior communicating artery aneurysms.**
5. **Presence of a daughter sac in an aneurysm is a risk factor for rupture.**
6. **Smoking has a favourable effect on aneurysm rupture.**
7. **Patients with autosomal dominant polycystic kidney disease have a higher incidence of cerebral aneurysm.**
8. **Screening for cerebral aneurysms is well established for the general population.**
9. **There is no chance of rupture for aneurysms smaller than 5mm in diameter.**
10. **Endovascular treatment of cerebral aneurysms has poorer immediate outcome compared with craniotomy and clipping.**

ANSWER SHEET FOR FEBRUARY 2011

Please return the completed answer sheet to the Federation Secretariat on or before 28 February 2011 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Management of Unruptured Intracranial Cerebral Aneurysms

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Answers to January 2011 Issue

An Update in AF Management

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

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(d) Satisfy the examiners in a qualifying examination, if required.

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Contrast Agents for Neuro-imaging

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Neuro-imaging is essential for making a diagnosis of the central nervous system diseases. Although intrinsic tissue contrast is present in the CT/MR images, neuro-imaging also relies heavily on contrast media to improve lesion detection (sensitivity) and to aid in lesion characterisation (specificity). Furthermore, contrast could be used for functional assessment of physiologic processes including perfusion/blood flow and vascular status. During the cerebral angiography, contrast is injected via the catheter to opacify the intracranial arteries. Contrast agents are essential for contemporary neuro-diagnosis. Although rare, contrast reactions do occur and one must be aware of this. Adverse reactions are more common with CT contrast agents ('iodinated' contrast) than with MR contrast agents ('gadolinium' contrast).

CT Scan

Since the contrast could not pass through the blood-brain barrier (BBB), only vascular structures and areas of the brain that have no BBB (such as choroid plexus, pineal and anterior lobe of pituitary gland) enhance normally. Three pathologic (abnormal) enhancements occur.[1] 1) Abnormal enhancement within enlarged vessels without breakdown of BBB, including AVM or neoplasm with enlarged vascular spaces. 2) Breakdown of BBB with leakage of contrast e.g. neoplasm, infarction and inflammation. 3) Lesions with no BBB such as meningioma, acoustic schwannoma.

Adverse reactions happen after contrast administration. They are more common with the 'iodinated' contrast than with 'gadolinium' contrast. Most adverse effects after iodinated contrast are mild or moderate, which do not require treatment. Approximately 3% of patients undergoing contrast examination will have some types of reaction, usually mild vasomotor symptoms. About 0.03% will require hospitalisation and 1.6% requires treatment.[1] Reactions leading to death are rare and occur in about 1:250,000 patients.

The contrast reaction is classified into:[2]

1) **Mild Reaction** (no need for treatment): Nausea/ vomiting, urticaria.
2) **Moderate Reaction** (not immediately life threatening but often requires treatment) : Vasovagal reaction, mild bronchospasm or hypotension.
3) **Severe Reaction** (potentially or immediately life threatening): severe vasovagal reactions, hypotension or bronchospasm, laryngeal oedema and cardiac arrest.
4) **Organ-specific Effect**: pulmonary oedema or seizure
5) **Delayed** (0.5% to 9%): headaches, muscle pains and flu-like symptoms up to 48 hours after contrast media. Delayed cutaneous reactions ranging from 3 hours to 7 days after contrast injection, most often with exanthem (self-limited).

Prior sensitisation to the contrast agent is not required for an adverse reaction to occur. There is no reliable screening test to predict which patients will have a severe reaction. Most reaction occurs within a few minutes of injection. High-risk patients (those with a history of allergy or asthma) should be pre-treated with steroids but it does not guarantee an absence of reactions. Patients with a history of documented iodinated contrast reaction should not be injected with iodinated contrast. It is better to consider alternative examinations such as MRI with gadolinium contrast. The iodinated contrast media can damage the kidneys (**contrast nephrotoxicity**). Patients with renal dysfunction taking Metformin could have risks of lactic acidosis and therefore, Metformin should be suspended at the time of contrast injection.

MR Imaging

In the presence of intact BBB, most of the normal neuronal structures will not enhance as in CT scan. Any pathology that disrupts the BBB will enhance after IV contrast injection. In order to avoid erroneous interpretation of the contrast enhancing lesion, unenhanced images must be available for comparison.[3]

Gadolinium chelates are extremely well tolerated by the vast majority of patients. The frequency of all acute events after an injection of 0.1 or 0.2mmol/kg of gadolinium chelate ranges from 0.07% to 2.4%. The vast majority of these reactions are mild. 'Allergic' responses are unusual (0.004% to 0.7%), including urticaria and very rarely bronchospasm. Severe life-threatening reactions are exceedingly rare (0.001% to 0.01%). Persons with asthma and various allergies are also at greater risks (up to 3.7%). [2]

Gadolinium agents are considered to have no nephrotoxicity at approved dosages. However, they can result in nephrogenic systemic fibrosis (NSF) for patients with actual renal failure or severe chronic kidney disease. NSF is a fibrosing disease, which will primarily be identified in skin and subcutaneous tissues but will also involve other organs including the lung, oesophagus, heart and skeletal muscles. It is estimated that patients with severe chronic kidney disease

(GFR<30) have a 1% to 7% chance of developing NSF after exposure to gadolinium agents, especially high doses or multiple doses. There has been no report of NSF in patients with normal renal function. Therefore, estimated GFR is recommended to be obtained within six weeks of a Gadolinium-enhanced study in patients with renal disease, over 60 years of age, with hypertension, DM, or severe liver disease. In patients with GFR<15 ml/min/1.73m2, the risk of NSF is greatest and there therefore should be absolute avoidance of contrast MRI. Alternative examinations should be suggested. For patients with GFR that is abnormal but greater than 15ml/min/1.732m2, judicious use of the lowest possible doses of selected macrocyclic agents are recommended.

The intra-thecal contrast (MR cisternography) is used for the diagnosis of Cerebrospinal fluid (CSF) rhinorrhea and spontaneous intracranial hypotension (SIH), which imply an abnormal communication between the subarachnoid space and nasal cavity or spinal canal. For these patients, confirmation, localisation and characteristics of the actual site/ sites of CSF leak are challenging but important for treatment planning.[4-10] Conventional myelography is now obsolete. Radionuclide cisternography (RC) has radiation hazards and poor spatial resolution. CT scan is sensitive for bony lesions but impossible to confirm the site of active CSF leak. Non-enhanced MR imaging has some use in demonstrating CSF fistulae but with relative high frequency of false positive findings of up to 42% and also false negatives.[5, 23-24] CT cisternography is more reliable. It can identify the spinal level of a CSF leak in 67% of patients compared with 50% and 55% with spinal MRI and RC respectively[21-22] but requires scanning of the whole spine and skull base. The patients will receive a considerable dose of radiation. The bony structures may also partially obscure the subtle site of CSF leakage.

MR cisternography can demonstrate the site of CSF leakage but with no radiation or bone artifacts. It requires a lumbar puncture and followed by a single low-dose gadolinium injection into the lumbar subarachnoid space. Many studies showed the relative safety and feasibility of low-dose gadolinium-enhanced MR cisternography in confirming and determining the focus of active CSF leaks. The results of initial human studies also revealed that the procedure do not manifest clinical evidence of gross physical or neurologic abnormalities, CSF changes, or electroencephalographic alterations.[11-18] The adverse reactions are rare, including nausea/vomiting, headache, anaphylactoid reaction and seizure.

Conclusion

Contrast agents used for CT, MRI scan and cerebral angiography play an important role in neuro-imaging. Although they are safe for use, adverse reactions may happen and therefore any contrast should be used judiciously. Recently, MR cisternography is more often used for the investigation of SIH and CSF rhinorrhea.

(with acknowledgment of Mr. Raymond Lee).

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Cavernous Haemangioma of Brain

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Introduction

Intracranial cavernous haemangioma (CH) was an uncommon pathology before the introduction of MRI examination. It was regarded as a mysterious pathology in neurological diagnosis because the lesion could not be easily revealed by cerebral angiogram or CT scan. It was therefore given the description of angiographically occult vascular malformation or cryptic vascular malformation.

When MRI became a common screen examination, CH became a common pathology identified in a routine MRI examination of the brain.

