SUPPLEMENT 4

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7th Hong Kong Neurological Congress cum 34th Annual Scientific Meeting of The Hong Kong Neurological Society

6 – 7 November 2021

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Scientific Programme

Full Virtual Webinar (6-7 November 2021)

6 November 2021, Saturday

09:15	Platform ready for log-in
09:15 – 09:30	Break / air time
09:30 – 10:10	**Movement Disorder Symposium 1** Chairpersons/Judges: *Germaine Chan, Shirley Pang* **Common movement disorders pitfalls** *Alberto J Espay (USA)*
10:10 – 10:15	Break / air time
10:15 – 11:00	**Epilepsy Symposium 1** Chairpersons/Judges: *TL Poon, Colin Lui* **Introductory sharing of local data on deep brain stimulation therapy** *TL Poon (Hong Kong)* **Neuro-modulation therapy in epilepsy** *Robert S Fisher (USA)*
11:00 – 11:10	Break / air time
11:10 – 11:50	**UCB Epilepsy Symposium 2** Chairperson/Judge: *Gardian Fong* **Challenges and solutions about management of uncontrolled primary generalised tonic-clonic seizures** *Terence O'Brien (Australia)*
11:50 – 12:00	Break / air time
12:00 – 12:40	**Inter-disciplinary Neurology Symposium** Chairperson/Judge: *Nelson Cheung* **Cobalamin and homocysteine** *Bun Sheng (Hong Kong)*
12:40 – 12:45	Break / air time
12:45 – 12:55	**Opening Ceremony** (Guest of Honour: *Prof Sophia Chan, JP, Secretary for Food and Health, Food and Health Bureau, The Hong Kong SAR Government*)
12:55 – 13:00	Break / air time
13:00 – 13:40	**Free Paper Presentation** Chairpersons/Judges: *WK Cheng, Nelson Cheung* **Low-density lipoprotein cholesterol and risk of recurrent vascular events in Chinese patients with ischaemic stroke with or without significant atherosclerosis** *Bryan J Chua (Hong Kong)* **Oedematous woman with demyelinating polyneuropathy: a missing letter in the acronym of POEMS** *YN Mew (Hong Kong)* **Case series of everolimus use in adult tuberous sclerosis complex related renal angiomyolipoma and refractory seizures** *YK Chan (Hong Kong)* **Is oxygen desaturation the link between sleep apnoea and cerebral small vessel disease burden in patients with stroke?** *Xiaodi Liu (Hong Kong)* **Direct oral anticoagulant and antiepileptic drug combination on risk of thromboembolism and major bleeding: a territory-wide review** *BYM Ip (Hong Kong)*

6 November 2021, Saturday

13:00 – 13:40	**Poster Presentation (in a separate Zoom Meeting Room)** Chairpersons/Judges: *Colin Lui, Winnie Wong* **Efficacy of erenumab for migraine in a private hospital in Hong Kong** *KK Lau (Hong Kong)* **Not too little and not too much: a case report of SCN4A-related congenital myopathy with two novel mutation variants** *YLT Lam (Hong Kong)* **Predictors of poor outcome in intraarterial thrombectomy** *MS Chi (Hong Kong)* **Unconscious and weak patient with COVID-19** *CH Cheung (Hong Kong)*
13:40 – 13:45	Break / air time
13:45 – 14:25	**BI Stroke Symposium 1** Chairpersons/Judges: *Gary Lau, Herrick Lau* **Time is cure: acute management for patients with atrial fibrillation on anticoagulants** *Alan Barber (New Zealand)*
14:25 – 14:30	Break / air time
14:30 – 15:10	**Daiichi Sankyo Stroke Symposium** Chairpersons/Judges: *Gary Lau, Herrick Lau* **Reduced dosage of novel oral anticoagulants for stroke prevention in frail patients with atrial fibrillation** *Lawrence Wong (Hong Kong)*
15:10 – 15:15	Break / air time
15:15 – 16:05	**Symposium on Autoimmune Encephalitis** Chairpersons/Judges: *Carlin Chang, Richard Li* **Updates on autoimmune encephalitis** *Josep Dalmau (Spain)*
16:05 – 16:45	**Movement Disorder Symposium 2** Chairpersons/Judges: *Helen Yip, Karen Ma* **Autoimmune movement disorders** *Bettina Balint (Germany)*

7 November 2021, Sunday

08:45	Platform ready for log-in
08:45 – 09:00	Break / air time
09:00 – 09:40	**Stroke Symposium 2** Chairperson/Judge: *Yannie Soo* **Insights and updates on stroke rehabilitation** *John Krakauer (USA)*
09:40 – 09:50	Break / air time
09:50 – 10:30	**Dementia Symposium 1** Chairpersons/Judges: *Bun Sheng, Betty Ng* **Mild behavioural impairment as a proxy marker for Alzheimer disease: what do biomarkers tell us?** *Zahinoor Ismail (Canada)*
10:30 – 10:40	Break / air time
10:40 – 11:15	**Dementia Symposium 2** Chairpersons/Judges: *Bun Sheng, Betty Ng* **Management of behavioural and psychological symptoms of dementia: a psychiatrist viewpoint** *John Chan (Hong Kong)*
11:15 – 11:20	Break / air time
11:20 – 13:15	**Dissertation Highlights** Chairpersons/Judges: *Richard Li, Yannie Soo* **Applicability of haemorrhagic transformation predicting tools in predicting post–tissue plasminogen activator symptomatic intracranial haemorrhage in local patients** *YW Wong (Hong Kong)* **Risk factors for key outcomes of motor neurone disease: a single-centre study** *HY Se (Hong Kong)* **Retrospective review of clinical characteristics and effect of integrated clinic on patients with motor neuron disease in regional hospitals in Hong Kong** *LH Siu (Hong Kong)* **Prevalence and clinical predictors of aspiration pneumonia in patients with Parkinson disease in Hong Kong** *YN Mew (Hong Kong)* **10-year review of inflammatory myopathy in a regional hospital in Hong Kong** *SN Cheung (Hong Kong)* **Autonomic dysfunction in Chinese patients with idiopathic Parkinson disease in a public hospital in Hong Kong** *YK Leung (Hong Kong)* **Case series of familial amyloid polyneuropathy in Hong Kong** *LP Ng (Hong Kong)* **Pilot study on the use of a smartphone application in assessing motor and gait disability in patients with Parkinson disease** *KK Yam (Hong Kong)* **Factors associated with generalisation of ocular myasthenia gravis and myasthenic crisis** *CK Yuen (Hong Kong)* **Clinical predictors and outcome of late-onset post-stroke seizure: a 10-year retrospective observational study** *ST Lui (Hong Kong)* **Impulse control and related behavioural disorders in patients with idiopathic Parkinson disease treated with different dopamine agonists** *HF Wu (Hong Kong)*

7 November 2021, Sunday

13:15 – 13:20	Break / air time
13:20 – 14:00	**Biogen SMA Symposium** Chairperson/Judge: *Betty Ng, Shirley Cheung* **Nusinersen in adults with spinal muscular atrophy: how to define treatment response?** *Elena Pegoraro (Italy)*
14:00 – 14:10	Break / air time (10 min)
14:10 – 14:50	**Novartis Migraine Symposium** Chairpersons/Judges: *Carlin Chang, Yannie Soo* **New-generation drugs for migraine prevention: anti–calcitonin gene-related peptide versus conventional preventives** *Uwe Reuter (Germany)*
14:50 – 15:00	Break / air time
15:00 – 15:40	**Novartis Multiple Sclerosis Symposium 1** Chairpersons/Judges: *Stephen Cheng, Jessica Li* **B-cell therapy: new era in management of multiple sclerosis** *Ludwig Kappos (Switzerland)*
15:40 – 15:45	Break / air time
15:45 – 16:40	**Merck Multiple Sclerosis Symposium 2** Chairpersons/Judges: *Jacky Lee, Ka Lock Shiu* **Update in magnetic resonance imaging in multiple sclerosis guidelines** *Frederik Barkhof (UK)*
16:40 – 16:45	Closing Remarks

Common movement disorders pitfalls

S 1

Alberto J Espay
James J and Joan A Gardner Center for Parkinson's disease and Movement Disorders, University of Cincinnati Academic Health Center, Department of Neurology and Rehabilitation Medicine, Cincinnati, OH, USA

Through a case-based approach, this session illustrates common errors of nosological classification or semiological interpretation during the evaluation of patients with movement disorders. These pitfalls have been selected for their important therapeutic implications.

Introductory sharing of local data on deep brain stimulation therapy

S 2

TL Poon
Queen Elizabeth Hospital, Hong Kong

Neuromodulative surgery for treatment of drug-refractory epilepsy is increasingly popular. It is indicated for non-resectable cases or failed previous resection cases. In Hong Kong, vagus nerve stimulation has been practised for more than 10 years. Deep brain stimulation was introduced in 1997 for treatment of movement disorders, and its indication has extended to drug-refractory epilepsy after the Simulation of the Anterior Nucleus of the Thalamus (SANTE) trial. The first deep brain stimulation for epilepsy in Hong Kong was performed in 2015. Totally nine patients in two hospitals have been treated. Anterior nucleus of thalamus was the stimulation target. This presentation briefly reviews these cases and the treatment outcome.

Neuromodulation for epilepsy

S 3

Robert S Fisher
Stanford Epilepsy Center and EEG lab, Stanford University, USA

In the US, there are three varieties of approved neuromodulation (also called neurostimulation): vagus nerve stimulation (VNS), responsive neurostimulation (RNS), and deep brain stimulation of the anterior thalamus (DBS). This talk will compare the efficacy and major adverse effects of each modality and provide some preliminary guidance on choice. In controlled trials, VNS reduced seizures by about one-third during the blinded phase and about one-half over 2 years. A systematic review showed 8% became seizure-free. Addition of VNS stimulation at times of seizure-induced tachycardia possibly increases efficacy. Most VNS adverse effects relate to the throat or voice. RNS reduced seizures by a median of 38% in the blinded phase and two-thirds over 6 years. About 20% became seizure-free. Adverse effects of RNS were infrequent and mainly related to the surgery. DBS reduced seizures by a median of 40% in the 3-month blinded phase and 70% over 7 years of open-label stimulation; 18% became seizure-free. Main adverse effects of anterior thalamic DBS included those related to acute surgery, local paresthesias, and transient worsening of depression or memory. Sudden unexplained death in epilepsy was reduced by all three methods. Choice of neurostimulation remains individualised. VNS is less invasive but less effective. It may be beneficial for comorbid depression. RNS and DBS have similar efficacy. RNS provides excellent chronic recording but requires knowledge of the seizure-onset location. DBS does not require knowledge of seizure location and is easier to manage but may cause transient depression or memory problems. Efficacy of DBS for posterior foci is uncertain. Neurostimulation provides a useful new therapy for medically and surgically refractory epilepsy.

