9th Advanced Neuroradiology Course

Singapore

28 – 29 Oktober 2009

Dr. Win Mar @ Salmah Jalaludin
Jabatan Radiologi
Pusat Pengajian Sains Perubatan

9th Advanced Neuroradiology Course
28 - 29 October 2009

Introduction

Day One
28 October 2009

Day Two
29 October 2009

Acknowledgements

This event is accredited by Singapore Medical Council for 8 CME points

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FOREWORD MESSAGE

Dear Colleagues and Friends,

On behalf of the organizing committee, I welcome you to the 9th Advanced Neuroradiology Course organized by the National Neuroscience Institute, Singapore.

This year's theme is "Vascular Pathology – State of the Art Imaging and Intervention". Besides structured lectures on topics related to imaging and intervention of neurovascular diseases, the Course will have a special focus symposia on Head and Neck imaging.

We have specially invited a distinguished panel of faculty speakers from overseas and locally, who will share their expertise and insights on developments in their respective fields.

I hope the meeting will bring about greater interactions among the different neuro-related subspeciality fields and be beneficial to the way we manage our patients. And for those who has traveled from beyond the shores, I extent a warm welcome for your stay in this beautiful island of Singapore!

9th Advanced Neuroradiology Course
28 - 29 October 2009

COURSE FACULTY

Professor Marius Hartmann
University of Heidelberg, Germany

Dr Mathieu Rodallec
Centre Cardiologique Du Nord, France

Dr Seung-Koo Lee
Yonsei University College of Medicine, Korea

Dr Francis Hui
National Neuroscience Institute

Dr Sitoh Yih Yian
National Neuroscience Institute

Assoc Prof Tchoyoson Lim
National Neuroscience Institute

Dr Lishya Liauw
National Neuroscience Institute

Dr Wickly Lee
National Neuroscience Institute

Dr Tan Tiong Yong
Changi General Hospital

Dr Winston Lim
Singapore General Hospital

Dr Chan Ling Ling
Singapore General Hospital

Dr Judy Tan
Singapore General Hospital Hospital

Dr Julian Goh
Tan Tock Seng Hospital

Dr Loke Siu Cheng
Tan Tock Seng Hospital

Dr Samuel Ng
Mount Elizabeth Hospital

ORGANISING COMMITTEE

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Dr Wickly Lee

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Mr Tien Sin Leong

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Ms Haslindah Salim
Mr Wee Liang Kwee
Mr Hong Tshun Vun
Mr James Tan
Ms Sim Hwee Peng

9th Advanced Neuroradiology Course
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Day One
Wednesday, 28 October 2009

8.00 am **Registration**

8.45 am **Welcome Address & Opening Ceremony**
Course Contents & Introduction

SCIENTIFIC SESSION 1

Chairperson: *Dr Winston Lim*

9.00 am **Endovascular Treatment of ICAD**
by Professor Marius Hartmann

9.35 am **Vascular Lesions in the Head & Neck**
- Diagnosis & Interventions
by Dr Winston Lim

10.10 am **From Acute Stroke Imaging to Intervention**
- Putting it All Together
by Dr Wickly Lee

10.45 am *Tea Break* **(Sponsored by Johnson & Johnson)**

Chairperson: *Dr Samuel Ng*

11.15 am **Angiography (CT & MR) of the Extracranial Circulation**
by Dr Mathieu Rodallec

11.50 am **The Role of CT Perfusion (CTP) in Stroke Imaging**
by Dr Samuel Ng

12.25 pm **Endovascular Treatment of Carotid Stenosis**
by Professor Marius Hartmann

1.00 pm *Lunch* **(Sponsored by Transmedic Pte Ltd)**

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Day One
Wednesday, 28 October 2009

SCIENTIFIC SESSION 2

Chairperson: *Dr Julian Goh*

2.00 pm **Anatomy of Temporal Bone**
by Dr Loke Siu Cheng

2.35 pm **Sensorineural Hearing Loss**
by Dr Tan Tiong Yong

3.10 pm ***Tea Break*** **(Sponsored by O'Connor's)**

Chairperson: *Dr Julian Goh*

3.45 pm **Imaging in Adult Conductive Hearing Loss**
by Dr Julian Goh

4.20 pm **Highways of the Head and Neck - Patterns of Disease Spread**
by Dr Judy Tan

4.55 pm **End of Day 1**

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Wednesday, 28 October 2009

ABSTRACTS

Endovascular Treatment of ICAD
Professor Marius Hartmann

Vascular Lesions of the Head & Neck
- Diagnosis and Interventions
Dr Winston Lim

From Acute Stroke Imaging to Intervention
- Putting It All Together
Dr Wickly Lee