Pathology

CH consists of a group of thin walled vessels as discrete, lobular and well circumscribed lesions inside the parenchyma of the brain. Grossly it appears as a raspberry-like lesion red to purple in colour (Figure 1).

Microscopically it consists of dilated thin walled capillaries with variable thin fibrous adventitia devoid of smooth muscle and elastin. There is no brain parenchyma between the vascular channels. Haemosiderin deposits are always present inside the surrounding normal parenchyma indicating that diapedesis of red blood cells is a common event in all CHs.

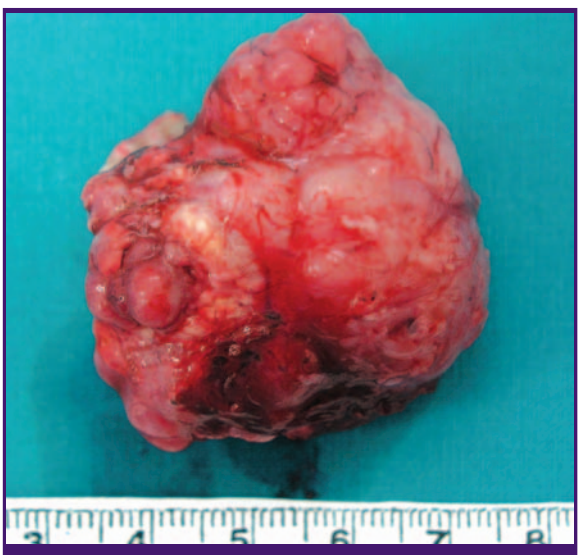

Figure 1 - Cavernous haemangioma after surgical removal as raspberry-like lesion.

Diagnosis

Clinical diagnosis of intracranial CH is impossible. Skull x-rays may occasionally reveal fine calcifications at the lesion but the finding is not diagnostic.

CT scan may be totally normal in most CHs. Hyperintense signals may be seen if old blood is accumulating. Enhancement after contrast CT scan is variable and is usually not diagnostic.

CH is well known to be invisible in cerebral angiograms and is therefore called angiographically occult. Occasionally venous pooling at the lesion may be seen and is again not diagnostic.

MRI is the most important imaging study for confirmation of the diagnosis (figure 2). In the absence of overt haemaorrhage, most CHs appear as a hyperintense lesion with a faint hypointense rim in T1 and lobulated heterogenous signals with a hypointense rim in T2. The most diagnostic image appears in the gradient-echo MRI which produces a blooming artifact from the magnetic susceptibility effect of haemosiderin. CH therefore appears characteristically as a dense hypointense signal in the gradient-echo MRI.[9]

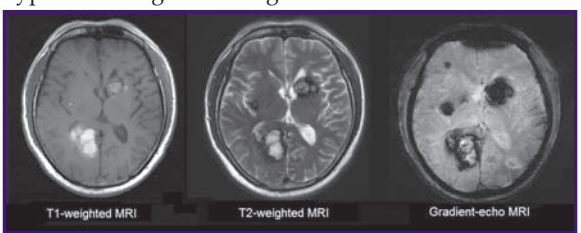

Figure 2 - Cavernous haemangioma appears in T1, T2 and gradient-echo MRI.

Epidemiology

CH of the brain is found in 0.1% to 4 % of all vascular malformations of the brain. In 4,068 cases of prospective autopsy study, McCormick identified CH in 0.4%, arteriovenous malformations in 0.6%, telangiectases in 0.7% and venous malformations in 2.6%.[8]

There is no sex prevalence and most symptomatic cases are found between 20 to 40 years old.

About 75% of the lesions are located in the supratentorial region (1/4 in the frontal and 1/6 in the temporal lobes) and 25% in the infratentorial region (50% in the pons or brainstem).

Multiple lesions are reported in 8 to 18 % of cases (figure 3). Multiple CHs are common in the south-western part of the United States among Hispanic patients. Familial incidence is reported in 50% of multiple CHs and an autosomal dominant inheritance with variable penetrance is suspected. There is no convincing hereditary link with single lesions.[4]

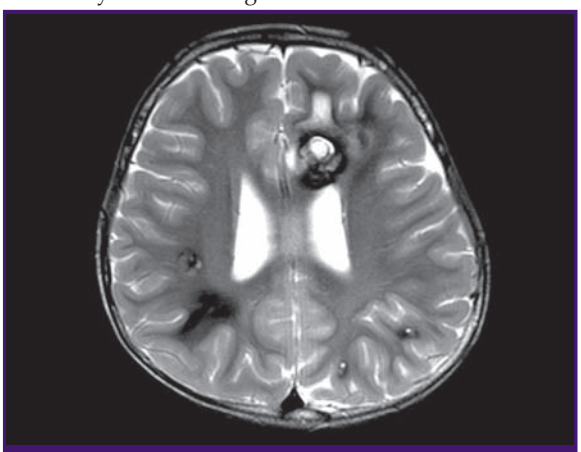

Figure 3 - Multiple Cavernous haemangioma

Clinical Presentation

The majority of CHs are found incidentally with no clinical symptoms. It is associated with headaches less commonly than arteriovenous malformations. Two major clinical symptoms related to CH are bleeding and epilepsy.

Haemorrhage in CH

The presence of haemosiderin around the lesion is commonly found and represents diapedesis of red blood cells through the thin walled vessels. This results in surrounding gliosis with a fibrotic layer covering the lesion. Minor intralesional bleeding and thrombosis are also commonly found in incidental cases. All these findings should not be considered as overt haemorrhage.

Robinson defined overt haemorrhage as acute or subacute blood accumulation outside the haemosiderin ring of the lesion (Figure 4). With this definition, the incidence of overt haemorrahage in virgin cases is estimated at 0.7% per lesion per year. However the risk of rebleeding after an overt haemorrhage is 25% in one year without treatment.[6,7,10] Rebleeding after the first haemorrhage is often related to physical exertion. Incidents of overt haemorrhage are more common in females. A higher chance of haemorrhage during pregnancy has been suggested. The consequence of overt haemorrhage is seldom fatal. The presentation depends on the location of the lesion.

Overt haemorrhage in supratentorial lesions is commonly associated with progressive hemiparesis and is often misdiagnosed as tumours in initial CT scan. Occasionally extensive intraventricular haemorrhage is found in periventricular lesions (figure 5).

Bleeding in the brainstem from CH is more serious and sixth nerve palsy with diplopia is a common initial presentation followed by multiple cranial nerve palsy, ataxia and long tract signs. Coma and life threatening brainstem insult can occur as a consequence of recurrent bleeding.

The chance of haemorrhage is independent of the lesion size. However bleeding from a large CH often results in significant neurological impairment that needs surgical treatment.

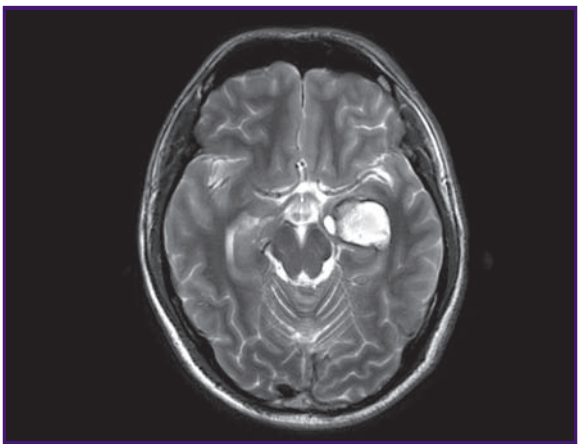

Figure 4 - Overt haemorrhage form cavernous haemangioma with acute blood outside the haemosiderin ring.

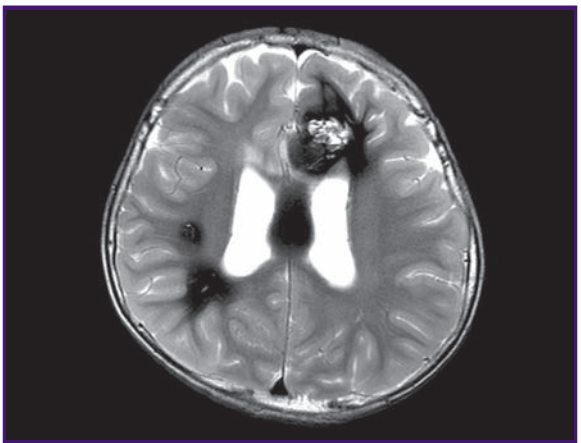

Figure 5 - Bleeding into third ventricle from a periventricular cavernous haemangioma.