Challenges and solutions about management of uncontrolled primary generalised tonic-clonic seizures

S 4

Terence O'Brien
Departments of Neuroscience, Central Clinical School, Department of Neurology, Alfred Health, Monash University, Melbourne, Victoria, Australia

Tonic-clonic seizures (TCS) with idiopathic generalised epilepsy (IGE), also known as primary generalised tonic-clonic seizures (PGTCS), are one of the most common and disabling seizure types in adults and teenagers with epilepsy. Many patients with PGTCS can achieve seizure control with currently available anti-seizure medications. However, in an estimated 15% to 36% of patients, the TGTCS are uncontrolled. Uncontrolled PGTCS are associated with an increased risk of injury and death. Epidemiological studies have shown that the risk of sudden unexpected death in epilepsy dramatically increases with the increasing numbers of TCS that occur per year, particularly nocturnal. Although >15 new anti-seizure medications have been introduced into clinical practice over the past 2 to 3 decades, only a limited number of these have been approved or demonstrated to be efficacious for PGTCS. In clinical assessment of patients with TCS, it is critical to differentiate between PGTCS and focal to bilateral TCS, as the pharmacoresponse profile, prognosis, aetiology, and treatment options differ between these two types of TCS. Evidence from the SANAD open-label randomised controlled trials indicate that valproate is the most effective anti-seizure medication for patients with PGTCS. However, valproate is not recommended as a first-line anti-seizure medication for women with child-bearing potential, because of the risk to unborn child. This risk needs to be balanced with the risk of uncontrolled seizures to the mother. For patients with drug-resistant PGTCS, clinical trials pose greater methodological and ethical challenges than trials for drug-resistant focal seizures. There is evidence from double-blind randomised controlled trials for efficacy of lamotrigine, levetiracetam, topiramate, and perampanel. The recently published VALOR double-blind randomised controlled trial of lacosamide versus placebo as adjunctive treatment in patients with uncontrolled PGTCS used a novel trial design and the time to occurrence of a second PGTCS (rather than a fixed treatment period). This design has methodical and ethical advantages and demonstrated that lacosamide was efficacious and well tolerated in patients with uncontrolled PGTCS. Other emerging adjunct treatment options for patients with drug-resistant PGTCS include neurostimulation and dietary therapies.

Cobalamin and homocysteine

S 5

Bun Sheng
Department of Medicine & Geriatrics, Princess Margaret Hospital, Hong Kong

Nature does not divide human diseases into organ systems according to medical textbook classifications. The rapid advancement of knowledge and technology enables 'subspecialisation' in an ever-narrowing field of interest under modern neurology, which inevitably distances us from the fundamentals of human life. In this lecture, we approach an important neurology issue from a different dimension by looking at medical problems not traditionally related to neurology. We shall see this extended knowledge in improving our understanding of the issue.

Cobalamin is a cofactor of methylmalonyl-CoA mutase and methionine synthase. Cobalamin deficiency suppresses these two enzymatic activities and results in methylmalonic acidaemia and hyperhomocystinaemia, respectively. Subacute combined degeneration of spinal cord and megaloblastic anaemia are the two most common diseases secondary to cobalamin deficiency, whereas hyperhomocystinaemia is a strong risk factor for thrombosis. We review the cobalamin metabolic pathways, the clinical effects of substrate accumulation and end-product depletion, and the pathophysiology of cobalamin deficiency through hereditary diseases of cobalamin-homocysteine metabolic defects. We also discuss the myth of methylenetetrahydrofolate reductase polymorphism in human disease and provide a critical appraisal of different homocysteine-reduction strategies.

Time is cure: acute management for patients with atrial fibrillation on anticoagulants

S 6

Alan Barber
Department of Medicine, University of Auckland, New Zealand

Novel oral anticoagulants are well proven for their stroke prevention efficacy and safety in patients with atrial fibrillation. However, in emergency conditions such as acute ischaemic stroke and intracerebral haemorrhage, the management could be challenging. The use of a specific reversal agent for novel oral anticoagulants may enable timely intervention. Prof Alan Barber will share clinical experience and review the latest real-world evidence and guidelines on acute stroke management in those with atrial fibrillation.

S 7

Reduced dosage of novel oral anticoagulants for stroke prevention in frail patients with atrial fibrillation

Lawrence Wong
Private Practice

Non–vitamin K antagonist oral anticoagulants (NOACs) are the oral anticoagulants of choice for stroke prevention in patients with atrial fibrillation. For patients with high bleeding risk, a reduced dose is offered to mitigate bleeding. However, the dose reduction criteria vary in different NOACs, and the amount and type of data supporting the dosing strategies differ. In fear of bleeding, reduced- or low-dose NOAC is often prescribed for frail patients regardless of the recommended reduction, resulting in off-label under-dosage. Such practice worsens NOAC effectiveness without any safety benefit. In this lecture, we will discuss the dosing strategies of NOACs based on randomised controlled trials and real-world evidence.

S 8

Updates on autoimmune encephalitis

Josep Dalmau
Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Facultat de Medicina, c/ Casanova 143, Neuroimmunology Lab P3A, CELLEX, Barcelona, Spain

Investigations over the last 15 years have revealed that many neurologic and psychiatric disorders are due to antibody-mediated mechanisms against neuronal proteins and neurotransmitter receptors. These discoveries have changed the landscape of how physicians approach the diagnosis and treatment of these patients. Indeed, cases of rapidly progressive memory loss, psychosis, seizures, abnormal movements or impaired levels of consciousness previously considered idiopathic are now known to be mediated by antibodies and curable with immunotherapy. In my presentation, I will show the process of discovery of these diseases from bedside to bench and will describe the main clinical manifestations and immunological triggers. On a more basic level, I will describe the underlying pathogenic mechanisms and show how some antibodies can cause symptoms through their functional interaction with the synaptic protein targets. Novel treatment strategies have improved patient outcomes and are helping us to understand other diseases in which the same receptors are affected by other mechanisms.

Autoimmune movement disorders

S 9

Bettina Balint
University Hospital Heidelberg, Germany

Autoimmune movement disorders are rare but not-to-be-missed diagnoses because of the treatment implications: the earlier immunotherapy is initiated, the better the outcome. Movement disorders may be the first or most prominent presentation of autoimmune encephalitis and can present with characteristic phenotypes, with associated red flags or other diagnostic clues. Importantly, they may also be a differential diagnosis of degenerative disease, particularly when signs and symptoms develop slowly. This lecture discusses the broadening spectrum of phenotypes and antibodies, characteristic presentations and red flags, differences in the underlying immunopathophysiology, and updates on newest developments in the field.

Insights and updates on stroke rehabilitation

S 10

John Krakauer
Brain, Learning, Animation, and Movement Lab, Johns Hopkins University School of Medicine, USA

Animal experiments suggest that enriched environments and intense training regimens can restore neurological function. I will discuss translating these ideas into the treatment of post-stroke hemiparesis in humans using a novel form of immersive animation.

Mild behavioural impairment as a proxy marker for Alzheimer disease: what do biomarkers tell us?

S 11

Zahinoor Ismail
Hotchkiss Brain Institute, Canada; O'Brien Institute for Public Health, University of Calgary, Canada

Early detection of Alzheimer disease (AD) is essential to optimise treatment outcomes. Failure to discover a disease-modifying drug results in poor recruitment of patients and little improvement of early-phase illness. Case ascertainment of prodromal and preclinical AD based on standard cognitive tests alone is imprecise and is associated with screen failure in clinical trials. This inflates trial costs when cases were later confirmed by cerebrospinal fluid and/or positron emission tomography studies. This inflated cost may be prohibitive for some drug developers and may render a trial infeasible. In clinical care, imaging and biomarkers are expensive and often inaccessible. Simple, inexpensive, and scalable approaches to detect the at-risk population are needed. As neuropsychiatric symptoms (NPS) can occur early in the disease course, systematic incorporation of NPS, which is manifested as mild behavioural impairment (MBI), provides such an opportunity. MBI is an at-risk state for incident cognitive decline and dementia, characterised by the de novo emergence and persistence of NPS in later life. For some, these new-onset NPS are the initial manifestation of neurodegeneration before cognitive decline. MBI has a separate cognitive and functional trajectory from late-life psychiatric illness, with a faster decline and higher incidence of dementia. However, traditional methods in research and clinical care have not differentiated longstanding/recurrent psychiatric conditions in late life from new-onset NPS. Rather, these separate clinical entities are conflated, with only symptom severity differentiating them. In this presentation, we will (1) discuss the genesis of the MBI construct, (2) review longitudinal data for MBI and incident cognitive decline and dementia in both cognitively normal and MCI populations, and (3) explore the rapidly emerging database linking MBI with AD biomarkers for amyloid, tau, and neurodegeneration.

Management of behavioural and psychological symptoms of dementia: a psychiatrist viewpoint

S 12

John Chan
Department of Psychiatry, Kwai Chung Hospital, Hong Kong

Behavioural and psychological symptoms of dementia (BPSD) is common among older people with dementia. Managing older people with dementia is challenging for carers and professionals. Behavioural or environmental modifications may not always be able to help these patients. BPSD can cause significant stress to the patients and carers. Drug treatment may be necessary for effective management. In this session, Dr John Chan will share the management of BPSD with you.

Nusinersen in adults with spinal muscular atrophy: how to define treatment response?

S 13

Elena Pegoraro
University of Padova, Italy

Spinal muscular atrophy (SMA) is an autosomal recessive neuromuscular disorder caused by homozygous deletion or intragenic mutation of the survival motor neuron 1 (SMN1) gene that encodes the SMN protein, which is essential for motor neuron survival. A limited amount of functional SMN protein is produced by another gene, SMN2, which is located on chromosome 5q. SMN2 differs from SMN1 by few nucleotides, one of which creates an alternative splicing motif in exon 7 that largely exclude it from the mature SMN2 mRNA. Nusinersen, an antisense oligonucleotide that targets pre-mRNA splicing of the SMN2 gene, is the first medication approved for SMA. Following two successful pivotal trials (ENDEAR for early-infantile-onset SMA and CHERISH for later-infantile-onset SMA), the drug was approved by Food and Drug Administration in 2016. As of mid-2021, >11 000 patients with SMA have been treated with nusinersen. Several studies have reported additional data in older children and adults and have expanded our knowledge on safety and efficacy of the drug in a larger patient population. In this presentation, clinical data, clinical programmes, and latest evidence of using nusinersen in adult patients with SMA are discussed, as are experience on multidisciplinary management of adult SMA and expectations of patients toward SMA treatment.

New-generation drugs for migraine prevention: anti–calcitonin gene-related peptide versus conventional preventives

S 14

Uwe Reuter
Charité - Universitätsmedizin Berlin, Germany

Prevention for migraine is important in improving patients' quality of life. Commonly used preventive medications for episodic migraine include β blockers, antiepileptics, and antidepressants. These conventional medications are not specifically developed for migraine and often lead to low compliance over time owing to tolerability issues and perceived lack of efficacy. The novel anti–calcitonin gene-related peptide (CGRP) drugs change the treatment landscape and provide an option with excellent efficacy and safety profile. In a study of anti-CGRP drug versus traditional preventive drug, erenumab not only shows superior tolerability and efficacy against topiramate but also vastly improves quality of life. The findings provide better guidance in clinical decision making for preventive medication of migraine and in the role of anti-CGRP drugs in migraine prevention.