Angiography (CT and MR) of the Extracranial Circulation
Dr Mathieu Rodallec

The Role of CT Perfusion (CTP) in Stroke Imaging
Dr Samuel Ng

Endovascular Treatment of Carotid Stenosis
Professor Marius Hartmann

Anatomy of Temporal Bone
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Sensorineural Hearing Loss
Dr Tan Tiong Yong

Imaging in Adult Conductive Hearing Loss
Dr Julian Goh

Highways of the Head and Neck
- Patterns of Disease Spread
Dr Judy Tan

Endovascular Treatment of ICAD

Professor Marius Hartmann
Head of the Division of Interventional Neuroradiology, Dept of Neurology
University of Heidelberg Medical School, Germany

Stroke and cerebrovascular diseases are the third leading cause of death and a leading cause of adult disability. The majority of stroke cases are due to ischemic causes. Depending on the population studied, intracranial atherosclerosis accounts for 10–29% of brain ischemic events. Currently, the primary treatment in intracranial atherosclerosis is the control of vascular risk factors such as hypertension, diabetes, hypercholesterolemia, and smoking. Unfortunately, a significant number of patients with intracranial atherosclerosis continue to suffer from repeated strokes or transient ischemic attacks despite optimized medical treatment. Technological advances over the past 10 years have enabled endovascular treatment of intracranial atherosclerotic disease (ICAD). The number of patients treated with angioplasty or stent-assisted angioplasty for this condition is increasing. There are only 2 prospective feasibility and safety trials using endovascular treatment of stenotic intracranial atherosclerosis (SSYLVIA- and WINGSPAN-trial). Up to now there are no randomised controlled trials for the treatment of ICAD. Based on this experience, the Society of Neurointerventional Surgery, Society of Interventional Radiology, and American Society of Neuroradiology recommended in 2005 angioplasty with or without stenting of a symptomatic patient with an intracranial stenosis of > 50% who failed medical treatment. Balloon angioplasty without or with stent has been tempered by concerns regarding the potential for intimal dissection, perforator and vessel occlusion, thrombus formation, parent vessel perforation, and vessel rupture. The advent of improved stent designs (e.g. self-expanding stents, WINGSPAN™) resulted in an increased technical success rate of more than 95%, even in distal lesions. Anyhow, cerebrovascular complications including stroke or death within 30 days of stenting have been reported in up to 32% of these patients.

Resent data suggest that adverse events after intracranial angioplasty and stenting may be associated with low volume sites, stenting soon after a qualifying event, and stroke as the qualifying event. Patient selection, careful periprocedural medical management, and a highly skilled interventionalists are all required in order to perform the procedure with an acceptable risk.

Restenosis after angioplasty and stent-assisted angioplasty is still a matter of debate. In the prospective intracranial stenting trials, restenosis rates range between 7.5% and 32.4%. Supraclinoid lesions seems to have much higher rates of both re-stenosis and symptomatic re-stenosis in comparison with all other locations.

Overall, the current data are weak and inconsistent. Therefore, these factors will need to be monitored in future prospective registries ore randomized trials comparing optimized medical treatment against intracranial angioplasty and stenting.

1. *SSYLVIA Investigators. Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA): study results. Stroke. 2004;35:1388 – 1392.*
2. *Bose A, Hartmann M, Henkes H, et al. A novel, self-expanding, nitinol stent in medically refractory intracranial atherosclerotic stenoses. The Wing Span Study. Stroke. 2007;38:1531–1537.*
3. *Nahab F.Lynn MJ, Kasner SE, et al. Risk factors associated with major cerebrovascular complications after intracranial stenting. Neurology® 2009;72:2014–2019.*
4. *Turk AS, Levy EI, Albuquerque FC, et al. Influence of patient age and stenosis location on Wingspan in-stent restenosis. Am J Neuroradiol. 2008;29:23–27.*

Vascular Lesions of the Head and Neck - Diagnosis and Interventions

Dr Winston Lim
Senior Consultant
Department of Diagnostic Radiology
Singapore General Hospital

This short overview will cover:

1. Review of Vascular anatomy pertinent to diagnosis and interventional procedures in the Head and Neck region, with specific emphasis on dangerous anastomoses between the the internal and external carotid vessels as well as the vertebro-basilar system.

2. Review of Vascular tumours of the Head and Neck region and intervention strategies for management of these lesions.

3. Review of the classification of Vascular anomalies of the Head and Neck region into LOW FLOW and HIGH FLOW entities and how management can be targeted to treat these lesions.