Epilepsy in CH

The actual incidence of epilepsy in patients with supratentorial CH is unknown. More incidents of epilepsy should be found in patients with CH at the hippocampus and motor area (figure 5). The abnormal vessels themselves are not epileptogenic. The surrounding tissue is rendered epileptogenic with the effect of pressure and trophic factors such as haemosiderin.

Treatment

Most CHs are diagnosed as incidental findings in MRI during investigation for other problems. As the risk of spontaneous bleeding is low and seldom catastrophic, treatment is not required if there is no clinical

symptoms. However a detailed history is often required to exclude the presence of complex partial seizure which is often ignored by patients.

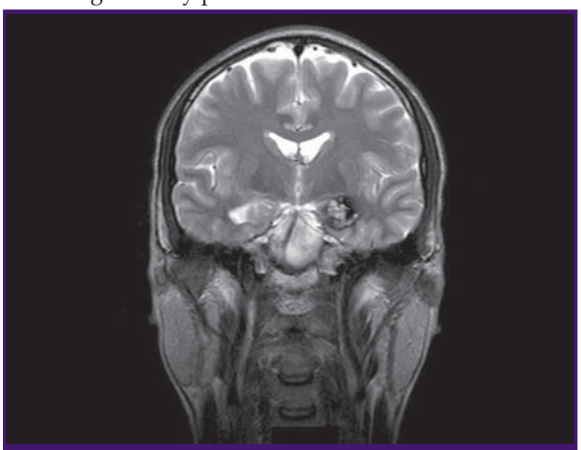

Figure 6 -avernous haemangioma at the hippocampus with epilepsy.

Conservative Treatment

Conservative treatment for overt bleeding in CH is often successful if neurological impairment is not significant. If rebleeding is minimised by the reduction of physical activities and tranexamic acid, neurological improvement is usually seen in a few weeks to a few months (figure 7). The patient is advised to withhold all activities that will increase venous pressure in the subsequent year.

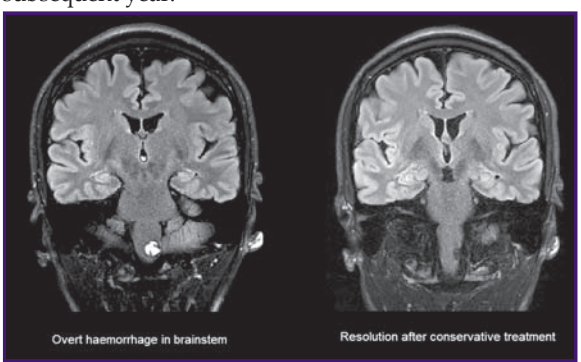

Figure 7 - Bleeding in a brainstem cavernous haemangioma (a) and resolution of blood signal after conservative treatment (b).

Surgical Treatment

Surgical removal is the only effective method for elimination of a CH. It is not a difficult procedure event in the brainstem. The existing gliosis around the CH serves as a good layer for microscopic dissection of the lesion without major damage to normal tissues. The use of navigator and microscopy allows safe removal of deep seated lesions.

However the indication for surgery must be justified. The common indication for surgical removal is a significant neurological impairment as a result of repeated haemorrhages resulting in a mass effect.

Epilepsy Treatment

CH is commonly found in investigations for poorly controlled epilepsy, especially in the hippocampus. Surgical removal of the CH for control of epilepsy is suggested if the epileptic focus is confirmed by electrophysiology studies. Good surgical result in the control of epilepsy has been reported at 70% complete seizure free after operation.[2, 3]

Removal is usually performed with clearance of the surrounding haemosiderin-loaded brain parenchyma in order to remove the epileptogenic zone. However there is a controversy in this point because no significant difference is found between surgical removal of the lesion alone and removal of surround haemosiderin.

Radiosurgery

The use of radiosurgery such as the Gamma Knife for treatment of CH remains debatable. There is a lack of convincing evidence in the literature to support the effectiveness of radiosurgery for the elimination of CH. The benefit of such therapy must be carefully evaluated against the potential radiation toxicity.[1, 5]

Summary

Cavernous haemangioma of brain is a common incidental finding in MRI examinations. The associated clinical events are haemorrhage and epilepsy. The risk of overt haemorrhage is 0.7% per lesion per year for virgin cases. Active treatment is usually not required for asymptomatic cases. Surgical removal is the only confirmed effective method for elimination of the CH and is indicated for progressive neurological impairment from repeated bleeding and uncontrolled epilepsy.

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Management of Intracranial Cerebral Arterial Stenosis

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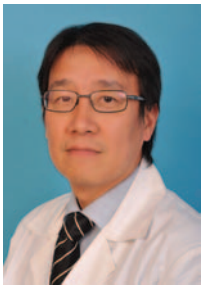

Dr. Wai-man LUI

Introduction

Intracranial arterial stenosis is used to be thought of as an uncommon cause of ischaemic stroke in Western literatures, accounting for about 10% in whites.[1-2] Wong in 2006[3] reported that larger artery intracranial stenosis affecting the middle cerebral artery, intracranial portion of the internal carotid artery, vertebrobasilar artery and posterior and anterior cerebral artery is more common in Asian patients. In fact, it is estimated to account for 33-50% of strokes and 50% of transient ischaemic attacks in the Chinese population; it was also found in 47% of patients with stroke in Thailand; and it was significant in approximately 48% of patients with stroke in Singapore.

Natural History

There are several important clinical trials that would give a clearer picture of the risks of strokes in patients having a large intracranial artery stenosis. The Extracranial-Intracranial (EC-IC) Bypass Study[4] provides prospective data on the risk of stroke in patients with symptomatic carotid siphon or middle cerebral artery (MCA) stenosis. In this trial, patients with carotid siphon or MCA stenosis who were treated medically (management of risk factors and 1300 mg/d aspirin) had an annual stroke rate of 8% to 10%.

Patients with symptomatic intracranial vertebral artery or basilar stenosis are at a higher risk of stroke, MI, or sudden death. Upon following up of 68 patients with 50-99% stenosis in the vertebrobasilar arteries for a median period of 13.8 months , 15 patients (22%) had an ischaemic stroke (4 fatal), 3 patients (4.5%) had a fatal myocardial infarction (MI) or sudden death. Overall, the estimated stroke risk of patients having a severe degree of arterial stenosis ranged from 10-20% yearly.

Medical treatment of Atherosclerotic Intracranial Arterial Stenosis

Antiplatelet vs. Anticoagulant

Calpan[5] proposed a pathophysiological rationale for anticoagulation to suggest warfarin is a common treatment choice for symptomatic intracranial stenosis.

However, the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial[6] showed that aspirin was safer and as effective as warfarin for stroke prevention in patients with symptomatic intracranial stenosis. WASID was stopped early after a mean follow-up of 1.8 years because of higher rates of death and major haemorrhage in the warfarin arm. The primary end point of ischaemic stroke, brain haemorrhage or vascular death, occurred in 22.1% of patients assigned aspirin and 21.8% of those in the warfarin group. The rates of myocardial infarction or sudden death were also higher in the warfarin arm. Even in the vertebrobasilar arterial stenosis with a higher risk of stroke, there is no clear evidence of any benefit of warfarin over aspirin.

Other antiplatelet agents (e.g. clopidogrel and combination of dipyridamole/aspirin) have been shown to have similar stroke recurrence rates in patients with various underlying causes of stroke and in a subset of patients with large artery atherosclerosis in the Prevention Regimen for Effectively Avoiding Second Strokes (PRoFESS) Study.[7] In summary, aspirin should be the drug of choice unless not tolerated by the patient.

Endovascular Therapy

Angioplasty and stenting have emerged as therapeutic options for symptomatic intracranial stenosis over the past few decades. Initially the risk of angioplasty was very high by borrowing the hardware from the cardiologists. Since that time, advances in microcatheter and balloon technology, the high risk of recurrent strokes in patients with intracranial stenosis despite medical management in WASID, and the success of endovascular treatments for other intracranial diseases have led to a renewed interest in intracranial angioplasty and stenting.