B-cell therapy: new era in management of multiple sclerosis

S 15

Ludwig Kappos
Research Center Clinical Neuroimmunology and Neuroscience Basel (RC2NB), University Hospital and University of Basel, Basel, Switzerland

The pathophysiology of multiple sclerosis involves B cells. Anti-CD20 monoclonal antibodies that induce B-cell depletion are effective disease-modifying therapies for multiple sclerosis. Ofatumumab, a fully human antibody used to treat chronic leukaemia, binds a region distinct from that of other anti-CD2 antibodies, including the smaller and the larger loop of CD20 receptors. In experimental models, a high-binding affinity and slow off-rate (slow dissociation of the binding between ofatumumab and the CD20 receptor in B cells) result in efficient B-cell lysis, which is mediated through complement-dependent and, to a lesser extent, antibody-dependent cytotoxicity. In patients with multiple sclerosis, ofatumumab can be given at lower doses than in patients with chronic lymphocytic leukaemia or rheumatoid arthritis. Ofatumumab can be administered subcutaneously by the patient after initial doses are given under medical supervision. In active controlled trials involving patients with relapsing multiple sclerosis, the relapse rate was significantly lower with ofatumumab than with teriflunomide. With ofatumumab in place, management of multiple sclerosis will enter a new era.

Update in magnetic resonance imaging in multiple sclerosis guidelines

S 16

Frederik Barkhof
Department of Radiology & Nuclear Medicine, VU University Medical Centre, Amsterdam, The Netherlands; Queen Square Institute of Neurology & Centre for Medical Image Computing, University College London, UK

The 2015 Magnetic Resonance Imaging in Multiple Sclerosis (MAGNIMS) and 2016 Consortium of Multiple Sclerosis Centres guidelines on the use of magnetic resonance imaging (MRI) in diagnosing and monitoring of multiple sclerosis made an important step towards appropriate use of MRI in routine clinical practice. Since then, there have been substantial relevant advances including the 2017 revisions of the McDonald diagnostic criteria, renewed safety concerns regarding intravenous gadolinium-based contrast agents, and the value of spinal cord MRI for diagnostic, prognostic, and monitoring purposes. These developments suggest a changing role of MRI in the management of multiple sclerosis. The 2021 revision of the previous guidelines on MRI use in patients with multiple sclerosis combines recommendations from the MAGNIMS, Consortium of Multiple Sclerosis Centres, and North American Imaging in Multiple Sclerosis Cooperative, and translates research findings into clinical practice to improve the use of MRI for diagnosis, prognosis, and monitoring of individuals with multiple sclerosis. We recommend changes in MRI acquisition protocols such as emphasising the value of three-dimensional fluid-attenuated inversion recovery as the core brain pulse sequence to improve diagnostic accuracy and its ability to identify new lesions to monitor treatment effectiveness. We provide recommendations for the judicious use of gadolinium-based contrast agents for specific clinical purposes. Additionally, we extend the recommendations to the use of MRI in patients with multiple sclerosis in childhood, during pregnancy, and in the post-partum period. Finally, we discuss promising MRI approaches for clinical practice in the near future.

FP 1

Low-density lipoprotein cholesterol and risk of recurrent vascular events in Chinese patients with ischaemic stroke with or without significant atherosclerosis

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Background: A low-density lipoprotein cholesterol (LDL-C) level of <1.80 mmol/L (<70 mg/dL) was reported to be associated with a reduced risk of major adverse cardiovascular events (MACE) in Caucasian ischaemic stroke patients with atherosclerosis. It is uncertain whether this finding can be generalised to Asians or whether similar LDL-C targets should be adopted in stroke patients without significant atherosclerosis.
Methods: We performed a prospective cohort study and recruited consecutive Chinese patients with ischaemic stroke who underwent magnetic resonance angiography of the intra- and cervico-cranial arteries at the University of Hong Kong between 2008 and 2014. Serial post-event LDL-C levels were measured. Patients with a mean post-event LDL-C level of <1.80 were compared with those with a level of ≥1.80 mmol/L in terms of the risk of MACE, stratified by the presence or absence of significant (>50%) large artery disease (LAD) and by ischaemic stroke subtypes.
Results: 904 patients (mean age, 69±12 years; 60% men) were followed up for a mean of 6.5±2.4 years, with a mean of nine LDL-C measurements per patient. Regardless of LAD status, patients with a mean post-event LDL-C level of <1.80 mmol/L was associated with a lower risk of MACE (LAD positive: multivariate-adjusted subdistribution hazard ratio [SHR]=0.65, 95% confidence interval [CI]=0.42-0.99, P<0.05; LAD negative: multivariate-adjusted SHR=0.53, 95% CI=0.32-0.88, P<0.05). Similar findings were noted in patients with ischaemic stroke attributable to large artery atherosclerosis (multivariate-adjusted SHR=0.48, 95% CI=0.28-0.84, P<0.05) and in patients with other ischaemic stroke subtypes (multivariate-adjusted SHR=0.64, 95% CI=0.43-0.95, P<0.05).
Conclusions: A mean LDL-C level of <1.80 mmol/L was associated with a lower risk of MACE in Chinese ischaemic stroke patients with or without significant LAD. Further randomised trials to determine the optimal LDL-C cut-off in stroke patients without significant atherosclerosis are warranted.

FP 2

Oedematous woman with demyelinating polyneuropathy: a missing letter in the acronym of POEMS

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POEMS (polyneuropathy, organomegaly, endocrinopathy, M-protein, and skin changes) is a rare paraneoplastic syndrome caused by plasma cell disorder. Patients with POEMS may be misdiagnosed as having demyelinating polyneuropathy, and the correct diagnosis is often delayed. Extravascular volume overload is a common diagnostic criterion of POEMS, but it is not included in the acronym and may be an overlooked diagnostic clue. We describe a case of POEMS syndrome with extravascular volume overload. We highlight the difficulties in getting histological confirmation of monoclonal gammopathy in POEMS and the importance of a targeted bone biopsy.

FP 3

Case series of everolimus use in adult tuberous sclerosis complex related renal angiomyolipoma and refractory seizures

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Background: Tuberous sclerosis complex (TSC) is a known genetic disease leading to multiple tumour growth and refractory seizures. TSC-associated epilepsy is usually difficult to control despite use of antiepileptic drugs. Surgical intervention is often required for complicated tumours. Everolimus, a mammalian target of rapamycin inhibitor, is a disease-modifying agent in controlling TSC-related refractory seizures and tumours. We report our experience of everolimus use in these patients.
Methods: Adult patients with TSC who were treated with everolimus at our hospital were identified.
Results: Four patients aged 26 to 52 years were treated with everolimus for renal angiomyolipoma (n=3) or refractory seizures (n=1). Everolimus 5-10 mg daily was initiated, with dose titration according to trough level during follow-up. Three patients were taking CYP3A4 inducer before treatment. One patient developed severe gastrointestinal bleeding and thus treatment was discontinued. One patient developed grade 2 stomatitis and thus treatment was interrupted and dose reduced. One patient with previous sirolimus exposure experienced angiomyolipoma progression later. One patient with refractory seizures experienced increasing seizure frequency during the drug titration phase and self-discontinued treatment.
Conclusion: Everolimus use in patients with TSC is challenging. Concomitant use of CYP3A4 inducer may make medication initiation and titration difficult. Adverse events are common although severe adverse events requiring discontinuation of treatment are rare. Therapeutic drug monitoring and adverse event management are key to successful treatment.

FP 4

Is oxygen desaturation the link between sleep apnoea and cerebral small vessel disease burden in patients with stroke?

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Background: Obstructive sleep apnoea (OSA) is a modifiable risk factor for stroke and cerebral small vessel disease (SVD). It is quantified by the frequency of apnoea and hypopnoea in sleep (apnoea-hypopnoea index, AHI) via overnight polysomnography assessment. However, AHI has limited capacity to predict clinical outcomes. Whether novel OSA-related metrics such as depth and duration of oxygen desaturation are associated with global cerebral SVD burden in stroke patients has not been explored.
Methods: We prospectively recruited patients with transient ischaemic attack (TIA) or minor stroke at the Queen Mary Hospital Acute Stroke Unit and TIA/Stroke Outpatient Clinics. They underwent magnetic resonance imaging of the brain, and their neuroimaging markers of SVD (lacunes, cerebral microbleeds, white matter hyperintensities, basal ganglia perivascular spaces, and brain atrophy) were graded using validated scoring scales, and the global SVD burden was determined. Home polysomnographic test was performed at 1-year after stroke onset. Associations between OSA-related metrics and SVD burden were determined using multivariate regression models.
Results: Among 60 patients with TIA/minor stroke (mean age, 64 years; 68% men), total SVD score was positively associated with AHI (odds ratio [OR]=1.04, 95% confidence interval [CI]=1.00-1.07, P=0.041), oxygen desaturation index (OR=1.09, 95% CI=1.01-1.09, P=0.029), and percentage of total desaturation time from total sleep time (OR=1.04, 95% CI=1.00-1.10, P=0.05), after adjusting for age, sex, vascular risk factors (blood pressure, diabetes, atrial fibrillation, body mass index, smoking), and polysomnography recording time. Similar patterns were observed in basal ganglia perivascular space score and deep white matter hyperintensity burden but not in other SVD markers.
Conclusion: OSA-related metrics (oxygen desaturation index and percentage of total desaturation time from total sleep time) were positively associated with SVD burden in patients with TIA/minor stroke. Duration of oxygen desaturation may be associated with SVD development in patients with stroke.

FP 5

Direct oral anticoagulant and antiepileptic drug combination on risk of thromboembolism and major bleeding: a territory-wide review

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Background: Clinical relevance of the drug-drug interactions between direct oral anticoagulants (DOACs) and antiepileptic drugs (AEDs) is uncertain. Determining the thromboembolic and major bleeding risks in different DOAC/AED combinations may inform appropriate treatment. This study aimed to elucidate the risk of ischaemic stroke and major bleeding in patients with different DOAC/AED combinations.
Methods: We performed a territory-wide retrospective review of patients who received concomitant DOACs (dabigatran, apixaban, rivaroxaban, and edoxaban) and AEDs between 1 January 2015 and 31 December 2020 in Hong Kong. Propensity-score weighting was used for comparisons between DOACs with cytochrome P450 (CYP) or P-glycoprotein (P-gp) non-modulating AEDs (gabapentin, pregabalin) and DOACs with CYP/P-gp–modulating AEDs (phenytoin, lamotrigine, levetiracetam, valproate, and topiramate). Secondary analyses included between-AED and between-DOAC comparisons. Primary outcome was ischaemic stroke. Secondary outcomes were major bleeding and death. Outcome events were analysed using cox regression.
Results: 8753 patients with DOAC/AED combination were identified; the median follow-up duration was 6 years. CYP3A4/P-gp–modulating AEDs (phenytoin, lamotrigine, levetiracetam) were associated with a higher risk of ischaemic stroke (adjusted hazard ratio [aHR]=2.30, 95% confidence interval [CI]=1.80-2.93, P<0.001), major bleeding (aHR=1.81, 95% CI=1.40-2.33, P<0.001), and death (aHR=1.89, 95% CI=1.67-2.14, P<0.001), compared with CYP3A4/ P-gp non-modulating AEDs. Levetiracetam and valproate were associated with a higher risk of ischaemic stroke. When used with AEDs, apixaban was associated with a lower risk of ischaemic stroke (aHR=0.57, 95% CI=0.37-0.89, P=0.01) and death (aHR=0.80, 95% CI=0.64-0.99, P=0.044), compared with other DOACs. AED polytherapy was associated with an increased risk of ischaemic stroke (aHR=2.35, 95% CI=1.11-2.11, P=0.009) and death (aHR=1.47, 95% CI=1.14-1.91, P=0.003), compared with AED monotherapy.
Conclusion: Drug-drug interactions between DOACs and AEDs may be clinically relevant. Compared with gabapentin and pregabalin, phenytoin, levetiracetam, valproate, and AED polytherapy were associated with higher risks of ischaemic stroke. Caution should be exercised on AED selection for DOAC patients. Further pharmacokinetic studies are warranted to elucidate the reasons behind these observations.