From Acute Stroke Imaging to Intervention – Putting It All Together

Dr Wickly Lee
Consultant Neuroradiologist
Department of Neuroradiology
National Neuroscience Institute
Singapore

The development of effective therapies for acute ischemic stroke presumes the existence of potentially salvageable ischemic tissue when therapy is initiated. The role of neuroimaging in the initial assessment of stroke patients has been well established. With the introduction of endovascular methods for the treatment of acute ischemic stroke, the fast triage and identification of patients with large vessel ischemic stroke now assumes a key aspect of the overall effort in acute stroke salvage. It is widely assumed that the effectiveness of most acute stroke therapies is related to reducing the ultimate infarct size to promote functional improvement. Such salvageable ischemic tissue labeled as the 'ischemic penumbra' must be distinguished from irreversible injury. Patient selection for endovascular therapy using clinical and neuroimaging criteria will be discussed. The experience and results of endovascular therapy for the treatment of patients with large vessel stroke will be presented.

Angiography (CT and MR) of the Extracranial Circulation

Dr Mathieu Rodallec
Department of Radiology
CENTRE CARDIOLOGIQUE DU NORD
France

Magnetic resonance (MR) angiography and computed tomography (CT) are robust imaging techniques for evaluation of the extracranial circulation.

CT angiography typically involves a volumetric helical acquisition that extends from the aortic arch to the circle of Willis. The examination is performed by using a time-optimized bolus of contrast material for vessel enhancement. Postprocessing is performed at a three-dimensional display workstation to generate multiplanar reformatted images and maximum intensity projection images.

Contrast-enhanced magnetic resonance angiography (CE-MRA) has also become a well-established noninvasive imaging method for the assessment of the extracranial circulation. However, CE-MRA is not a standardized technique, but encompasses different concurrent techniques. Without the necessity for additional bolus timing, time-resolved 3D contrast-enhanced MR-angiography reveals excellent diagnostic image quality in neurovascular imaging. In addition, the dynamics of time-resolved-CE-MRA can offer additional information on vascular pathologies.

The radiologist must be aware of the advantages and limitations of the different techniques available.

The Role of CT Perfusion (CTP) in Stroke Imaging

Dr Samuel Ng
Consultant
Medi-Rad Associates
Mount Elizabeth Hospital

The advent of ultrafast CT scanning with multi-array detectors allows for entire brain coverage in a single revolution, acquiring 320 slices of 0.5mm thickness in 0.75sec. Given this capability, intermittent scanning can detail the first pass effect of contrast though the entire brain over 19 time points, spread over a 1 minute span. This is in addition to providing pre and post contrast enhanced conventional images as well as a CT angiogram of the Circle of Willis.

The 6080 images are then processed to yield four typical perfusion maps – reflecting Cerebral Blood Flow (CBF), Cerebral Blood Volume (CBV), Time To Peak (TTP) and Mean Transit Time (MTT). Some vendors provide another measure of collateralization, the Delay map.

The ischaemic penumbra can be defined by the CBF/CBV mismatch, the equivalent to the Perfusion-Diffusion mismatch of MRI. Comparison will be made of this penumbra, generated via MRI and CT scan.

The above capability of CT allows the radiologist to answer the following in a stroke patient

1. any haemorrhage
2. any large vessel occlusion
3. Define tissue under risk of infarct
4. Define which of (3) is salvageable

Important criteria for favourable neuro-interventional outcome include:

- Plain CT entirely normal
- CBF def >> CBV def
- Overall CBV def < 1/3rd size of MCA
- CBV of 2.0 ml/100g or less
- MTT > 145%
- CBF defect > 2 cm
- CBF-CBV mismatch > 20%

Endovascular Treatment of Carotid Stenosis

Professor Marius Hartmann
Head of the Division of Interventional Neuroradiology, Dept of Neurology
University of Heidelberg Medical School, Germany

Stenting of carotid artery stenosis (CAS) continues to remain a controversial procedure. Early outcome data from two large, European, randomised, multicentre trials were published in 2006, the SPACE and the EVA-3S trial. The EVA-3S trial was stopped early, owing to a significantly lower rate of stroke or death in the patients who were treated surgically. Among symptomatic patients with high-grade stenosis of the carotid artery, SPACE failed to prove noninferiority of CAS for lack of power from 1214 patients recruited. However, in SPACE there was no statistical difference in the rate of outcome events between carotid endarterectomy (CEA) and CAS. For secondary stroke prevention after 2 years' follow-up, there were no significant differences in the rate of recurrent ipsilateral ischemic strokes. The incidence of recurrent carotid stenosis at 2 years, as defined by ultrasound, was significantly higher after carotid artery stenting. However, it cannot be excluded that the degree of in-stent stenosis is slightly overestimated by conventional ultrasound criteria.