Angioplasty alone

Retrospective angioplasty studies reported high technical success rates with reduction of stenosis to <50%, but the 30-day rate of stroke or death has varied widely (4-40%). Restenosis rates after angioplasty have been reported between 24-50%.[8]

Overall, available data on intracranial angioplasty suggest that it can be performed relatively safely in stable patients, but the long-term outcome after angioplasty has not been prospectively studied. Moreover, there are numerous technical drawbacks to angioplasty including immediate elastic recoil of the artery, dissection, acute vessel closure, residual stenosis after the procedure, and high restenosis rates.

Angioplasty plus Stenting

The Wingspan system is currently the only FDA-approved device for treating symptomatic intracranial stenosis. In 2005, the Wingspan Stenting System (Boston Scientific) was approved by the FDA for use under an HDE (humanitarian device exemption) in patients with symptomatic intracranial stenosis who are refractory to medical therapy. Hong Kong was also one of the study sites. It was a prospective multicentre international Phase I trial which included 45 patients with symptomatic 50% to 99% intracranial stenosis who had recurrent strokes despite antithrombotic therapy. The technical success rate was 97.7% and the 30-day stroke or death rate was 4.5%. The 1-year rate of ipsilateral stroke was 9.3%. The restenosis rate was 7.5% at 6 months and none was symptomatic.[9]

In WASID the most important baseline predictors of stroke in the territory were severity of stenosis and time from qualifying event to enrollment. The rate of stroke in the territory in patients with >70% stenosis was 18% at 1 year (95% CI =13% to 24%) vs. 7% at 1 year (95% CI = 5% to 10%) in patients with <70% stenosis. The National Institute of Health (NIH) recruited patients from sixteen medical centres enrolled consecutive patients being treated with a Wingspan stent in this registry between November 2005 and October 2006. A total of 129 patients with symptomatic 70-99% intracranial stenosis were enrolled. The technical success rate was 96.7%. The frequency of any stroke, intracerebral haemorrhage, or death within 30 days or ipsilateral stroke beyond 30 days was 14.0% at 6 months. The frequency of >50% restenosis on follow-up angiography was 13/52 (25%). The results indicate that the observed rates of any stroke or death within 30 days or stroke in the territory beyond 30 days are similar in the two groups up to 3 months but diverge afterwards (lower in the stented patients).

Comparison of the event rates in high-risk patients in WASID vs. this registry does not rule out either that stenting could be associated with a substantial relative risk reduction (e.g., 50%) or has no advantage compared with medical therapy. Further randomised control study is required.

Future Directions

The best treatment for prevention of another stroke or TIA in patients with narrowing of one of the arteries in the brain is uncertain. There are several aspects that clinicians should be focused and require further research.

Aggressive management of risk factors

In WASID, elevated blood pressure was significantly associated with an increased risk of ischaemic stroke.[6] Raised low density lipoprotein (LDL) was also strongly associated with poor outcomes in patients, because 25.0% of patients with LDL >115 mg/dL had the primary end point compared with 18.5% of patients with a mean LDL<115 mg/dL Among the mere 10% of patients with mean LDL <70 mg/dL only 7% had a primary end point compared to 23% of the patients with LDL >70 mg/dL (P<0.09).

Recent research has also indicated a benefit in the prevention of recurrent strokes by Intensive Medical Therapy, which is defined as treating risk factors for stroke like high blood pressure, elevated LDL (low density lipids - the "bad" form of cholesterol) and diabetes.[10]

Clinical trial

The Stenting and Aggressive Medical Management for Preventing Recurrent stroke in Intracranial Stenosis (SAMMPRIS) Trial is a NIH sponsored, on-going randomised trial at 60 US sites designed to determine whether angioplasty and stenting plus aggressive medical management is superior to aggressive medical management alone for the prevention of recurrent stroke in patients with 70% to 99% stenosis of a major intracranial artery.[11] Patients will be randomised 1:1 to either arm. Aggressive medical management in SAMMPRIS will consist of dual antiplatelet therapy (aspirin+clopidogrel) for 90 days in all patients. All patients will also receive protocol driven risk factor management targeting a LDL <70 mg/dL and systolic blood pressure <140 mm Hg (<130 if diabetic) and a comprehensive lifestyle modification programme to assist with weight reduction, exercises, smoking cessation, and nutrition.

Summary

Symptomatic atherosclerotic intracranial stenosis is a high-risk condition. WASID showed that aspirin is safer and as effective as warfarin for preventing recurrent strokes. Angioplasty and stenting cannot be justified in patients with < 70% stenosis, given the low risk of stroke in the territory of a stenotic artery (6 % at 1 year) and the inherent risk of current technology. Patients with severe stenosis, recent ischaemic symptoms and an NIH stroke scale score of > 1, and females are at the highest risk for strokes, and therefore have the greatest likelihood of benefiting from angioplasty and stenting.[12] The linear relationship between the degree of stenosis and stroke risks with medical therapy also supports a mechanical approach to revascularisation. At present, however, there is no level 1 evidence to support angioplasty and stenting for patients with symptomatic intracranial atherosclerotic disease. A randomised controlled trial is needed to prove the efficacy of this therapy. It should also be noted that these patients as a group have frequent vascular risk factors and will require aggressive medical management. In addition, rates of restenosis and the clinical consequences of restenosis will need to be closely monitored in future studies.

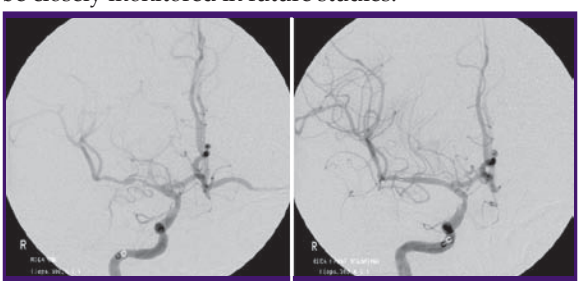

Fig. 1a. 56-yr old lady presented with recurrent TIA. Cerebral DSA showing a 90% right MCA stenosis.

Fig. 1b. Cerebral angiogram showing complete recanalization of right MCA after stenting with a 3mm x 15mm Wingspan stent.

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Incidental Sellar Lesions

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Introduction

Magnetic resonance imaging (MRI) studies and computerised tomography (CT) are becoming more readily available and widely used modalities of clinical investigations. An increasing number of mass lesions involving the sellar region are now being detected on MRI and CT performed for reasons unrelated to pituitary diseases. These incidental sellar lesions include neoplastic conditions such as pituitary adenomas, meningiomas, craniopharyngiomas, gliomas and metastases. Non-neoplastic lesions may include Rathke's cleft cysts, carotid artery aneurysms, granulomas, hypophysitis, mucocoeles, and other uncommon pathologies. This article will focus on the two most commonly encountered entities - incidental pituitary adenomas[1] and Rathke's cleft cysts.[2]

Pituitary Incidentaloma (PI)

The incidence of PI is around 10% at autopsy, distributed equally throughout the age groups and between the sexes. The reported incidences of PI on contrasted MRI range widely from 2 to 34 %. It is of note that some normal individuals may have 'normal pituitary hypertrophy' that exceeds the normal size boundary of 9 mm, and which may occasionally mimic a PI. Artefacts such as beam-hardening effects on CT and susceptibility distortions on MRI may also cause diagnostic difficulties.

PI may be functioning (hormone-secreting) or non-functioning lesions. However, about 75% of the latter are in fact gonadotroph adenomas, and others may stain positively for adrenocorticotropic hormone (ACTH), growth hormone (GH), prolactin (PRL) or thyrotropin (TSH) singly or in combinations. These are sometimes referred to as 'silent' corticotroph, somatotroph, lactotroph, thyrotroph or mixed adenomas, respectively.

Clinical presentation

The fact that these adenomas are incidental findings does not necessarily mean that they are clinically silent. A detailed history and clinical examination are essential for the detection of subtle symptoms and signs that may suggest hormonal hypersecretion, hypothalamic/pituitary hypofunction, and visual field deficits. Rarely, PI may be associated with hydrocephalus due to third ventricular obstruction, and cranial nerve palsy due to cavernous sinus involvement.

Evaluations of pituitary incidentaloma

PI diagnosed on CT should be further evaluated by contrasted MRI of the pituitary region. The visual field should be formally assessed for optic chiasm or optic nerve compression - the former being classically associated with bitemporal hemianopia, whilst the latter may cause loss of vision in the ipsilateral eye and a junctional scotoma in the contralateral eye.