Efficacy of erenumab for migraine in a private hospital in Hong Kong

P 1

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Background: Migraine is a common neurological problem. In the STRIVE study, 60%, 40%, and 20% of patients on erenumab for 24 weeks had 50%, 75%, and 100% reduction in migraine at 52 weeks, respectively. Erenumab has been used for patients with trigeminal neuralgia or medication overuse headache. We studied the efficacy of erenumab in patients with migraine or cluster headache.
Methods: Between May 2020 and July 2021, 16 women and two men aged 26 to 73 (mean, 46.1) years were treated with erenumab for migraine (n=17) or cluster headache (n=1). The mean duration of headache was 18.2 (range, 1-50) years. Five patients had auras.
Results: The mean number of medications prior to erenumab treatment was 4.2 (range, 2-7). The mean follow-up time after first dose of erenumab was 6.9±4.8 months. The mean number of erenumab injections received was 2.5 (range, 1-6). In the last follow-up, pain score improved by 81% from a median of 8 (range, 5-10) to 1.5 (range, 0-3) out of 10 (P<0.001). Pain frequency reduced by 75% from a median of 4 (range, 2-7) days to 1 (range, 0-3) day out of 7 days (P<0.001). One patient had elevated blood pressure and erenumab treatment was stopped; her migraine was controlled by other medications. Another patient had pre-existing condition of depression before erenumab treatment. No other significant adverse effects or complications were noted.
Conclusion: Erenumab, a CGRP blocker, is an effective preventive treatment for migraine, with pain score improved by 81% and pain frequency reduced by 75%. No significant adverse effect except hypertension was noted. Further study is necessary for better understanding of this drug.

Not too little and not too much: a case report of SCN4A-related congenital myopathy with two novel mutation variants

P 2

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Missense mutations of the sodium channel gene SCN4A of skeletal muscles are associated with diverse neuromuscular disorders. In addition to the dominant SCN4A gain-of-function mutation disorders such as hyperkalaemic periodic paralysis and paramyotonia congenita, loss-of-function (LOF) mutation disorders such as congenital myopathy and myasthenic syndromes are increasingly recognised. We report a case of late-onset congenital myopathy in a 28-year-old Chinese woman. She was found to have two novel compound heterozygous novel missense LOF SCN4A mutation. She was born preterm with hemiplegic cerebral palsy. Since teenage, she has had marked progressive proximal muscle weakness, along with prominent fatigability and myalgia. Tensilon test and anti-acetylcholine receptor antibody were negative. Dried blood spot metabolic screening and plasma acylcarnitine and urine metabolic profiles were unrevealing. Alpha-glucosidase activity was normal. She only had occasional hypokalaemia and mild creatine kinase elevation (up to 379 U/L). Electromyography showed neurogenic changes without myotonia. Short exercise protocol was negative despite cooling effect, and long exercise protocol exhibited decremental CMAP response in Fournier V pattern. Repeated muscle biopsies merely expressed non-specific myopathic changes. Eventually, next-generation sequencing genetic study with targeted panel confirmed the diagnosis. Our patient was identified to be heterozygous for two previously unreported missense variants in the SCN4A gene (OMIM*603967): NM_000334.4:c.1841A>T (p.Asn614Ile) and c.4420G>A (p.Ala1474Thr) (rs778417596). Analysis of family members confirmed the compound heterozygosity of the two variants; the healthy father and younger sister of the patient were heterozygous for c.4420G>A (p.Ala1474Thr) variant, whereas the mother was heterozygous for the c.1841A>T (p.Asn614Ile) variant. The two novel missense LOF SCN4A mutations resulted in neurophysiological features resembling hypokalaemic periodic paralysis. Molecular genetic study conferred an important role in identifying recessive mutations of congenital myopathy. Our case report expands the understanding of phenotypes in SCN4A mutations.

Predictors of poor outcome in intraarterial thrombectomy

P 3

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Background: Intraarterial thrombectomy is the standard treatment for acute ischaemic stroke with large vessel occlusion (LVO). However, patients with poor premorbid condition or having posterior circulation stroke may not be included in randomised controlled trials. At 3 months after intraarterial thrombectomy, functional outcome and mortality were not as good as expected. This study aims to identify the predictors of poor outcome for intraarterial thrombectomy.
Methods: Patients who underwent intraarterial thrombectomy for acute ischaemic stroke at Tuen Mun Hospital between 2019 and July 2021 were retrospectively included. Patients without LVO or intraarterial mechanical thrombectomy attempted were excluded. Primary outcomes were functional outcome (modified Rankin Scale [mRS] score) and mortality at 3 months. Baseline characteristics, stroke location, severity, number of passes, and success in recanalisation were analysed using univariate analysis. Significant variables were analysed using multivariate logistic regression to determine predictors for primary outcomes.
Results: Of the 196 patients included, 67.3% had poor functional outcome (mRS >2) at 3 months, and the mortality was 21.4%. Predictors of poor function outcome at 3 months included underlying diabetes mellitus, higher National Institute of Health Stroke Scale score at presentation, and failure to achieve racanalisation. Predictors for mortality at 3 months were underlying malignancy, premorbid mRS >1, and posterior circulation stroke.
Conclusion: Our cohort had higher proportion of patients with diabetes mellitus and higher National Institute of Health Stroke Scale score; we also included patients with premorbid mRS >1 and posterior circulation stroke. Thus, functional outcome and mortality of our patients were not as good as expected.

Unconscious and weak patient with COVID-19

P 4

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Coronavirus Disease 2019 (COVID-19) is known to affect the nervous system in different ways. COVID-19–associated encephalopathy has been reported in the literature. We present a case of COVID-19–associated encephalopathy in Hong Kong, with characteristic radiological findings at the midbrain. A 34-year-old obese man with good past health was infected with COVID-19 and required intubation and intensive care. Although he had improvement of the respiratory status and developed IgG antibody to the virus, he remained in deep comatose state after extubation and weaning of sedation, with a Glasgow Coma Scale score of 3 out of 15. Electroencephalography showed encephalopathic changes. Blood and cerebrospinal fluid tests for causes of decreased level of consciousness were unremarkable, including the autoimmune encephalitis panel. Magnetic resonance imaging of the brain showed T2 and FLAIR hyperintense signals at bilateral midbrain cerebral peduncles, with restricted diffusion and without contrast enhancement. After a course of intravenous immunoglobulin therapy, his level of consciousness recovered markedly, with the Glasgow Coma Scale score returning to 15. However, he remained tetraplegic owing to critical illness polyneuropathy. In our patient, a special pattern of brain lesion involving bilateral midbrain with symmetrical restricted diffusion at the cerebral peduncles was noted. The patient responded well to intravenous immunoglobulin therapy. This suggests an immune-mediated mechanism. More studies are needed to further characterise encephalopathy with this radiological pattern.

DH 1

Applicability of haemorrhagic transformation predicting tools in predicting post–tissue plasminogen activator symptomatic intracranial haemorrhage in local patients

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Administration of intravenous (IV) thrombolytic therapy within the therapeutic window is the standard of treatment for acute ischaemic stroke. Nonetheless, after IV thrombolytic therapy, a substantial number of patients may develop symptomatic intracranial haemorrhage (SICH) resulting in poor functional outcomes or even death. Factors related to a high haemorrhagic transformation rate include old age, Asian ethnicity, male sex, and large infarct on presentation. To accurately predict the haemorrhage transformation rate, multiple risk-predicting tools have been developed based on the patient characteristics. In the present study, we identify patients in the Kowloon Easter Custer who received IV thrombolytic therapy for acute ischaemic stroke. Scores of three risk-predicting tools were calculated on each patient: the Hemorrhage After Thrombolytic therapy score, the Glucose Race Age Sex Pressure Stroke Severity score, and the Safe Implementation of Thrombolytic therapy in Stroke score. The Glucose Race Age Sex Pressure Stroke Severity score had moderate performance, whereas the Hemorrhage After Thrombolytic therapy score and the Safe Implementation of Thrombolytic therapy in Stroke score did not perform well.

DH 2

Risk factors for key outcomes of motor neurone disease: a single-centre study

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Background: Motor neurone disease is a degenerative disease that causes progressive weakness. Patients usually die of respiratory failure or other complications within 2 to 3 years despite best medical care. To date, there is no cure for the disease. Local studies regarding the disease prognosis and outcomes are lacking.
Methods: Records of patients who were diagnosed with motor neurone disease in a local hospital between January 2008 and December 2017 were retrospectively reviewed. Key outcomes such as survival, assisted ventilation, and potential factors were collected. Descriptive analysis was performed for demographic data and patient outcomes. Kaplan-Meier survival curves were plotted for various independent variables, and Cox regression model was used for multivariate analysis.
Results: A total of 126 patients were included. The mean patient age at symptom onset was 55.3±11.6 years, and the male-to-female ratio was 1.8:1. The median duration from onset of symptom to diagnosis was 10 months; the median survival from symptom onset was 27 months. In multivariate analysis, longer duration of survival was associated with diagnostic delay (hazard ratio [HR]=0.96, 95% confidence interval [CI]=0.95-0.98, P<0.01) and use of riluzole (HR=0.49, 95% CI=0.31-0.78, P< 0.01). Older age at disease onset was associated with shorter duration of survival (HR=1.02, 95% CI=1.00-1.04, P=0.05). Use of assisted ventilation was predictive of a shorter interval from symptom onset to non-oral means of feeding (HR=1.93, 95% CI=1.16-3.23, P=0.01), whereas use of non-oral feeding was predictive of earlier need for assisted ventilation (HR=2.13, 95% CI=1.30-3.47, P<0.01).
Conclusion: Diagnostic delay and use of riluzole were associated with longer duration of survival, whereas older age at onset of symptom was associated with shorter duration of survival. A multidisciplinary approach is beneficial in managing patients with motor neurone disease.