The use of protection devices and stents with different cell designs in CAS is a subject of controversy. SPACE was notable because many of the German neuroradiologists did not use protection devices, but in the trial there was no significant difference in procedural stroke rates in those stented with and without a protection device. Additionally, the results of a secondary analysis from the SPACE trial demonstrate that the choice of the stent design (open vs. closed cell) has a direct impact on the periprocedural complication rate in CAS. Furthermore, in younger patients (< 68 years of age) the risk of a primary outcome event was significantly lower after CAS than after CEA.

Moreover, every operative therapy is profoundly dependent on the manual ability and the experience of the local operators, the used materials and their preparation. There is large experience confirming significantly lower mortality and stroke rates with higher operator experience and at hospitals providing a higher annual hospital volume.

There are no prospective randomized data available concerning the treatment of asymptomatic carotid stenosis with CAS. Therefore, the objective of the SPACE 2 trial a randomised multicentre study is to compare up-to-date medical treatment with CAS and CEA in addition to conservative treatments in individuals with asymptomatic atherosclerotic carotid artery stenoses.

Anatomy of Temporal Bone

Dr Loke Siu Cheng
Associate Consultant
Department of Diagnostic Radiology
Tan Tock Seng Hospital, Singapore

Temporal bone imaging is integral to current clinical practice in evaluation of causes of hearing loss.

This talk aims to give an overview of the anatomy of the temporal bone with regards to diagnostic imaging, in particular the structures concerned with hearing.

The developmental origin of the ear and its structures will be briefly described.

With the aid of relevant images and diagrams, structures in the outer, middle and inner ear are reviewed, with relations of the structures to each other.

Michael Aplasia – congenital.

Chochlear Aplasia – vestibule is (+)
5th wk gestation develop-arrest.

Cystic chochlea – arrest develop 5th Gest. wk.

Mondini deform – arrest later in developt
at 7th gest. week.

Large vestibular aqueduct syn.

Labyrinthine Schwannoma

Bilat labyrinthitis –
Autoimmune "
Suppurative " → Labyrinthitis ossificans.

Spont. Labyrinthine Hge.

Retrofenestral otosclerosis

Sensorineural Hearing Loss

Dr Tan Tiong Yong
Chief of Radiology
Changi General Hospital, Singapore

The auditory system comprises the external ear, the middle ear, the inner ear, the cochlear nerve and the central acoustic pathway. The external ear collects and directs the sound, the middle ear amplifies and transmits the sound as mechanical energy, and the inner ear converts the mechanical energy into electrical energy. This is then transmitted via the cochlear nerve through the central acoustic pathway, which comprises a series of fibres and nuclei, to the central processing area in the superior temporal gyrus. Sensorineural hearing loss (SNHL) results, when a lesion involves the cochlea, the cochlear nerve or the component of the central acoustic pathway.

Lesions affecting the cochlea can be divided into broad groups, including congenital anomalies, trauma, tumours, infection, inflammation and other miscellaneous causes like otodystrophies. Michael aplasia is a rare congenital anomaly whereby there is total absence of any labyrinthine development. This usually results from an insult that resulted in arrest in labyrinthine development at about 3 weeks' gestation. Other congenital anomalies include cochlear aplasia, cochlear hypoplasia, incomplete partition type of anomaly; all the result of arrest of development due to insults occurring at different gestational age between the 4^{th} and 7^{th} week. Labyrinthine schwannoma is an uncommon tumour that affects the cochlea and/or the vestibule that leads to hearing loss. This is demonstrated on MRI as an intralabyrinthine enhancing mass. Suppurative labyrinthitis that results from meningitis can lead to labyrinthitis ossificans, where the labyrinths are replaced by bone. MRI would demonstrate the loss of fluid signal in the labyrinth and CT scan would demonstrate the bony replacement. Sensorineural hearing loss that results from fractures involving the labyrinth is uncommon and

usually complete and permanent. Fractures are well delineated by CT scan and pneumolabyrinth can sometimes be demonstrated.