Symptoms of hypersecretion may be very subtle, and biochemical evaluation is warranted even when no clinical signs and symptoms are detected. This is particularly relevant for the diagnosis of silent somatotroph and corticotroph adenomas. Although it is not clear whether such lesions are associated with the increased risks for metabolic and oncological complications like their symptomatic counterparts, there is evidence to suggest that these tumours may have a worse prognosis than those which produce overt symptoms. Early detection and timely management is therefore important. In general, up to 40% of macroadenomas are associated with hypopituitarism and careful endocrinological evaluation is indicated. The reported incidence of hypopituitarism in microadenomas may range from 0 to 50%, and it is controversial whether routine screening is necessary.

A detailed discussion of pituitary evaluation is beyond the scope of this article. Briefly, screening tests for Cushing's disease include the overnight dexamethasone suppression test, the 24-h urinary free cortisol level and, more recently, a midnight salivary cortisol level. The latter has greater than 93% specificity and sensitivity. A random serum insulin-like growth factor-1 (IGF-1) level is useful for the screening of acromegaly. Hyperprolactinaemia may result from genuine hypersecretion or pituitary stalk dysfunction secondary to tumour compression. A prolactinoma commonly causes a markedly raised PRL level of greater than five times the upper limit of normal. A very large prolactinoma may produce enough PRL to saturate the antibodies in the assays (the 'hook effect'), resulting in a misleadingly low serum PRL level.

Other tests include total testosterone level in men, oestradiol in women, early morning serum cortisol for hypocortisolism, and T4 and TSH for secondary hypothyroidism. Lutenising hormones (LH), follicular stimulating hormone (FSH) and alpha-subunit may also be tested as part of the assessment of the gonadal axis. Diabetes insipidus is uncommon before surgery in the case of PI. The selective loss of a single pituitary hormone (e.g., ADH or ACTH) is even rarer and, when

associated with a thickened pituitary stalk, should raise the suspicion of hypophysitis.

Management of incidental pituitary adenomas

Tumours which are hypersecreting require treatment. For non-functioning PIs, the indications for surgery include the initial tumour size, the presence of mass effect and tumour progression. Not all PIs grow. For microadenomas, the lesion size may increase in around 10%, decrease in 6 %, and remain static in over 80% of patients. Upfront surgery is generally not indicated and patients may be followed by repeated MRI, initially at 6-month, then at year-1, -2 and -5. For macroadenoms, close to 24 to 50% of cases will increase in size. The tumour volume doubling time has been found to vary widely from 0.8 to 27.2 years, however. Most authorities advocate surgery for incidental macroadenomas given their greater propensity for growth. Macroadenomas which are managed conservatively should be followed up very closely.

Patients with established visual defects certainly require treatment. Surgery may also be considered for lesions abutting the optic chasm in young patients even in the absence of visual field loss. It is controversial if hypopituitarism alone would indicate surgery since although hypopituitarism is potentially correctable with tumour resection, the latter may also cause iatrogenic loss of function. Careful counselling is needed especially for female patients of reproductive age who may have concerns about future child-bearing. Some authorities also recommend surgery for lesions which show evidence of recent haemorrhages.

The treatment of choice for most PI is transsphenoidal removal. Recent development has seen the increasing use of endonasal endoscopic transsphenoidal surgery as a minimally invasive alternative to the conventional transseptal microscopic approach. Medical therapy alone with dopamine agonists or octreotide is effective for only 10 to 20% of non-prolactinomas but may be considered for patients who are unfit for or reluctant to have surgery. There is at present not enough evidence to support radiosurgery as a standard first-line treatment for PI.

Rathke's Cleft Cyst (RCC)

Rathke's cleft cysts are benign lesions commonly thought to be the remnants of the Rathke's pouch. The cyst content may vary from clear CSF-like to thick mucoid-like materials. Histologically, RCCs are lined by single or pseudo-stratified cuboidal or columnar epithelium although squamous metaplasia can occur that may mimic craniopharyngiomas. The incidence of RCC found at autopsy is around 13 to 33%.

It is important but at times difficult to distinguish between RCC, cystic pituitary adenoma and craniopharyngioma radiologically. These conditions have different natural histories and require very different treatment approaches. On CT, all may appear as hypo- or isodense lesions although craniopharyngiomas are more likely to show calcifications. On MRI, both RCC and craniopharyngiomas may show a wide range of intensities on T1- and T2-weighted images, depending on the nature of the cyst contents. For example, higher protein concentrations may lead to shortened T1 and T2 relaxation times, increasing the intensity of T1-weighted and decreasing the intensity of T2-weighted images. Rim enhancement may be seen in a number of RCCs and may be attributed to the presence of a circumscribed area of pituitary tissue, inflammation, haemosiderin, cholesterol crystals, or squamous metaplasia in the cyst wall. A small intracystic nodule corresponding to proteinaceous deposition that has high T1 and low T2 intensities, and which does not enhance, is a very characteristic appearance of RCC. A RCC with hyperintense signals on both T1- and T2-weighted images may also resemble a haemorrhagic pituitary adenoma. Recently, diffusion weighted images (DWI) have been found to be useful for the distinction between these conditions. [3]

The majority of RCC are asymptomatic. The common clinical presentations resemble those of pituitary adenomas, and may include headache, visual field loss, and hypopituitarism. The evaluation of a newly diagnosed RCC should follow those listed above for PI except for hypersecretion, which is not a feature of RCC.

The propensity of growth is relatively low for RCC. The majority would remain static. Asymptomatic RCCs should be observed. Even those with mild symptoms may be managed conservatively because some of these lesions have been known to shrink or disappear spontaneously. RCCs with significant symptoms or increase in size can be readily treated surgically by means of transsphenoidal removal. The endocrine outcome following surgery, however, remains suboptimal - reversal of pituitary deficits is uncommon, and diabetes insipidus may occur not infrequently. The reported risks of recurrence may range from 8% to close to 40%. Surgical biopsy is also indicated when a histological exclusion of other sellar pathologies is required.

Summary

Both PI and RCC should receive thorough imaging, visual, and hormonal evaluations preferably by endocrinologists and neurosurgeons. Due to the benign nature of these conditions, most can be managed conservatively. The main indications for treatment include the presence of visual field deficits, hormonal hypersecretion, and disease progression. Transsphenoidal resection is the treatment of choice in the majority of cases.

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中醫藥理論精華 與
中西醫結合專題療護課程

（香港護士持續教育學分項目）

中醫藥治療已成為本港醫療體系的一環，讓病人在醫治上有多一項選擇却病調身。學會藉其中西醫學的資材，應護理知識的需要舉辦本課程，以深入淺出的方法介紹中醫理論與運用、藏象學、診斷學、中藥學、方劑學及針灸學等中醫藥理論的精華；而療護中西醫專題方面會特別探討痛症、骨節勞損、腸胃病和皮膚病。課程另一特色將因應現時中西醫護理所遇到的困難而設計相向討論會，講者與學員以互動形式討論臨床的解決辦法。課程讓學員增進中醫療護知識，同時建立繼續進修的良好基礎。

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MRI Study of Brain and Incidental Finding of White Matter Hypertensities and Microbleeds

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Introduction

As with any diagnostic test that screens for diseases, the risks of imaging need to be outweighed by the benefits of identifying a treatable disease with acceptable sensitivity and specificity. Plain skull X-ray with its inherent ionising radiation is therefore not recommended for routine assessments of the central nervous system except in the detection of skull lesions. CT used to be the modality of choice for non-invasive assessment of all brain diseases. However with the advent of MR, CT is now mainly employed in the setting of emergency or trauma in which CT serves as the quickest imaging modality for the detection of acute intracranial bleeding (traumatic or non-traumatic) and skull fractures.

MR studies of the brain is considered the better imaging technique than CT for two reasons. Firstly, MR has a much higher contrast resolution when compared with CT for exquisite depiction of normal anatomical structures and pathologies of the brain. Unlike CT angiography, intravenous contrast injection is not required for MR angiography. This is advantageous in patients with impaired renal function, contrast allergy or no intravenous venous access. Secondly, MRI does not involve radiation exposure. CT brain scans which use x-ray to produce images may expose patients to about 2 mSv of radiation which is twenty times that of chest x-rays. In simple terms, the radiation exposure from one non-contrast CT brain study is equivalent to the amount of background radiation one experiences in about 8 months, considering that the average person in Hong Kong receives an effective dose of about 3.2 mSv per year from naturally occurring radioactive materials and cosmic radiation. MR imaging has thus replaced CT in the imaging of the brain except in patients with contraindications to MR, for instance, patients with pacemakers or metallic devices.