DH 3

Retrospective review of clinical characteristics and effect of integrated clinic on patients with motor neuron disease in regional hospitals in Hong Kong

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Background: Motor neuron disease (MND) is a rare neurodegenerative disease with heterogenous clinical presentation and disease progression. Multidisciplinary care can extend survival, reduce unplanned hospitalisation, and improve quality of life. However, local clinical data on its efficacy are lacking.
Methods: This is a retrospective observational study of patients with MND in the Kowloon West Cluster between October 2014 and September 2019. Their clinical features, treatment, and survival were examined. Comparison was made between patients attending integrated MND clinic and those attending general neurology clinics.
Results: A total of 69 patients with MND were identified. The median patient age at diagnosis was 58.7 years. The annual incidence was 0.526/100 000/year, and the point prevalence in September 2019 was 1.387/100 000. Younger age at onset and delay in diagnosis were favourable prognostic factors. Compared with patients attending the general neurology clinic, patients attending the integrated MND clinic were more likely to sign the advance directive (62.5% vs 24.5%), have a higher rate of elective nasal intermittent positive pressure ventilation initiation (80% vs 30%) and a lower rate of unplanned intubation (0% vs 22.2%), and have a lower annual mean number of hospitalisation (0.88 vs 2.52) and shorter annual mean length of hospital stay (22.3 days vs 34.2 days). A trend towards longer median survival was observed in patients attending the integrated MND clinic (36.5 months vs 32.0 months).
Conclusion: Integrated MND clinic was associated with a higher rate of elective nasal intermittent positive pressure ventilation initiation, fewer hospital admissions, shorter length of stay, and fewer unplanned intubation.

DH 4

Prevalence and clinical predictors of aspiration pneumonia in patients with Parkinson disease in Hong Kong

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Introduction: Aspiration pneumonia is the leading cause of death in patients with Parkinson disease (PD). Local data in this aspect are lacking. We conducted a retrospective cross-sectional review on the prevalence of aspiration pneumonia in a cohort of patients with PD and evaluated the clinical characteristics predisposing to aspiration pneumonia.
Methods: Records of 267 consecutive patients with idiopathic PD who attended a local hospital between January 2019 and January 2020 were retrospectively reviewed through the Clinical Management System. Data collected included demographics, disease severity, cognitive assessment, medications, psychiatric comorbidities and medications, swallowing assessment, and management of aspiration pneumonia. Patients who hospitalised for aspiration pneumonia were compared with those who did not.
Results: The mean patient age was 70.59±9.75 years, and the mean disease duration was 10.13±5.28 years. The prevalence of hospitalisation for aspiration pneumonia was 5.6% (n=15). In univariate analysis, patients hospitalised for aspiration pneumonia were of older age (77.9 vs 70.15, P=0.002), higher Hoehn and Yahr (H&Y) stage (3.9 vs 2.79, P=0.001), higher Unified Parkinson's Disease Rating Scale III score during ON phase (37.79 vs 21.3, P<0.001), lower Montreal Cognitive Assessment (MoCA) score (13 vs 20.9, P=0.003), more use of antipsychotics (40% vs 11.5%, P=0.007), and more prior fall admissions (73.3% vs 32.9%, P=0.03), compared with those did not hospitalise for aspiration pneumonia. In those at H&Y stage 4-5, those hospitalised for aspiration pneumonia had a lower levodopa equivalent dose (571.91 mg vs 834.91 mg, P=0.039) and a lower MoCA score (9.67 vs 18.03, P=0.005). Multivariate analysis showed that a lower MoCA score was the only significant factor for hospitalisation for aspiration pneumonia in all H&Y groups and in H&Y 4-5 groups.
Conclusion: A lower MoCA score was a predictor for hospitalisation for aspiration pneumonia in patients with PD. Clinicians should be alert of these risk factors and optimise management to prevent aspiration pneumonia in patients with PD.

10-year review of inflammatory myopathy in a regional hospital in Hong Kong

DH 5

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Background: Inflammatory myopathy is an autoimmune disease with heterogenous clinical manifestation. Recent advancements in myositis specific antibody (MSA) and myositis associated antibody (MAA) have helped classify different subtypes and identify different clinical associations. This study aims to evaluate the clinical features, diagnostic workup, and extramuscular association of inflammatory myopathy.
Methods: This was a retrospective cohort study in a single centre. Patients with a clinical diagnosis of inflammatory myopathy between 1 January 2010 and 31 May 2020 were analysed. Their serological workup, histological findings, lung involvement, and malignancy association were reviewed.
Results: A total of 106 patients were included. Creatine kinase level was variable in different subtypes of inflammatory myopathy, with higher levels among immune-mediated necrotising myopathy. MSA/MAA were detected in 87% of patients. Anti-TIF1 was associated with malignancy, whereas anti-MDA5 and anti-synthetase antibodies were associated with interstitial lung disease.
Conclusion: Creatine kinase levels, MSA/MAA, and histological findings are important in the diagnosis and management of inflammatory myopathy. Further studies with larger cohorts are warranted.

Autonomic dysfunction in Chinese patients with idiopathic Parkinson disease in a public hospital in Hong Kong

DH 6

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Background: Autonomic dysfunction is common in patients with idiopathic Parkinson disease (PD) and severely impacts activities of daily living. Patients may experience symptoms before motor abnormalities. Data related to autonomic dysfunction in patients with idiopathic PD in Hong Kong are lacking. We aimed to explore the occurrence of dysautonomia in a regional hospital and evaluate the correlations of various demographic variables and disease-related factors with the severity of dysautonomia.
Methods: This was a prospective observational study of patients with idiopathic PD who had long-term follow-up in a regional hospital. The demographics and disease-related factors were recorded. The autonomic function was evaluated using the Ewing's battery. Autonomic symptoms were evaluated using the Chinese version of the Scale for Outcomes in Parkinson Disease – Autonomic Dysfunction (SCOPA-AUT).
Results: A total of 50 patients who met the criteria of the United Kingdom Parkinson Disease Society Brain Bank were enrolled prospectively. Their motor severity was between 1 and 4 of modified H&Y (H&Y) stage. The median patient age was 64.3, and the mean disease duration was 80.9 months. 36 (72%) patients were determined to have autonomic dysfunction according to the Ewing's battery. Severity of dysautonomia correlated with H&Y stage, sex, levodopa equivalent dose, disease duration, and SCOPA-AUT total score. The SCOAP-AUT total score also correlated with motor severity. H&Y stage and age were the main determinants of autonomic dysfunction.
Conclusion: Autonomic dysfunction is a common non-motor manifestation of idiopathic PD. It can occur even in the early stages of PD. Severity of dysautonomia increases with motor severity and disease duration. The Chinese version of SCOPA-AUT is a reliable screening tool for dysautonomia in patients with PD.

Case series of familial amyloid polyneuropathy in Hong Kong

DH 7

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Background: Familial amyloid polyneuropathy (FAP) is an autosomal dominant inheritable disease caused by mutations in the gene encoding transthyretin (TTR). Patients typically have a combination of peripheral neuropathy, dysautonomia, and restrictive cardiomyopathy. We aimed to examine the local FAP genotype and phenotype, clinical course, disease burden, and treatment responses, and to propose a medical monitoring and treatment algorithm.
Methods: We included the largest local case series comprising 23 symptomatic FAP patients and seven pre-symptomatic TTR mutation carriers. Patient demography, genotypes, clinical findings, treatments, and outcomes were recorded. Comparisons were made between local patients and previously reported patients from endemic or nearby regions.
Results: 23 symptomatic patients and seven asymptomatic mutation carriers were included. In Hong Kong, p.Ala117Ser was the most prevalent pathological variant, followed by p.Gly87Glu and p.Val50Ala. We observed a bi-modal peak of symptom onset, and most patients had a rapid progressive disease course regardless of their onset age. A mixed pattern of cardiac and neuropathic involvement was common in both early and late onset. Carpal tunnel syndrome was common and frequently preceded other symptoms by years. Regarding treatment, five of the six patients who received liver transplant died within 10 years. Eight patients received tafamidis treatment; early data suggested symptomatic improvement especially in gastrointestinal symptoms.
Conclusion: We proposed a follow-up and treatment algorithm for better coordinated management of FAP in Hong Kong.

Pilot study on the use of a smartphone application in assessing motor and gait disability in patients with Parkinson disease

DH 8

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Introduction: Patients with idiopathic Parkinson disease (IPD) is commonly assessed using the Unified Parkinson's Disease Rating Scale (UPDRS), which is administrated by trained personnel and is subjective. 'mHealth' is advocated by the World Health Organization and is defined as medical practice supported by the use of mobile devices. We aimed to compare phone-based kinematic tests with UPDRS in patients with IPD.
Method: Patients with IPD were recruited from outpatient clinics of Queen Mary Hospital and Tung Wah Hospital. Patients were assessed using a full rating of MDS-UPDRS (Part III motor) by a trained clinician, followed by the use of a smartphone application called EncephalogClinic, which obtains objective phone-based motor parameters (PMP) based on the timed up and go test (TUGT), finger tap test (FTT), and tests for tremor.
Results: 65 normal subjects and 65 patients with IPD (mean age, 65 years) who had a median of Hoehn and Yahr stage II were recruited. The UPDRS gait sub-score correlated with the total completion time (ρ=0.53, P<0.001), stand-up time (ρ=0.82, P<0.001), and mean step length (ρ= -0.56, P=0.005) of 5-metre TUGT. The UPDRS bradykinesia sub-score correlated with mean press radius (ρ=0.52, P=0.01), mean time between successive taps (ρ=0.58, P=0.03), and the total number of taps (ρ= -0.50, P=0.02) in FTT. A cut-off at 62 cm of stride length during 5-metre TUGT had sensitivity of 75.4% and specificity of 76.9% to distinguish IPD patients from normal subjects (area under the curve=0.821, 95% CI=0.744-0.898, P<0.001). None of the phone-based motor parameters could predict fall in the 6-month follow-up period.
Conclusion: The smartphone application can be self- or home-administered; its TUGT, FTT, and tests for tremor correlated with motor scores of UPDRS and hence are potentially useful for remote monitoring of motor disability in patients with IPD. This is especially relevant during COVID-19 pandemic when patients may not be able to come to outpatient clinics for follow-up. The 5-metre TUGT and its mean stride length also serve as a potential self-screening tool of IPD. Further large-scale studies are warranted to determine the predictive value for falls by the phone-based motor parameters.

DH 9

Factors associated with generalisation of ocular myasthenia gravis and myasthenic crisis

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Background: Myasthenia gravis (MG) is an autoimmune disorder targeting the neuromuscular junction. It can be classified as ocular MG (OMG) or generalised MG (GMG). OMG may progress to GMG. We aimed to identify factors associated with progression of OMG to GMG.
Methods: This was a retrospective cohort study. Records of patients with MG in Tuen Mun Hospital between 2000 and 2020 were reviewed. Adult patients with seropositive OMG at presentation were included. Patients with GMG at onset, those seronegative or with unknown antibody status, and those with OMG for <3 years were excluded. Primary outcome was the progression to GMG; secondary outcome was the occurrence of MG crisis. Clinical characteristics between OMG-O and OMG-G and clinical characteristics in OMG-G patients with or without MG crisis were compared.
Results: A total of 97 patients with MG were included. 47.4% of patients with OMG progressed to GMG. OMG progression to GMG was positively associated with the presence of thymoma (P=0.001) and the presence of high anti-AChR titre (P=0.008). Among the 46 OMG-G patients, 17.4% experienced MG crisis. MG crisis was associated with older age of onset (P=0.020) and female sex (P=0.028).
Conclusion: The presence of thymoma and high anti-AChR titre were associated with progression of OMG to GMG. Older age of onset of OMG and female sex were associated with MG crisis.

