Association of Sri Lankan Neurologists

14 ANNUAL ACADEMIC SESSIONS

Improving neurology care through introspection

Programme and Abstracts

12 - 14 February 2021

Shangri-La Colombo | Sri Lanka

Association of Sri Lankan Neurologists

WFN
WORLD FEDERATION OF NEUROLOGY

14th Annual Academic Sessions

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Association of Sri Lankan Neurologists Council 2020/21

Standing from Left to Right: Dr Kumarangie Vithanage, Dr Kishara Gooneratne (Joint Editor), Dr Champika Gunawardhana, Dr Damith Liyanage, Dr Arjuna Fernando, Dr AT Alibhoy, Prof. Udaya Ranawaka, Dr Sudath Gunasekera, Dr Bimsara Senanayake, Dr Athula Dissanayake

Seated from Left to Right: Dr Saraji Wijesekara (Joint Secretary), Prof. Saman Gunatilake (Joint Editor), Dr Sunethra Senanayake, Dr Gamini Pathirana (President Elect), Dr JB Peiris (Patron), Dr Senaka Bandusena (President), Prof. Thashi Chang (Immediate Past President), Prof. Ranjanie Gamage, Dr MTM Riffsy, Dr T Thivakaran (Treasurer), Dr Manjula Caldera (Joint Secretary)

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Dr Damith Liyanage
Dr Kumarangie Vithanage

Message from the President

I warmly welcome all of you to the 14th Annual Academic Sessions of the ASN which is the highlight of the academic calendar. This year's sessions will be extra special as it will be the first time the membership is getting together physically after last year's meeting.

We have prepared an exciting academic program which will be delivered by seven eminent overseas speakers from UK, Israel, and Malaysia together with another six from Sri Lanka. We are greatly indebted to all the overseas speakers for their commitment and generosity in contributing to our sessions during these difficult times. Unlike in the past where we encouraged participation of registrars and junior medical officers, this year we have been compelled to limit physical participation at the sessions to the ASN members and neurology trainees only due to the restrictions on the number of attendees at the conference venue. Accordingly, the program has been planned with topics relevant to the membership. As in the past we will have the JB Peiris oration, oral and poster presentations of research papers and a quiz as part of the sessions. This year for the first time, an associate member of the ASN - the Young Neurologists' Forum winner, will be given the opportunity to present at the sessions.

At the pre-congress session all attendees will be given the opportunity to test their neurology knowledge. This session which is to be held virtually, will consist of 3 quizzes: an open quiz – Neurology Challenge, a quiz for the medical officers attached to neurology units throughout the country and another for the registrars. There will be attractive prizes, certificates, and medals on offer for the winners. The 3 quizzes will be conducted in a manner where important clinical points will be emphasized and discussed. These quizzes will complement the ASN webinar series where 30 key topics in neurology have been presented up to now. These webinars have become a popular CME activity for medical officers, students, and registrars. Going virtual, we have been able to reach out to all doctors working in far corners of the country interested in updating their neurology knowledge, who would otherwise not have had access to the academic programs conducted by the ASN. Furthermore, it is pleasing to note that there is an increasing number of doctors and students from overseas also following these webinars. In the run-up to the sessions, the webinars already conducted will be made available for viewing on-demand. Through these activities we have tried to generate further interest in neurology and offer an interactive learning experience for all.

In addition to the ASN webinars, the ASN organized several other academic programs during the year. These included a series of webinars covering 8 important paediatric neurology topics in collaboration with the Sri Lanka College of Paediatricians; a series of webinars titled "Midweek NeuroBites by ASN" covering advanced neurological and non-neurological topics of interest for the members; and another series of themed quizzes for the ASN members in preparation for the next Tournament of the Minds, World Congress of Neurology.

'Improving neurology care through introspection' was selected as the theme for 2020 and based on this it was decided that we try to focus inward to explore how we can further improve patient care as specialists in neurology. The break during the lock-down period brought on by COVID gave us time to reflect on our practices.

In addition to the various academic programs mentioned above we have initiated several other ambitious projects in keeping with this year's theme. These projects include the compilation of a neurology handbook for medical officers attached to neurology units, developing a neurology drug formulary for Sri Lanka, and commencing a dialogue on ASN guidelines and registries. Plans are also underway to conduct an Interfaculty Neuro Quiz for medical students in 2021. Hopefully, these will come to reality in the not-so-distant future.

Finally, I sincerely thank all resource persons, quiz masters, poster and oral free paper judges, the abstract evaluation committee and all members of the Council who contributed in numerous ways to organize the sessions, and of course all those who played an active role in other projects that have been initiated.