While MRI study of the brain is increasingly utilised in clinical practice and health screening owing to its increased availability and recognition among clinicians, incidental findings showing abnormalities of potential clinical relevance that are unrelated to the purpose of the study are unexpectedly discovered on these MR brain studies. Reports and studies on the prevalence of these incidental abnormalities are growing in number. However, the clinical course of some of these incidental findings is still uncertain, and their management is not standardised. It is the purpose of this review article to categorise the major groups of incidental findings and to discuss their clinical relevance.

Prevalence of Incidental MR Brain Findings

No large scale study of incidental MR brain findings is available in Hong Kong but the significance of this issue is well reflected in studies conducted abroad. In a recent systemic review with meta-analysis on incidental findings in brain magnetic resonance imaging by Morris et al[1], it was found that neoplastic incidental brain findings had a prevalence of 0.7% (135 of 19559 people out of 16 studies) with increased prevalence with age. The non-neoplastic incidental findings were even more prevalent at 2.0% (375 of 15559 in 15 studies). The overall prevalence of incidental brain findings on MRI was 2.7 %, equivalent to one for every 37 subjects scanned.

Another remarkable observation is that the prevalence was further increased from 1.7% to 4.3% when the sensitivity was enhanced by more state-of-the-art MR scanners and higher resolution MRI sequences, including MR angiography. Nowadays, advanced sequences such as three-dimensional T1 spoiled or fluid attenuated inversion recovery (FLAIR) or MR angiography are commonly included in routine clinical scans, leading to discoveries of more incidental findings.

This overall prevalence of 2.7% pointed out by the Morris group was in fact 'conservative' as their study had already excluded the most common incidental findings, namely the white matter hyperintensities (WMHs), silent brain infarcts and brain microbleeds (BMB). In a study of MRI brain scans on patients of the general population[2], it was found that the prevalence of asymptomatic infarcts was 7.2% (145 of 2000).

Incidental findings on brain MRI studies can be broadly divided into three groups; vascular, neoplastic and non-neoplastic cystic lesions. This article mainly focuses on brain white matter hyperintensities and microbleeds. Detailed discussion on other vascular lesions (vascular malformations and aneurysms), incidental neoplasms or non-neoplastic cysts will be covered by other authors.

White Matter Hyperintensites (WMH) & 'Silent' Infarcts

The most common incidental vascular lesion is white matter hyperintensities (WMHs). These lesions are sometimes referred to as leukoaraiosis or age-related white matter change (ARWMC). MRI is highly sensitive for the detection of white matter pathologies with

conventional PD or T2 weighted spin echo or fast-spin echo sequences but are even more conspicuous on fluid-attenuated inversion recovery (FLAIR) images. FLAIR sequences have the advantage of making cerebrospinal fluid (CSF) looks dark while the white matter lesion still appears bright. This improves the lesion conspicuity, especially in areas close to the CSF spaces such as periventricular areas (Figure 1c).

Pathologically, WMH or ARWMC is an area of myelin pallor, tissue rarefaction associated with loss of myelin and axons, and mild gliosis. These lesions are most commonly located in the deep white matter and often associated with disease of small vessels (intraparenchymal cerebral arteries and arterioles), which probably induce the WMH lesions through chronic or transient but repeated hypoperfusion of the white matter. The hypoperfusion results in an incomplete form of infarction with disruption of the blood-brain barrier, leading to chronic leakage of plasma into the white matter and activation of astrocytes.[3] Activated and swollen astrocytes, typically seen in areas of WMHs may contribute to the alterations commonly detected by MRI.

As the name ARWMC implies, the WMHs are dependent on the age of patients (Figure 1a and 1b). In the general population, their prevalence shows obvious positive correlation with age, ranging from only 4% in the age group of 45-59 to 6.8% in age group of 60-74. An even higher prevalence of 18.3% was found in the age group of 75-97[4,5].

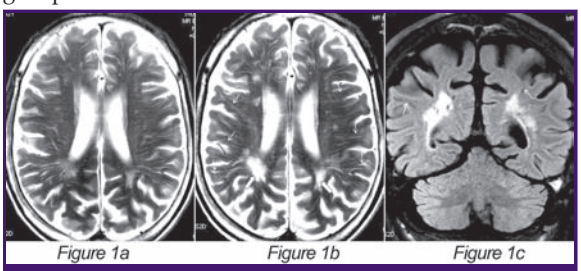

Figure 1 – Axial T2 weighted images of the same octogenarian taken 5 years apart showed obvious progression of the white matter hyperintensities (WMHs) in both the deep and periventricular white matter. The confluent periventricular WMHs are also more obvious in the parietal lobes (open arrow). These WMHs are well depicted on the coronal FLAIR images (figure 1c). Please note that the age-related prominent perivascular spaces (arrows on figure 1b) are confirmed to be CSF-filled linear structures on the FLAIR study (arrow on figure 1c) with no hyperintense signals.

WMHs are indeed more common and extensive in patients with symptomatic cerebrovascular diseases or cardiovascular risk factors which include hypertension, hypercholesterolaemia, hyperlipidaemia and diabetes mellitus among other less common causes. Numerous researches have been done on this subject with various systems to scale the WMH load either using visual assessment (Figure 2) or quantitative analysis. Mild lesions are usually punctate lesions less than 5 mm in diameter. The more severe lesions are comprised of patchy confluent lesions in the periventricular and deep white matter.

A meta-analysis study by Debette et al[6] included 22 studies which evaluated the association of white matter hyperintensities with risks of stroke, cognitive decline, dementia or death. It was concluded WMHs were associated with an increased risk of stroke, dementia and death. An association with a faster decline in global cognitive performance, executive function, and processing speed was also suggested. WMHs therefore predict an increased risk of cerebrovascular events. The discovery of significant WM load on MR scan should prompt detailed screening for risk factors of stroke and dementia, especially in relatively young patients.

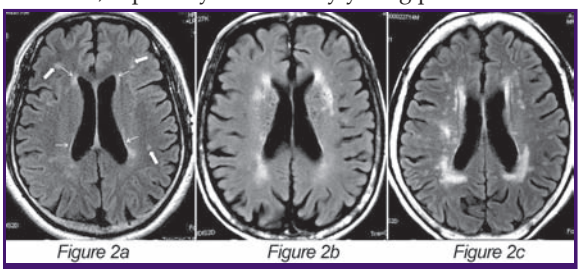

Figure 2 – Axial FLAIR images demonstrate different grades of white matter hyperintensities (WMHs) in the brain. Figure 2a in a 55-year-old lady shows mild degree of WMHs with only a few punctate hyperintense lesions in the subcortical and deep white matters (bold arrow). The normal age-related periventricular hyperintense smooth thin rims as well as triangular-shaped "caps" around the frontal horns (short arrow) are also depicted. Figures 2b and 2c in two different elderly gentlemen in their seventies show moderate and severe degrees of WMH with patchy confluent WMHs in the deep and periventricular white matters, most obvious in the parietal lobes.

Brain Microbleed (BMB)

Brain microbleeds (BMBs) are typically seen as tiny homogeneous foci of low signal intensity with a 'blooming' appearance on magnetic resonance imaging gradient echo (GRE) T2* sequences. The recently introduced susceptibility-weighted imaging (SW) can even detect these microhaemorrhages better than a gradient-recalled echo sequence due to its high sensitivity to blood degradation products[7] as in Figure 3.

Pathologically, BMBs are found in areas fed by deep perforating arteries showing lipohyalinosis and occasional amyloid deposits[8] or ruptured arteriosclerotic microvessels[9]. BMB can therefore be considered a biomarker of bleeding-prone small-vessel diseases, in particular hypertensive small-vessel arteriopathy and cerebral amyloid angiopathy (CAA).

Cordonnier et al[10] systematically reviewed and critically appraised 54 studies of 53 case series involving 9073 participants, 4432 of whom were people with cerebrovascular diseases. The prevalence of BMBs was 5% in healthy adults, 34% in people with ischaemic stroke, and 60% in people with non-traumatic intracerebral haemorrhage (ICH). BMBs were more prevalent among recurrent strokes than first-ever strokes; recurrent intracerebral haemorrhage (ICH) than first-ever ICH.