DH 10

Clinical predictors and outcome of late-onset post-stroke seizure: a 10-year retrospective observational study

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Background: Stroke is the leading cause of epilepsy. Late-onset post-stroke seizure (PSS), which occurs 2 weeks after an index stroke, is not an uncommon sequela. Treatment and control of seizure can be effective if the clinical course is delineated. However, data on late-onset PSS in Hong Kong are scarce. This retrospective study aims to determine the predictors and clinical outcomes of late-onset PSS in Hong Kong.
Methods: We included patients with ischaemic stroke, intracerebral haemorrhage or subarachnoid haemorrhage who were admitted to Ruttonjee Hospital between 1 July 2008 and 30 June 2018. 117 patients with late-onset PSS and 232 patients with stroke but no seizure were included. Those with stroke but no seizure and those with late-onset PSS were compared in terms of clinical characteristics and outcomes, as were those with single late-onset PSS and those with recurrent late-onset PSS.
Results: Independent predictors for late-onset PSS were cortical involvement (odds ratio [OR]=8.82, P<0.001), ischaemic stroke with haemorrhagic transformation (OR=3.34, P=0.034), and male sex (OR=1.83, P=0.035). Good functional outcomes (modified Rankin Scale score of 0-2) after stroke was a protective predictor against late-onset PSS (OR=3.62, P=0.010). Independent predictors for recurrent late-onset PSS were age <70 years (OR=3.62, P=0.010) and cortical involvement (OR=3.09, P=0.034).
Conclusion: Cortical involvement, ischaemic stroke with haemorrhagic transformation, and male sex were independent predictors for late-onset PSS, whereas good functional outcomes was a protective factor for late-onset PSS. Young age and cortical involvement predicted recurrence of late-onset PSS.

DH 11

Impulse control and related behavioural disorders in patients with idiopathic Parkinson disease treated with different dopamine agonists

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Objective: To assess the clinical characteristics and risk factors of impulse control and related behavioural disorders (ICRD) and their incidence in Chinese patients with idiopathic Parkinson disease (IPD) treated with different dopamine agonists.
Methods: This is an observational cohort study based on clinical interviews and medical records of patients with IPD who were treated with dopamine agonists for >6 months in Yan Chai Hospital, Princess Margaret Hospital, and Pamela Youde Nethersole Eastern Hospital. Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease was used to screen for ICRD, followed by the use of ICRD diagnostic criteria. Clinical characteristics, risk factors, and incidence of ICRD among different dopamine agonists were examined.
Results: Of 512 patients included, 78 (15.2%) had ever developed ICRD. Pathological gambling and compulsive shopping were the most frequent subtypes. Incidence of ICRD was analysed in 430 patients who took their first, single dopamine agonist. Bromocriptine and rotigotine users had lower ICRD incidence rates. In multivariate analysis, higher risk of ICRD was independently associated with pramipexole (adjusted hazard ratio [HR]=7.65, 95% confidence interval [CI]=2.94-19.92, P<0.001) and ropinirole (adjusted HR=6.69, 95% CI=2.90-15.39, P<0.001), compared with bromocriptine. Rotigotine and bromocriptine appeared to have similar risks. ICRD remained independently associated with male sex (adjusted HR=2.11, 95% CI=1.07-4.15, P=0.030], younger age (<50 years) of IPD onset (adjusted HR=2.82, 95% CI=1.45-5.49, P=0.002), and history of psychiatric disorders (adjusted HR=2.07, 95% CI=1.11-3.88, P=0.023).
Conclusion: Bromocriptine and rotigotine have a lower risk of ICRD, compared with pramipexole and ropinirole. Male sex, younger age of IPD onset, and history of psychiatric disorders are risk factors for ICRD.

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Take back your life with Keppra

Keppra offers patients ease of administration, efficacy and long-term tolerability across the different stages of their life

- Levetiracetam monotherapy demonstrated comparable rates of seizure freedom to CBZ CR in patients with newly diagnosed focal epilepsy[1]
- The risk of MCMs in infants born to WWE with levetiracetam is signicantly lower compared to the lowest dose of VPA (≤650mg; p=0.0069)[2]
- Levetiracetam is effective in focal epilepsy in a variety of special populations of patients with focal epilepsy, including BTRE, PSE, the elderly and children above 4 years[3-6]

Available in different formulations

SAFETY INFORMATION – SIDE EFFECTS

Contraindication

- Hypersensitivity to the active substance or other pyrrolidone derivatives or to any of the excipients

Warnings and precautions

- Discontinuation of treatment – withdraw gradually
- Renal or hepatic impairment – dose adjustment might be necessary. Patients with renal impairment may require dose adjustment. In patients with severely impaired hepatic function, assessment of renal function is recommended before dose selection
- Depression and/or suicidal ideation – small increased risk of suicidal thoughts and behaviour – monitor for signs of depression and/or suicidal ideation and behaviours
- Paediatric population – available data in children did not suggest impact on growth and puberty. However, long term effects on learning, intelligence, growth, endocrine function, puberty and childbearing potential in children remain unknown

Very common adverse reactions (1/10)

- Somnolence, headache, nasopharyngitis

Common adverse reactions (1/100 to <1/10)

- Asthenia/fatigue, dizziness, anorexia, depression, hostility/aggression, anxiety, insomnia, nervousness/irritability, convulsion, balance disorder, lethargy, tremor, vertigo, cough, abdominal pain, diarrhoea, dyspepsia, vomiting, nausea, rash

ABBREVIATIONS BTRE: brain tumor Related Epilepsy; CBZ: carbamazepine; CR: controlled release; MCMs: major congenital malformations; PSE: post-stroke epilepsy; VPA: valproate; WWE: women with epilepsy

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Name of medicinal product: Keppra **Qualitative and quantitative composition:** *Tablets* 250 mg / 500 mg / 1000 mg; *Oral Solution* 100 mg/ml; *Concentrate for solution for infusion* 100 mg/ml **Indication:** ***Monotherapy*** Treatment of partial onset seizures with or without secondary generalisation in adults and adolescents from 16 years of age with newly diagnosed epilepsy ***Adjunctive therapy*** Treatment of partial onset seizures with or without secondary generalisation in adults, adolescents and children from 1 month of age (tablets and oral solution) or 4 years of age (concentrate for solution for infusion) with epilepsy, myoclonic seizures in adults and adolescents from 12 years of age with Juvenile Myoclonic Epilepsy, primary generalised tonic-clonic seizures in adults and adolescents from 12 years of age with Idiopathic Generalised Epilepsy. **Dosage and Route of Administration** ***Dosage*** **Tablets** Not adapted for use in infants and children under the age of 6 years. The available dose strengths of the tablets are not appropriate for initial treatment in children weighing less than 25 kg, for patients unable to swallow tablets or for the administration of doses below 250 mg. ***Monotherapy*** Adults and adolescent from 16 years of age Recommended starting dose is 250 mg twice daily and increased to an initial therapeutic dose of 500 mg twice daily after 2 weeks. Dose can be further increased by 250 mg twice daily every 2 weeks. Maximum dose is 1500 mg twice daily. Childen and adolescent below 16 years No data are available. ***Add-on therapy*** Adults (≥18 years) and adolescent (12 to 17 years) weighing ≥50 kg Initial therapeutic dose is 500 mg twice daily and can be started on the first day of treatment. Daily dose can be increased up to 1500 mg twice daily. Dose changes can be made in 500 mg twice daily increases or decreases every 2 to 4 weeks. Infants aged from 6 to 23 months, children aged from 2 to 11 years of age and adolescents aged from 12 to 17 years of age weighing < 50 kg Oral solution is preferred for use in infants and children under the age of 6 years. For children or adolescent of 25 kg, the starting dose should be 250 mg twice daily with a maximum dose of 750 mg twice daily. The lowest effective dose should be use. Children weighing ≥50 kg Same dosing regimen as in adults. Infants aged from 1 to 6 months Oral solution should be used. **Oral Solution** Preferred formulation for use infants and children under the age of 6 years. Children 6 years and above Oral solution should be used for doses under 250 mg, for doses not multiple of 250 mg when dosing recommendation is not achievable by taking multiple tablets and for patients unable to swallow tablets. ***Monotherapy*** No data are available. ***Add-on therapy*** Infants aged from 1 to less than 6 months Initial therapeutic dose is 7 mg/kg twice daily and can be increased up to 21 mg/kg twice daily. Dose changes should not exceed increases or decreases of 7 mg/kg twice daily every 2 weeks. The lowest effective dose should be used. Infants from 6 months of age, children and adolescents weighting <50 kg Initial therapeutic dose is 10 mg/kg twice daily. Dose can be increased up to 30 mg/kg twice daily. Dose changes should not exceed increases or decreases of 10 mg/kg twice daily every 2 weeks. The lowest effective dose should be used. Children 50 kg or greater Same dosing regimen as in adults. **Concentrate for solution for infusion** An alternative for patients when oral administration is temporarily not feasible. ***Monotherapy*** Adults and adolescent from 16 years of age Recommended starting dose is 250 mg twice daily and increased to an initial therapeutic dose of 500 mg twice daily after 2 weeks. Dose can be further increased by 250 mg twice daily every 2 weeks. Maximum dose is 1500 mg twice daily. Children and adolescent below 16 years No data are available. ***Add-on therapy*** Adults (≥ 18 years) and adolescents (12 to 17 years) weighing > 50 kg Initial therapeutic dose is 500 mg twice daily. Dose can be started on the first day of treatment and can be increased up to 1500 twice daily. Dose changes can be made in 500 mg twice daily increases or decreases every 2 to 4 weeks. There is no experience with an administration of longer period than 4 days. Children aged 4 to 11 years and adolescent (12 to 17 years) weighing <50 kg Initial therapeutic dose is 10 mg/kg twice daily and can be increased up to 30 mg/kg twice daily. Doses changes should not exceed increases or decreases of 10 mg/kg twice daily every 2 weeks. The lowest effective dose should be used. Children weighing ≥50 kg Same dosing regimen as in adults. Children less than 4 years No data are available. ***Route of Administration*** Therapy can be initiated with either intravenous or oral administration. Conversion to or from oral to intravenous administration can be done directly without titration. The total daily dose and frequency of administration should be maintained. *Film-coated tablets and Oral solution* may be taken with or without food and the daily dose is administered in two equally divided doses. *Concentrate for solution for infusion* is for intravenous use only and the recommended dose must be diluted in at least 100 ml of a compatible diluent and administered intravenously as a 15-minute intravenous infusion. **Discontinuation** It is recommended to withdraw it gradually (e.g. in adults and adolescents weighing ≥50 kg: 500 mg decreases twice daily every 2 to 4 weeks; in infants ≥6 months, children and adolescents weighing <50 kg: dose decrease should not exceed 7 mg/kg twice daily every 2 weeks). **Contraindications** Hypersensitivity to the active substance or other pyrrolidone derivatives or any of the excipients. **Warnings and Precautions** ***Renal impairment*** Adjustment of the dose is recommended in elderly patients (≥65 years old) with compromised renal patients. In patients with severely impaired hepatic function, assessment of renal function is recommended before dose selection. ***Acute kidney injury*** Very rarely associated with acute kidney injury, with a time to onset ranging from a few days to several months. Blood cell counts Rare cases of decreased blood cells (neutropenia, agranulocytosis, leucopenia, thrombocytopenia and pancytopenia) have been described, generally at the beginning of the treatment. Complete blood cell counts are advised in patients experiencing important weekness, pyrexia, recurrent infections or coagulation disorders. ***Suicide*** Suicide, suicide attempt and suicidal ideation have been reported in patients treated with anti-epileptic agents (including levetiracetam). A meta-analysis of randomized placebo-controlled trials of anti-epileptics medicinal products has shown a small increased risk of suicidal thoughts and behavior. The mechanism of this risk is not known. Therefore patients should be monitored for signs of depression and/or suicidal ideation and behaviours and appropriate treatment should be considered. Patients (and caregivers of patients) should be advised to seek medical advice should any symptoms of depression and/or suicidal ideation or behavior emerge. ***Paediatric population*** The tablet formulation is not adapted for use in children under the age of 6 years and initial treatment in children weighing <25 kg. Available data in children did not suggest impact on growth and puberty. However, long term effects on learning, intelligence, growth, endocrine function, puberty and childbearing potential in children remain unknown. ***Excipients – oral solution*** Keppra 100 mg/ml oral solution contains methyl parahydroxybenzoate (E218) and propyl parahydroxybenzoate (E216) which may cause allergic reactions. It also contains maltitol liquid; patients with rare hereditary problems of fructose intolerance should not take this medicinal product. ***Excipients – concentrate for solution for infusion*** This medicinal product contains 2.5 mmol (or 57 mg) sodium per maximum single dose (0.8 mmol [or 19mg] per vial). To be taken into consideration by patients on a controlled sodium diet. **Interactions:** Methotrexate, laxatives, macrogol. **Pregnancy and Lactation** ***Women of child bearing potential*** Specialist advise should be given to women who are of childbearing potential. Treatment should be reviewed when a woman is planning to become pregnant. ***Pregnancy*** Can be used during pregnancy, if after careful assessment it is considered clinically needed. The lowest effective dose is recommended. ***Lactation*** Breast-feeding is not recommended. Benefit/risk of the treatment should be weighed considering the importance of breastfeeding. ***Fertility*** No impact on fertility was detected in animal studies. No clinical data are available. The potential risk for humans is unknown. **Ability to perform tasks that require judgement, motor or cognitive skills:** Levetiracetam has minor or moderate influence on the ability to drive and use machines. Caution is recommended in those patients when performing skilled tasks, e.g. driving vehicles or operating machinery. Patients are advised not to drive or use machines until it is established that their ability to perform such activities is not affected. **Adverse Reactions** Nasopharyngitis, anorexia, depression, hostility/aggression, anxiety, insomnia, nervousness/ irritability, somnolence, headache, convulsion, dizziness, tremor, balance disorder, lethargy, veretigo, cough, abdominal pain, diarrhoea, dyspepsia, nausea, vomiting, rash, asthenia/fatigue. **Overdosage** Somnolence, agitation, aggression, depressed level of consciousness, respiratory depression and coma were observed with overdoses. After an acute overdose, the stomach may be emptied by gastric lavage or by induction of emesis. There is no specific antidote for levetiracetam. Treatment of an overdose will be symptomatic and may include haemodialysis. The dialyser extraction efficiency is 60% for levetiracetam and 74% for the primary metabolite. **Please read the full prescribing information prior to administration.** Full prescribing information is available on request from GlaxoSmithKline Ltd, 23/F, Tower 6, The Gateway, 9 Canton Road, Tsimshatsui, Kowloon, Hong Kong. Abbreviated Prescribing Information based on PI version HK082018 (NCDS08/EMA20180601) for Tablets and Concentrate for solution for infusion and NCDS 08 for Oral Solution. For adverse events reporting, please call GlaxoSmithKline Limited at (852) 3189 8989 (Hong Kong) or send an email to us at HKAdverseEvent@gsk.com.