Sensorineural hearing loss from lesions affecting the cochlear nerve is most commonly due to inflammation/infection and tumours. In most cases of cochlear nerve neuritis, the MRI is normal. Uncommonly, post-contrast scan would demonstrate the linear enhancement of the cochlear nerve. The most common tumour of the cerebellopontine angle and internal auditory canal is the acoustic schwannoma. This more commonly arises from the inferior or superior vestibular nerves than from the cochlear nerve. In these cases, the sensorineural hearing loss result from compression of the cochlear nerve by the vestibular nerve schwannoma. Meningioma is the next most common tumour of the cerebellopontine angle that gives rise to sensorineural hearing loss. Wallerian degeneration of the cochlear nerve can result from long standing cochlear lesion like labyrinthitis ossificans.

Lesions of the central acoustic pathway causing sensorineural hearing loss are uncommon. These include infarct/haemorrhage involving the cochlear nerve nuclei, demyelinating disease like multiple sclerosis and tumours. Usually, when the sensorineural hearing loss is due to a lesion in the central acoustic pathway, there would also be other cranial nerves involved, usually the 5^{th}, 6^{th}, 7^{th} cranial nerves.

Imaging in Adult Conductive Hearing Loss

Dr Julian Goh
Consultant
Department of Diagnostic Radiology
Tan Tock Seng Hospital, Singapore

Conductive hearing loss in adults occurs when there is an abnormality in the conductive pathway responsible for the transmission of sound from the environment to the inner ear. There is a plethora of causes, encompassing the external auditory canal and the middle ear. Acquired conductive hearing loss is by far more common; causes include chronic otitis media, cholesteatomas and trauma. In younger patients with no known antecedent history of trauma or chronic inflammation conditions of the ear, congenital causes should be considered. In this lecture the more common causes of acquired conductive hearing loss will be discussed. Congenital causes will also be discussed.

Highways of the Head and Neck – Patterns of Disease Spread

Dr Judy Tan
Consultant
Department of Diagnostic Radiology
Singapore General Hospital

Anatomy of the head and neck region can be daunting for the radiologist as it is a compact area and yet numerous important small vessels and nerves are seen in that region. The aim of my talk is to discuss relevant anatomy that is important in the understanding of disease spread. Cases will be illustrated to emphasize important anatomic structures and routes of disease spread. At the end of the talk, it is hoped that the participant can identify perineural spread along key nerves.

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Day Two
Thursday, 29 October 2009

8.00 am **Registration**

SCIENTIFIC SESSION 3

Chairperson: *Dr Lishya Liauw*

9.00 am **Quantification of Brain Disease - T1 and T2 Relaxometry Studies**
by Dr Seung-Koo Lee

9.35 am **Advances in and Updates on Neuroimaging in Systemic Lupus Erythematosus**
by Dr Chan Ling Ling

10.10 am *Tea Break* **(Sponsored by Malex Medical Asia / EV3)**

Chairperson: *Assoc Prof Tchoyoson Lim*

10.45 am **Cerebral Venous Thrombosis**
by Dr Mathieu Rodallec

11.20 am **Blood Brain Barrier: What We Know & How We Can See**
by Dr Seung-Koo Lee

11.55 am **Stroke Mimics on DWI**
by Assoc Prof Tchoyoson Lim

12.30 pm *Lunch* **(Sponsored by Transmedic Pte Ltd)**

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SCIENTIFIC SESSION 4

Chairperson: *Dr Francis Hui*

1.30 pm **Demonstration of Interventional Neuroradiology Procedure and Case Discussions**
by Professor Marius Hartmann

2.05 pm **Intra-arterial Therapy of Acute Intracranial Vessel Occlusion**
by Professor Marius Hartmann

2.40 pm **Mechanical Thrombectomy in Acute Ischaemic Stroke**
by Dr Francis Hui

3.15 pm ***Tea Break*** **(Sponsored by United BMEC Pte Ltd)**

Chairperson: *Dr Sitoh Yih Yian*

3.45 pm **Imaging in Vascular Dementia and Cognitive Disorders**
by Dr Sitoh Yih Yian

4.20 pm **Vasculitis of the Paediatrics CNS**
by Dr Lishya Liauw

4.55 pm **Closing Remarks**
by Dr Wickly Lee

Other ↑ SI

- Hipp fissure remnant.
- Choroid plexus cyst
- T/m

T_2* Relaxo.

- Brain Iron content - in Basal G.
- moyamoya — numerous, fine collat around B. ganglia
  - ↑ micro-vessel.
  - ↑ r CBV - d/t compensatory VD^n.

T* Relax for vessel + neovascularizn ⇒ ↓ Time

- Local Iron concentration
- " microvas. (A/Vein)
- possible indicat^n for neovascular

T_1 Relaxo — limited for ~~refs~~ msk, liver

- Assess in Parkinson d/s.

Neuromelanin Imaging

thin T1W1.