My special thanks to the two energetic Joint Secretaries, Dr Saraji Wijesekara and Dr Manjula Caldera, Treasurer Dr Thivakaran, President-elect Dr Gamini Pathirana, JB Peiris orator – Prof. Thashi Chang, our guiding light Prof. Saman Gunatilake, our Patron Dr JB Peiris and our dynamic and ever dependable Administrative Secretary Dr Himal Jayatilake who has been a driving force this year.

A grateful acknowledgement to pharmaceutical companies for their generosity in supporting the ASN activities throughout the year.

Let us enjoy the learning experience of the sessions as well as the fellowship and camaraderie to follow while adhering to the COVID safety guidelines.

Take care and stay safe.

Senaka Bandusena

Message from the Joint Secretaries

As the Joint Secretaries, it gives us immense pleasure to send this welcome message to all of you attending the 14th Annual Academic Sessions of the Association of Sri Lankan Neurologists.

The year 2020 was a challenging year for all of us due to the COVID 19 pandemic and the resultant health guidelines that were implemented globally, which restricted the conduct of physical meetings.

Hence, in order to disseminate knowledge a webinar series was conducted via the zoom platform, 'Ten pearls in neurology', targeting the medical officers in neurology units. The Midweek NeuroBites by ASN was specially targeted at the neurology fraternity where eminent neurologists and senior registrars in neurology gave exotic presentations of both rare and common conditions in neurology. Also, we had colleagues from other specialties sharing their experiences. A parallel webinar series which was conducted by the Paediatric Neurologists was also highly appreciated.

While some council meetings were held physically, we were compelled to have a few via the zoom platform. The quizzes conducted on different topics in neurology by our own members every month via the zoom platform was an enjoyable and challenging experience for all.

We would like to thank our President, Dr Senaka Bandusena for his excellent leadership and initiative of forming the ASN committees to work on different aspects inclusive of the Interfaculty Neurology Quiz, neurology handbook for medical officers, drug formulary in neurology, neurology guidelines, coffee-table books, and registries. The committees have started working on them and are showing progress in their work which will definitely lead to an improvement in quality of neurology services in Sri Lanka.

We wish to extend our sincere gratitude to the overseas speakers who accepted our invitation to join virtually, and the local speakers for sharing their experiences.

We wish to place on record our appreciation for our Patron, Dr JB Peiris and all Council members who actively participated in the yearly activities and encouraged us to achieve the objective of 'Improving neurology care through introspection'. We also wish to thank our event manager, Ms Nimalka Morahela, the staff of Shangri-La Colombo, generous sponsors and all others who helped us in many ways to make this event a memorable one.

Our special thanks to the very dynamic and enthusiastic administrative secretary Dr Himal Jayatilake for his tireless efforts in implementing all the activities in year 2020/21.

We look forward to your active participation and hope this event would provide opportunities to gain knowledge, fun and fellowship.

Dr Saraji Wijesekara **Dr Manjula Caldera**

SCIENTIFIC PROGRAMME

DAY 1

PRE-CONGRESS QUIZZES

Friday 12 February 2021

Virtual session broadcasted from National Epilepsy Centre, National Hospital of Sri Lanka

Pre-congress (virtual)

08.00 – 09.00	Registration and Log-in
09.00 – 11.00	NEUROLOGY CHALLENGE (Interactive Question Session - Open For All)
11.00 – 11.30	Tea
11.30 – 12.30	INTER-NEUROLOGY UNIT MEDICAL OFFICER'S QUIZ
12.30 – 13.00	Lunch
13.00 – 14.30	REGISTRAR'S NEUROLOGY QUIZ

DAY 2

ACADEMIC SESSIONS

Shangri-La Colombo

Saturday 13 February 2021

08.00 – 09.00	Registration and Log-in	
09.00 – 11.00	INAUGURATION CEREMONY	
	JB PEIRIS ORATION - Autoimmune encephalitis: A Sri Lankan experience	**Prof. Thashi Chang,** *SL*
11.00 – 11.30	Tea	
11.30 – 12.00	Parkinson's disease: update on non-motor aspects	**Prof. Shen-Yang Lim,** *Malaysia*
12.00 – 13.00	Oral Research Presentations	
13.00 – 14.00	Lunch	
14.00 – 14.30	Understanding Parkinson's: where has genetics got us?	**Prof. Nicholas Wood**, *UK*
14.30 – 15.00	Update on stroke thrombolysis and thrombectomy	**Dr Anthony Pereira,** *UK*
15.00 – 15.30	Emerging therapies in migraine	**Dr Manjit Matharu**, *UK*
15.30 – 16.00	The impact of epilepsy in the elderly: challenges and opportunities	**Prof. Ley Sander**, *UK*
16.00 – 16.30	Tea	