GlaxoSmithKline Limited 23 / F, Tower 6, The Gateway, 9 Canton Road, Tsimshatsui, Kowloon, Hong Kong. Tel: (852) 3189 8989 Fax: (852) 3189 8931 hk.gsk.com

PM-HK-LVTPSTR-210001 (09/2023) Date of preparation: 10/2021

Keppra levetiracetam

Pradaxa®
dabigatran etexilate

Praxbind®
idarucizumab

Closing the Circle

YOUR FORESIGHT THEIR FUTURES

Start with Pradaxa

CONFIDENCE[1-3]
with vetted global evidence

REVERSIBILITY[4]
in facing emergency situation

PROTECTION[5-6]
for extended medical condition

Abbreviated Prescribing Information PRADAXA® (aPI-PRA-20-21-23-V1)
Presentation: Dabigatran etexilate (Hard Capsules; 75mg, 110mg, 150mg). Indications: Prevention of stroke and systemic embolism in adult patients with non-valvular atrial fibrillation (NVAF), with one or more risk factors, such as prior stroke or transient ischemic attack (TIA); age ≥ 75 years; heart failure (NYHA Class ≥ II); diabetes mellitus; hypertension. **Dosage and administration:** The recommended daily dose of PRADAXA® is 300 mg taken as one 150 mg capsule twice daily. Therapy should be continued long term. For patients aged ≥ 80 years or patients who receive concomitant verapamil, the recommended daily dose of PRADAXA® is 220 mg taken as one 110 mg capsule twice daily. For the patients aged between 75-80 years, patients with moderate renal impairment, patients with gastritis, esophagitis or gastroesophageal reflux or other patients at increased risk of bleeding, the daily dose of PRADAXA® is 300 mg or 220 mg and should be selected based on an individual assessment of the thromboembolic risk and the risk of bleeding. Patients can stay on PRADAXA® while being cardioverted. Catheter ablation can be conducted in patients on 150 mg twice daily PRADAXA® treatment and PRADAXA® treatment does not need to be interrupted. Patients with NVAF who undergo a percutaneous coronary intervention (PCI) with stenting can be treated with PRADAXA® in combination with antiplatelets after haemostasis is achieved. **Contraindication:** Hypersensitivity to the active substance or to any of the excipients. Patients with severe renal impairment (CrCL < 30 mL/min). Active clinically significant bleeding. Lesion or condition, if considered a significant risk factor for major bleeding, which may include current or recent gastrointestinal ulceration, presence of malignant neoplasms at high risk of bleeding, recent brain or spinal injury, recent brain, spinal or ophthalmic surgery, recent intracranial haemorrhage, known or suspected oesophageal varices, arteriovenous malformations, vascular aneurysms or major intraspinal or intracerebral vascular abnormalities. Concomitant treatment with any other anticoagulants e.g. unfractionated heparin (UFH), low molecular weight heparins (enoxaparin, dalteparin etc), heparin derivatives (fondaparinux etc), oral anticoagulants (warfarin, rivaroxaban, apixaban etc) except under specific circumstances of switching anticoagulant therapy or when UFH is given at doses necessary to maintain an open central venous or arterial catheter. Hepatic impairment or liver disease expected to have any impact on survival. Concomitant treatment with the following strong P-gp inhibitors: systemic ketoconazole, cyclosporine, itraconazole, dronedarone and the fixed-dose combination glecaprevir/pibrentasvir. Prosthetic heart valves requiring anticoagulant treatment. **Special warnings and precautions:** PRADAXA® should be used with caution in conditions with an increased risk of bleeding or with concomitant use of medicinal products affecting haemostasis by inhibition of platelet aggregation. In clinical trials, PRADAXA® was associated with higher rates of major gastrointestinal (GI) bleeding. An increased risk was seen in the elderly (≥ 75 years) for the 150 mg twice daily dose regimen. The presence of lesions, conditions, procedures and/or pharmacological treatment (such as NSAIDs, antiplatelets, SSRIs and SNRIs), which significantly increase the risk of major bleeding requires a careful benefit-risk assessment. Close observation for signs of bleeding or anaemia is recommended throughout the treatment period, especially if risk factors are combined. Patients who develop acute renal failure must discontinue PRADAXA®. The use of fibrinolytic medicinal products for the treatment of acute ischemic stroke may be considered if the patient presents with a dTT, ECT or aPTT not exceeding the upper limit of normal (ULN) according to the local reference range. Patients on PRADAXA® who undergo surgery or invasive procedures are at increased risk for bleeding and may therefore require temporary discontinuation of PRADAXA®. PRADAXA® treatment should be resumed / started after the invasive procedure or surgical intervention as soon as possible provided the clinical situation allows and adequate haemostasis has been established. No treatment experience is available for patients with elevated liver enzymes > 2 ULN, and therefore the use of PRADAXA® is not recommended in this population. Direct acting oral anticoagulants (DOACs) including PRADAXA® are not recommended for patients with a history of thrombosis who are diagnosed with antiphospholipid syndrome, in particular for patients that are triple positive (for lupus anticoagulant, anticardiolipin antibodies, and anti-beta 2-glycoprotein I antibodies) Myocardial infarction. **Interactions:** Dabigatran etexilate is a substrate for the efflux transporter P-gp. Concomitant administration of P-gp inhibitors is expected to result in increased dabigatran plasma concentrations. There is no or only limited experience with the following treatments which may increase the risk of bleeding when used concomitantly with PRADAXA®: anticoagulants such as unfractionated heparin (UFH), low molecular weight heparins (LMWH), and heparin derivatives (fondaparinux, desirudin), thrombolytic medicinal products, and vitamin K antagonists, rivaroxaban or other oral anticoagulants, and antiplatelet aggregation medicinal products such as GPIIb/IIIa receptor antagonists, ticlopidine, prasugrel, ticagrelor, dextran, and sulfinpyrazone. **Adverse reactions:** Common: Anaemia, Epistaxis, Gastrointestinal haemorrhage, Abdominal pain, Diarrhoea, Dyspepsia, Nausea, Skin haemorrhage, Genitourological haemorrhage including haematuria. Uncommon: Haemoglobin decreased, Thrombocytopenia, Drug hypersensitivity, Rash, Pruritus, Intracranial haemorrhage, Haematoma, Haemorrhage, Haemoptysis, Rectal haemorrhage, Haemorrhoidal haemorrhage, Gastrointestinal ulcer including oesophageal ulcer, Gastroesophagitis, Gastroesophageal reflux disease, Vomiting, Dysphagia, Hepatic function abnormal/ Liver function Test abnormal, Alanine aminotransferase increased, Aspartate aminotransferase increased. Rare: Haematocrit decreased, Anaphylactic reaction, Angioedema, Urticaria, Hepatic enzyme increased, Hyperbilirubinaemia, Haemarthrosis, Injection site haemorrhage, Catheter site haemorrhage, Traumatic haemorrhage, Incision site haemorrhage. **Storage conditions:** Store in the original package in order to protect from moisture. Store below 30°C. Do not remove capsules from blister pack until just before use. **Note:** Before prescribing, please consult full prescribing information.