BMBs are usually related to hypertensive illness, especially in the setting of uncontrolled or untreated patients. In elderly patients, old microbleeds can also be related to amyloid angiopathy which occurs mainly in older patients. In CAA, accumulation of amyloid β-protein renders vessel walls less elastic and more fragile, resulting in microhaemorrhages.

Although a definite relationship of BMB and use of antiplatelet treatment cannot be established, it may be prudent in taking extra caution in administrating this kind of drug to patients who have a significant degree of BMB on MR brain. There was one study [11] considering BMB a biomarker for bleeding-prone small-vessel diseases and might be associated with antiplatelet-related ICH. The risks of ICH could outweigh the benefits of antiplatelet therapy in patients with significant lobar microbleeds. In another local study of BMB as a risk factor for aspirin-associated ICH[12], it was found that BMBs are more frequent and more extensive in the intracerebral haemorrhage group than in the control group.

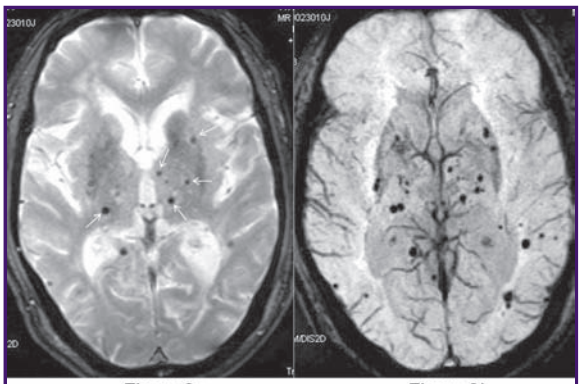

Figure 3a Figure 3b

Figure 3 – Axial GRE T2 weighted and susceptibility-weighted images of a 65 years old patient with poorly controlled hypertension. Multiple old microbleeds are demonstrated at the thalami and basal ganglia (arrow). The subcortical microbleeds are only vaguely seen on GRE T2* weighted sequence (figure 3a) but appear more conspicuous and numerous on the SWI.*

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News from Member Societies

1. **Hong Kong College of Paediatricians**
   Updated office-bearers for the year 2010-2011 are as follows: President: Prof. Pak-cheung NG; Honorary Secretary: Dr. Winnie Wing-yee TSE; Honorary Treasurer: Dr. Chi-sik CHAN

2. **The College of Dental Surgeons of Hong Kong**
   Updated office-bearers for the year 2010-2011 are as follows: President: Dr. Roch K.H. LEE; Honorary Secretary: Dr. Edmond H.N. POW; Honorary Treasurer: Dr. Alfred C.C. TSANG

3. **The Hong Kong Society for Colposcopy and Cervical Pathology**
   Updated office-bearers for the year 2010-2011 are as follows: President: Dr. Siu-keung LAM; Honorary Secretary: Dr. So-fan YIM; Honorary Treasurer: Dr. Alice CHAN

4. **The Hong Kong Society of Occupational and Environmental Medicine**
   Updated office-bearers for the year 2010-2011 are as follows: President: Dr. Mandy Mang-yee HO; Honorary Secretary: Dr. Joan Pui-chu FOK; Honorary Treasurer: Dr. Wai-man WOO

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

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

Date	Course No	Course Name	Target Participants	CME/CNE
01/04/2011 - 13/05/2011	C173	Certificate Course on Management of Common Psychiatric Disorders 2011	Medical and Health Professionals	9 CNE Points; CME/CPD Accreditation in application
06/04/2011 - 11/05/2011	C176	Certificate Course on Paediatric Nephrology 2011	General Practitioners, Health Care Providers and Public Who Are Interested in Common Kidney Problems in Children	9 CNE Points; CME/CPD Accreditation in application

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

(Effective from October 2009)

Venue or Meeting Facilities	Member Society (Hourly Rate HK$)			Non-Member Society (Hourly Rate HK$)		
	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays	Peak Hour	Non-Peak Hour	All day Sats, Suns & Public Holidays
Multifunction Room I *(Max 15 persons)*	150.00	105.00	225.00	250.00	175.00	375.00
Council Chamber *(Max 20 persons)*	240.00	168.00	360.00	400.00	280.00	600.00
Lecture Hall *(Max 100 persons)*	300.00	210.00	450.00	500.00	350.00	750.00
Non-Peak Hour: 9:30am - 5:30pm Peak Hour: 5:30pm - 10:30pm						
LCD Projector	500.00 per session					
Microphone System	50.00 per hour, minimum 2 hours					

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
		★HKMA Council Meeting 1	2	3	4	5
6	7	8	9	★FMSHK Officers' Meeting 10	11	★Refresher Course for Health Care Providers 2010/2011 ★Public Education Day at NTW district 12
★HKMA Certificate Course on Family Medicine 2011 13	14	★Allergic Airway Diseases and Asthma 15	★Hong Kong Neurosurgical Society Monthly Academic Meeting - Congenital craniocervical abnormalities & Open forum on the future development of neurosurgery as a specialty in HK 16	★HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2011 – Pain Management ★Economist Conferences: Healthcare in Asia 2011 17	★HKMA Shatin Doctors Network – Update on outpatient management of asthma ★Economist Conferences: Healthcare in Asia 2011 ★Certificate Course on Dental Nursing in Oral Surgery 18	19
★Certificate Course on Management of Drug Abuse Patients for Family Doctors 20	21	★FMSHK Executive Committee & Council Meeting 22	23	★Managing BPH in 2011 ★International Colorectal Disease Symposium 2011 ★中醫藥理論精華與中西醫給合專題療護課程 24	★HKMA Photographic Society - 1st Seasonal Photo Sharing & Competition 2011 - Submission Deadline ★International Colorectal Disease Symposium 2011 ★Certificate Course on Dental Nursing in Oral Surgery 25	★PPI Lecture Series_Acute Abdominal Pain ★International Colorectal Disease Symposium 2011 26
★Certificate Course on Management of Drug Abuse Patients for Family Doctors 27	28					