DAY 3

Shangri-La Colombo

Sunday 14 February 2021

08.00 – 09.00	Registration and Log-in	
09.00 – 09.30	Arterial ischaemic stroke: a paediatric perspective	**Dr Pyara Ratnayake**, *SL*
09.30 – 10.00	Principles of CSF diversion	**Dr Stravinsky Perera**, *SL*
10.00 – 10.30	Clinicopathological features of muscular dystrophy. Are we missing chronic autoimmune myopathy?	**Prof. Roshitha Waduge**, *SL*
10.30 – 11.00	Use of mobile apps in clinical neurology	**Dr Dilum Palliyeguruge**, *SL*
11.00 – 11.30	Tea	
11.30 – 12.00	Alzheimer's disease: past, present and future	**Prof. Amos Korczyn**, *Israel*
12.00 – 12.30	Mild cognitive impairment - is it a legitimate diagnosis?	**Prof. Amos Korczyn**, *Israel*
12.30 – 13.00	Recent advances in Paediatric MS and its mimics	**Dr Cheryl Hemingway**, *UK*
13.00 – 14.00	Lunch	
14.00 – 14.30	Young Neurologists' Forum Best Presentation	**Dr Hasini Munasinghe**, *SL*
14.30 – 16.00	ASN Quiz	**Prof. Saman Gunatilake,** *SL*
16.00 – 16.30	Tea	

FACULTY

Prof. Shen-Yang Lim

MBBS, MD, FRACP, FASc

Prof. Shen-Yang Lim is a Consultant Neurologist and Professor at the University of Malaya, Kuala Lumpur. He obtained his medical and research doctorate degrees from the University of Melbourne and was admitted to Fellowship of the Royal Australasian College of Physicians after completing advanced training in adult Internal Medicine and Neurology in Melbourne. Thereafter, he trained in Parkinson's & Movement Disorders at world-renowned centres in Melbourne and Toronto. He has published widely in the field of movement disorders with close to 100 peer-reviewed journal papers and book chapters. He serves on the International Parkinson & Movement Disorder Society (MDS) Committee for evidence-based medicine as well as the task force for palliative care and is an immediate past-Secretary of the MDS Asian-Oceanian section. He is also a medical advisor to the Malaysian Parkinson's Disease Association, and a founding member and current Chair of the Malaysian Movement Disorder Council. He was elected a Fellow of the Malaysian Academy of Sciences in 2017. Lim was part of the 4-member Malaysian team that won the Tournament of the Minds at the 19th World Congress of Neurology in 2009.

His research interests are in PD, Parkinson-plus syndromes, and orphan movement disorders.

Prof. Nicholas Wood

MB ChB, PhD, FRCP, FMedSci

Prof. Nicholas Wood qualified in medicine from the University of Birmingham. He went on to take a PhD in Cambridge and was promoted to personal chair in clinical neurology and neurogenetics in 2001. He established the Chair of Genetics at UCL in 2009. He was elected to the Fellowship of the Academy of Medical Science in 2004 and to senior investigator of the NIHR in 2008 (now emeritus). He is currently the UCL Professor of Genetics, a Consultant Neurologist and NIHR Programme Director for Neurosciences at UCLH.

His main research focus is the dissection of the aetiology and pathogenic mechanisms of movement disorders. These represent major health problems, and the burden of these conditions is high and increasing in our aging populations. His lab has worked across several disease areas to find genes causing neurological disease and has been responsible for identifying several genes that cause familial PD - PINK1, LRRK2, GCH1 and PLA2G6. They have also identified genes for ataxia (SCA11 and RFC1) and 5 dystonia genes.

He also works with international collaborators (IPDGC and GP2) to elucidate the commonly occurring genetic risk factors underlying PD.

Dr Anthony Pereira

MA (Cantab.), MD (Cantab.) FRCP (Lond.)

Dr Anthony Pereira trained in medicine at Cambridge and in neurology in London. He is a Consultant Neurologist and Stroke Physician at St. George's Hospital, London, UK. He is a member of the neurology and stroke SACs and helps run a large neurology and stroke training programme at St. George's.

Dr Manjit Matharu

BSc, MBChB, PhD, FRCP

Dr Manjit Matharu is an Associate Professor at UCL Queen Square Institute of Neurology and Honorary Consultant Neurologist at the National Hospital for Neurology and Neurosurgery, London, UK. He is the clinical and academic lead of the Headache and Facial Pain Group at the National Hospital for Neurology and Neurosurgery and UCL Queen Square Institute of Neurology, respectively. Dr Matharu is a member of the headache and pain subcommittee of the Association of British Neurologists. He was a member of the National Institute for Health and Clinical Excellence (NICE) guideline development group (GDG) for headache disorders.