Abbreviated Prescribing Information PRAXBIND® (aPI PRAX 03 V1)
Presentation: Idarucizumab (solution for injection/infusion; vial of 2.5g/50mL). **Indications:** Praxbind is a specific reversal agent for dabigatran and is indicated in adult patients treated with Pradaxa (dabigatran etexilate) when rapid reversal of its anticoagulant effects is required: For emergency surgery/urgent procedures; In life threatening or uncontrolled bleeding. **Dosage and administration:** Restricted to hospital use only. The recommended dose of Praxbind is 5 g (2x2.5 g/50 mL), administered intravenously as two consecutive infusions over 5 to 10 minutes each or as a bolus injection. No dose adjustment is required in renally impaired patients, patients with hepatic injury, and in elderly patients aged 65 years and above. **Contraindication:** None. **Special warnings and precautions:** Idarucizumab binds specifically to dabigatran and reverses its anticoagulant effect. It will not reverse the effects of other anticoagulants. Praxbind treatment can be used in conjunction with standard supportive measures, which should be considered as medically appropriate. If an anaphylactic reaction or other serious allergic reaction occurs, administration of Praxbind should be discontinued immediately and appropriate therapy initiated. The recommended dose of Praxbind contains 4 g sorbitol as an excipient. Therefore, in patients with hereditary fructose intolerance the risk of treatment with Praxbind must be weighed against the potential benefit of such an emergency treatment. Patients being treated with dabigatran have underlying disease states that predispose them to thromboembolic events. Reversing dabigatran therapy exposes patients to the thrombotic risk of their underlying disease. To reduce this risk, resumption of anticoagulant therapy should be considered as soon as medically appropriate. Praxbind causes transient proteinuria, which is not indicative of renal damage. This medicinal product contains 50 mg sodium per dose, equivalent to 2.5% of the WHO recommended maximum daily intake of 2 g sodium for an adult. **Interactions:** No formal interaction studies with Praxbind and other medicinal products have been performed. Based on the pharmacokinetic properties and the high specificity in binding to dabigatran, clinically relevant interactions with other medicinal products are considered unlikely. **Adverse reactions:** No adverse reactions have been identified. **Storage conditions:** Store in a refrigerator (2°C - 8°C). Do not freeze. **Note:** Before prescribing, please consult full prescribing information.

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Boehringer Ingelheim

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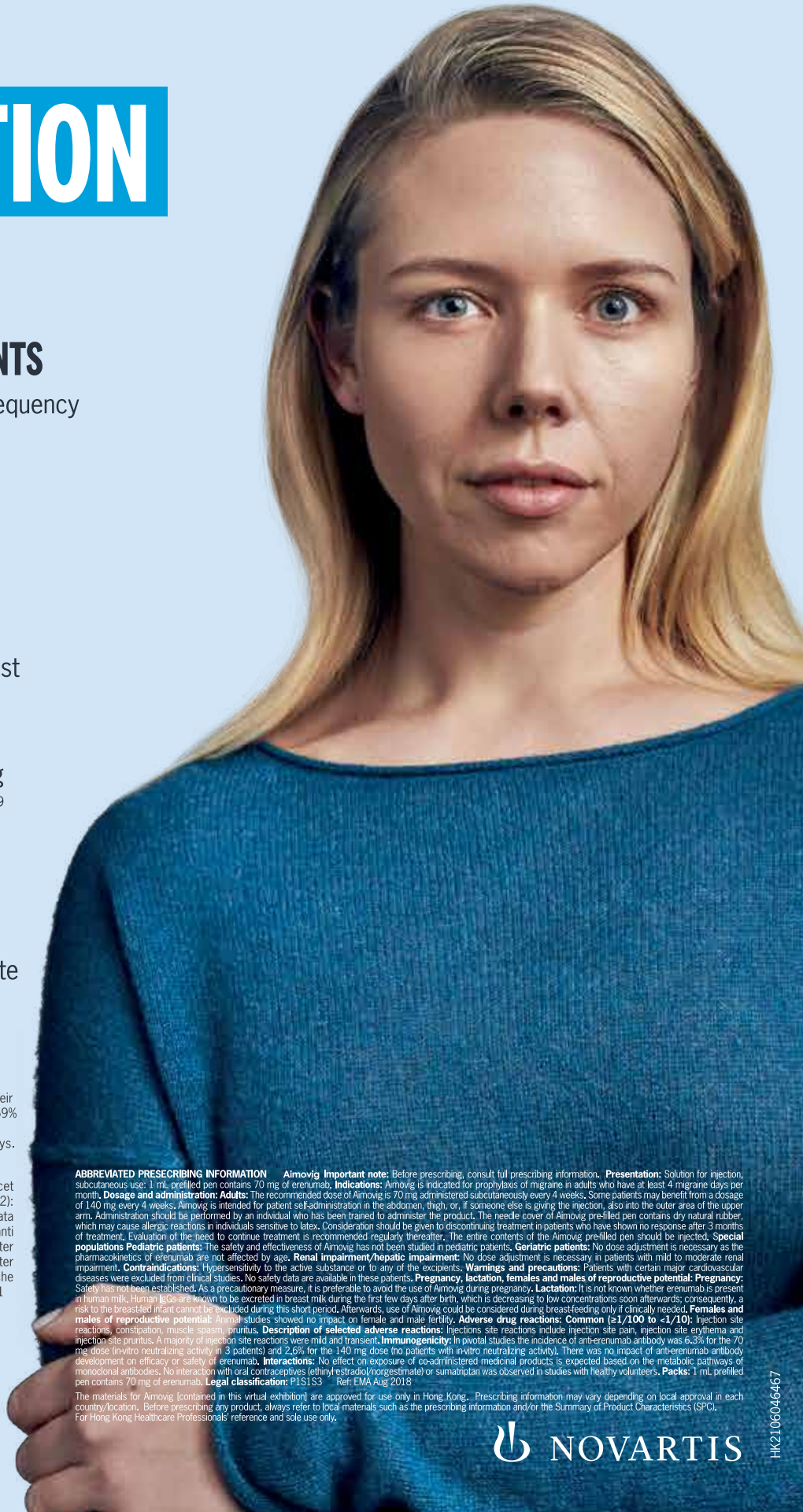
THIS IS HEAD-TO-HEAD DATA.
THIS IS PREVENTION



MORE DAYS WITHOUT MIGRAINE FOR MORE PATIENTS

- Significantly reduces migraine frequency and severity[1]
- Results may even improve over time among responders*[2-4]

MORE YEARS OF EVIDENCE THAN EVER BEFORE

- Evaluated to 5 years in the longest -running study of an anti-CGRP[4]
- Most experience in the real world,[5-7] and results in this setting may exceed those seen in trials[6-9]

MORE CONFIDENCE FOR SIMPLE MANAGEMENT

- Proven superiority over topiramate for patient adherence, efficacy and quality of life[1]

*Among responders continuing on Aimovig, the percentage who cut their MMDs in half increased from 46% at 3 months, to 65% at 1 year and 69% at 5 years.[2-4]

CGRP, Calcitonin Gene-Related Peptide, MMDs, Monthly Migraine Days.

REFERENCES **1.** HER-MES FIR. 2020; Data on File. **2.** Sun H et al. Lancet Neurol 2016; 15: 382-390. **3.** Ashina M et al. Neurology 2017; 89(12): 1237-1243. **4.** Ashina M et al. Cephalalgia 2020; 40(S1): 60-61. **5.** Data on Novartis. (Aimovig: Real World Evidence November 2020). **6.** Barbanti P et al. Neurol Sci 2019; 40(Suppl 1): 177-179. **7.** Jenkins B et al. Poster presented at IHC, 5-8 September 2019. **8.** Israel-Willner H et al. Poster presented at IHC, 5-8 September 2019. **9.** Ornello R et al. J Headache Pain 2020; 21(1): 32. Date of preparation: March 2021 103904-1

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NOVARTIS

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ONE OF THESE IS POSSIBLE WITH EMGALITY®

Reduction in monthly migraine headache day[1,2]

≥50% in up to **3 in 5** patients

≥75% in more than **1 in 3** patients

100% in up to **1 in 7** patients

Favorable safety profile

Less than **2.5%** of patients discontinued Emgality® due to treatment-related adverse events[4,5†]

Improvements in quality of life

Up to **80%** of patients saw their overall disability cut in half, based on MIDAS Total Score[3*]

Find out what else is possible for your patients with Emgality®

* The Migraine Disability Assessment (MIDAS) questionnaire provides numerical scores representing the number of days patients missed or lost productivity at work or school, as well as the missed days from family/social/leisure activities.[3]
† Pooled discontinuation rate from the double-blind treatment phase of the three Phase 3 studies (1.8% for 120 mg; 3.0% for 240 mg).[5]

References: 1. Stauffer VL, Dodick DW, Zhang Q, Carter JN, Ailani J, Conley RR. Evaluation of galcanezumab for the prevention of episodic migraine: the EVOLVE-1 randomized clinical trial. JAMA Neurol. 2018;75(9):1080–1088. **2.** Skljarevski V, Matharu M, Millen BA, Ossipov MH, Kim BK, Yang JY. Efficacy and safety of galcanezumab for the prevention of episodic migraine: results of the EVOLVE-2 phase 3 randomized controlled clinical trial. Cephalalgia. 2018;38(8):1442-1454. **3.** Ford JH, Ayer DW, Zhang Q, Carter JN, Skljarevski V, Aurora SK. Changes in patient functioning and disability: results from two phase 3 double-blind placebo-controlled clinical trials evaluating galcanezumab for episodic migraine prevention (EVOLVE-1 and EVOLVE-2). Poster presented at: 60th Annual Scientific Meeting of the American Headache Society; June 28–July 1, 2018; San Francisco, CA. **4.** Emgality®. Hong Kong Prescribing Information. 2020. **5.** Stauffer VL, Wang S, Bangs ME, Oakes TM, Carter JN, Aurora SK. Safety data from phase 3 clinical studies comparing galcanezumab and placebo in patients with episodic and chronic migraine. Poster presented at: 12th European Headache Federation Congress; September 28-30, 2018; Florence, Italy.

Abbreviated Prescribing Information
Emgality® 120 mg solution for injection in pre-filled pen
Presentation: Each pre-filled pen contains 120 mg of galcanezumab in 1 mL. **Indications:** Emgality® is indicated for the prophylaxis of migraine in adults who have at least 4 migraine days per month. **Dosage and administration:** The recommended dose is 120 mg galcanezumab injected subcutaneously once monthly, with a 240 mg loading dose as the initial dose. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and precautions:** Patients with recent acute cardiovascular events (including MI, unstable angina, CABG, stroke, DVT) and/or those deemed to be at serious cardiovascular risk were excluded from the galcanezumab clinical trials. No safety data are available in these patients. Serious hypersensitivity reactions including cases of anaphylaxis, angioedema and urticaria have been reported. If a serious hypersensitivity reaction occurs, administration of galcanezumab should be discontinued immediately and appropriate therapy initiated. This medicinal product contains less than 1 mmol sodium (23 mg) per 120 mg dose, i.e., is essentially "sodium-free". **Adverse reactions:** Very common: injection site pain, injection site reactions. Common: vertigo, constipation, pruritus, rash. Uncommon: urticaria. Rare: anaphylaxis, angioedema. **Drug interactions:** No drug interaction studies were conducted. No pharmacokinetic drug interactions are expected based on the characteristics of galcanezumab.
Full prescribing information is available upon request.

Emgality® (galcanezumab) injection

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