Date / Time	Function	Enquiry / Remarks
1 TUE 8:00pm	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. K Choi, Venue: HKMA Head Office (5/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
10 THU 2:30pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
12 SAT 2:30pm 3:00pm	**Refresher Course for Health Care Providers 2010/2011** Organiser: The Hong Kong Medical Association, Speaker: Dr. LEE Ching Yin, Venue: OLMH **Public Education Day at NTW district** Organiser:The Hong Kong Medical Association, Venue: Tuen Mun Town Plaza	Ms. Clara Tsang Tel: 2354 2440 2 CME Points Miss Carman WONG Tel: 2527 8285
13 SUN 2:00pm	**HKMA Certificate Course on Family Medicine 2011** Organiser: The Hong Kong Medical Association, Speaker: Dr. TAM Kui Fu, Stanley; Dr. WONG Wai Hong, Venue: QEH	Miss Viviane LAM Tel: 2527 8452 3 CME Points
15 TUE	**Allergic Airway Diseases and Asthma** Organiser: HKMA Kowloon West Community Network, Speaker: Dr. TAM Yat Cheung, Alfred, Venue: Panda Hotel	Ms. Candice TONG Tel: 2527 8285
16 WED 7:30am	**Hong Kong Neurosurgical Society Monthly Academic Meeting - Congenital craniocervical abnormalities & Open forum on the future development of neurosurgery as a specialty in HK** Organiser: Hong Kong Neurosurgical Society,Chairman:Dr. Sui-to WONG, Speaker:Dr. Chun-pong TSANG, Venue: Multifunction Room, Ground Floor, Block D, Queen Elizabeth Hospital	Dr. Gilberto LEUNG Tel: 2255 3368 Fax: 2818 4350 1.5 CME Points
17 THU 2:00pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2011 – Pain Management** Organiser: The Hong Kong Medical Association, Speaker: Dr. LI Ching Fan, Carina, Venue: The HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road Central, Hong Kong **Economist Conferences: Healthcare in Asia 2011** Organisation: The Economist Group, Venue: Harbour Grand Kowloon Hong Kong, Website:www.economistconferences.com/health2011	Miss Viviane LAM Tel: 2527 8452 1 CME Point Tel: (852) 2585 3312 Email:conferencesasia@economist.com
18 FRI 2:00pm 7:00pm - 8:30pm	**HKMA Shatin Doctors Network – Update on outpatient management of asthma** Organiser: HKMA Shatin Doctors Network,Speaker: Dr. LI Sing Tao, Thomas, Venue: Shatin **Economist Conferences: Healthcare in Asia 2011** Organisation: The Economist Group, Venue: Harbour Grand Kowloon Hong Kong, Website:www.economistconferences.com/health2011 **Certificate Course on Dental Nursing in Oral Surgery** Jointly organised by The Federation of Medical Societies of Hong Kong and The Hong Kong Association of Oral and Maxillofacial Surgeons Limited, Topic: Common Oral Diseases and Oral & Maxillofacial Surgery, Speaker: Dr. Ken Wai-kuen CHIU, Venue: Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Miss Carman WONG Tel: 2527 8285 1 CME Point Tel: 2585 3312 Email:conferencesasia@economist.com The Secretariat of The Federation of Medical Societies of Hong Kong Tel: 2527 8898 Fax: 2865 0345 Email:info@fmshk.org 9 CNE Points (Total 6 sessions)
20 SUN 1:00pm	**Certificate Course on Management of Drug Abuse Patients for Family Doctors** Organiser: The Hong Kong Medical Association, Speaker: Dr. CHAN Lam Fung, Lambert; Ms. CHUNG Yin Ting, Brenda, Venue: Tuen Mun	Miss Queenie LAM Tel: 2527 8285 4 CME Points
22 TUE 7:00pm - 10:00pm	**FMSHK Executive Committee & Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms. Sonia CHEUNG Tel: 2527 8898 Fax: 2865 0345
24 THU 7:00pm - 10:00pm	**Managing BPH in 2011** Organiser: HKMA Yau Tsim Mong Community Network, Speaker: Dr Szeto Shek, Petrus **International Colorectal Disease Symposium 2011** Organiser: Minimal Access Surgery Training Centre & Hong Kong Society for Coloproctology, Venue: HKEC Training Centre for Healthcare Management and Clinical Technology, Location: Pamela Youda Nethersole Eastern Hospital **中醫藥理論精華與中西醫結合專題療護課程 - Session 1:中醫理精華簡介與運用** Organiser: Hong Kong Association for Integration of Chinese-Western Medicine, Chairman: Dr. Yu Chau Leung, Edwin, , Speaker: 陳平順博士, Venue: SGN, G/F., Rm 08 Queen Elizabeth Hospital, 30 Gascoigne Road, Kowloon, Hong Kong	Ms. Candice TONG Tel: 2527 8285 CME Accreditation in Application Tel: 2595 6362 Fax: 2505 7101 Email: info@icds-hk.org Miss Y.C. YEUNG Tel: 3119 1858 Fax: 2301 2414 27 CNE Points (Total 9 sessions)
25 FRI 7:00pm - 8:30pm	**HKMA Photographic Society - 1st Seasonal Photo Sharing & Competition 2011 - Submission Deadline** Organiser: HKMA Photographic Society, Chairman: Dr. Amy PANG **International Colorectal Disease Symposium 2011** Organiser: Minimal Access Surgery Training Centre & Hong Kong Society for Coloproctology, Venue: HKEC Training Centre for Healthcare Management and Clinical Technology, Location: Pamela Youda Nethersole Eastern Hospital **Certificate Course on Dental Nursing in Oral Surgery** Jointly organised by The Federation of Medical Societies of Hong Kong and The Hong Kong Association of Oral and Maxillofacial Surgeons Limited, Topic: Minor Oral Surgery in Dental Office, Speaker: Dr. Julianna Cho-hwei LIEW, Venue: Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Miss Alice TANG Tel: 2527 8285 Tel: 2595 6362 Fax: 2505 7101 Email: info@icds-hk.org The Secretariat of The Federation of Medical Societies of Hong Kong Tel:2527 8898 Fax:2865 0345 Email:info@fmshk.org 9 CNE Points (Total 6 sessions)
26 SAT	**PPI Lecture Series_Acute Abdominal Pain** Organiser: HKMA Hong Kong East Community Network, Speaker: Dr. Tsang Kin Lun **International Colorectal Disease Symposium 2011** Organiser: Minimal Access Surgery Training Centre & Hong Kong Society for Coloproctology, Venue: HKEC Training Centre for Healthcare Management and Clinical Technology, Location: Pamela Youda Nethersole Eastern Hospital	Ms. Candice TONG Tel: 2527 8285 CME Accreditation in Application Tel: 2595 6362 Fax: 2505 7101 Email: info@icds-hk.org
27 SUN 1:00pm	**Certificate Course on Management of Drug Abuse Patients for Family Doctors** Organiser: The Hong Kong Medical Association, Speaker: Dr. CHEUNG Kin Leung, Ben; Dr. CHENG Chi Man; Ms. CHENG Oi Kwan, Silvia, Venue: Tuen Mun	Miss Queenie LAM Tel: 2527 8285 4 CME Points

Course / Meeting

24/02-28/04/2011	**中醫藥理論精華與中西醫結合專題療護課程** Organiser: Hong Kong Association for Integration of Chinese-Western Medicine, Chairman: Dr. Yu Chau Leung, Edwin, Venue: SGN, G/F., Rm 08 Queen Elizabeth Hospital, 30 Gascoigne Road, Kowloon, Hong Kong, Tel: 3119 1858, Fax: 2301 2414, 27 CNE Points (Total 9 Sessions)

CERTIFICATE COURSE FOR General Practitioners,
Health Care Providers and Public Who Are Interested in Common Kidney Problems in Children

CME/CNE Course **Course No. C176**

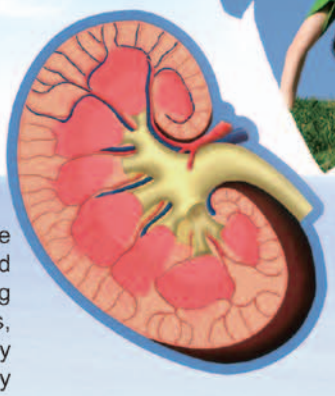
Certificate Course on

Paediatric Nephrology 2011

Jointly organised by

The Federation of Medical Societies of Hong Kong

Hong Kong Paediatric Nephrology Society



Objectives:

The course is designed for general practitioners, health care providers and general public who are interested and involved in caring of children. It contains a series of 6 lectures ranging from common childhood problems like nocturnal enuresis, urinary tract infection, hypertension to more specific kidney disease like nephritis, nephrotic syndrome, hereditary kidney disease and renal failure. Participants can update the knowledge in the respective field and facilitate the provision of care to this group of children.

Date	Topics	Speakers
6 April 2011	Children with Enuresis – What do parents need to know? 夜遺尿知多少？	Dr. Stella CHIM 詹愷怡醫生 Associate Consultant Queen Mary Hospital
13 April 2011	Hypertension in Children – Early Detection and Prevention 兒童高血壓的探討與預防	Dr. Lettie C.K. LEUNG 梁竹筠醫生 Consultant Paediatrician Kwong Wah Hospital
20 April 2011	Urinary Tract Infection in Children - Diagnosis and Treatment 尿道感染 - 診斷與治療	Dr. Kwok-piu LEE 李國彪醫生 Senior Medical Officer Alice Ho Miu Ling Nethersole Hospital
27 April 2011	Hereditary Kidney Disease in Childhood 遺傳性的兒童腎病	Dr. Kwok-wai LEE 李國偉醫生 Specialist in Paediatrics Queen Elizabeth Hospital
4 May 2011	From Urinary Abnormalities to Nephritic and Nephrotic Syndrome 小兒腎病及腎炎綜合症	Dr. Wai-ming LAI 賴偉明醫生 Consultant Paediatrician Princess Margaret Hospital
11 May 2011	Prevention of Renal Failure 預防腎衰竭 – 何去何從?!	Dr. Niko Kei-chiu TSE 謝紀超醫生 Consultant Paediatrician Department of Paediatrics & Adolescent Medicine Princess Margaret Hospital & Yan Chai Hospital

Time : 7:00 p.m. – 8:30 p.m.
Venue : Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Language Media : Cantonese (Supplemented with English)
Course Fee : HK$750 (6 sessions)
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 / CPD Accreditation in application
A total of **9 CNE** points for the whole course and the points will be awarded according to the number of hours attended.
Application form can be downloaded from website: http://www.fmshk.org