Prof. Ley Sander

MD, PhD, FRCP, FEAN

Ley Sander is a Department Head at UCL Queen Square Institute of Neurology and Consultant Neurologist at the National Hospital for Neurology and Neurosurgery, London. He directs the Chalfont Centre for Epilepsy and is the R&D Director of SEIN in the Netherlands. Sander's main research interests are in epidemiology, genetics and co-morbidities of epilepsy, the management of chronic epilepsy and the delivery of epilepsy care in resource-poor settings. His research group was nominated as the highest-ranked epilepsy research group in the world in 2013 and has continued to maintain this top-ranking position until the present day (http://expertscape.com/ex/epilepsy). Sander has more than 900 publications to his name (h-index: 113). Sander has obtained many postgraduate awards including the Research Recognition Award for Clinical Sciences from the American Epilepsy Society, Innovation and Sustainability in Health Award, BUPA Foundation Epidemiology Award, European Gower's Prize, and the ILAE/IBE Ambassador for Epilepsy Award.

Prof. Amos Korczyn

MD, MSc

Professor Korczyn graduated from the Hebrew University–Hadassah Medical School in Jerusalem in 1966 (MD), where he also received an MSc degree in pharmacology (cum laude) in 1966. He trained in neurology at Beilinson Hospital and at the National Hospital for Nervous Diseases, Queen Square, London. He was the Chairman of the Department of Neurology at the Tel-Aviv Medical Center from 1981 until 2002, and the incumbent of the Sieratzki Chair of Neurology at Tel-Aviv University, 1995-2010. Professor Korczyn has a particular interest in neurodegenerative diseases. He has authored or co-authored over 700 articles in peer-reviewed journals, as well as many chapters in books. He edited several books and special issues in journals and is Regional Editor of the Journal of Alzheimer's Disease. He is or has been an Editorial Board member of 20 international journals, and organized several neurological conferences, mainly in the field of dementia, Parkinson's disease and other degenerative brain disorders, as well as CONy – the International Congress on Controversies in Neurology and has organized the Mental Dysfunction in Parkinson's disease congresses since 1993. Professor Korczyn served on advisory boards in several drug discovery programs.

Professor Korczyn is the Chairman of the Scientific Medical Board of the Israeli Alzheimer's disease association (EMDA), member of the SAB of Alzheimer Disease International (ADI), and has been the chairman of the WFN Research Committee for Clinical Neuropharmacology. Professor Korczyn is an honorary member of the neurological societies of Israel, Serbia, Poland, Russia, and Romania.

Dr Cheryl Hemingway

MBChB, BA (Hons), MMed, FCP, MRCP, FRCPCH, PhD

Dr Cheryl Hemingway has been a Consultant Paediatric Neurologist for 20 years. She has worked and trained in neurology at Red Cross Children's Hospital, South Africa, Johns Hopkins, USA and in the UK, and has been a full time Neurology Consultant at Great Ormond Street Hospital (GOSH) from 2005. Her PhD from Imperial College, London used ground-breaking techniques to explore the host immune response to brain inflammation and infection, and she has used the expertise gained from this to provide specialised clinical care to children with rare neuro-inflammatory disorders, as well as in providing care for children with many other neurological problems. She leads the GOSH Demyelinating Disease Service, the largest clinic in the UK for children with Multiple Sclerosis and other rare demyelinating disorders, and is also research active, with a number of recent important publications. She is on the steering committee for the International Paediatric MS Study group and sits on a number of advisory panels for MS trial design.

Dr Pyara Ratnayake

MBBS, MD (Paed)

Paediatric Neurologist working at the Lady Ridgeway Hospital for Children in Sri Lanka. She trained at the Children's Hospital at Westmead, Australia. She was awarded the George Gregan Epilepsy Fellowship. Her special interests are epilepsy, neurorehabilitation and neurometabolic disorders. She is a member of the national task force for community-based rehabilitation. She established ketogenic diet and visual rehabilitation in Sri Lanka.

Dr Stravinsky Perera

MBBS, MRCSEd, MD (Surgery)

Dr Stravinsky Perera is a Consultant Neurosurgeon at National Hospital of Sri Lanka. He obtained his MBBS from Faculty of Medicine University of Kelaniya, Sri Lanka with second class honours and MD (Surgery) in 2009, MRCSEd in 2011 and was board certified in Neurosurgery in 2013. He is the present Joint Secretary of the Neurosurgeons Association of Sri Lanka and is a Member Specialty Board of Neurosurgery PGIM and College of Surgeons of Edinburgh. He Initiated the Sri Lankan participation in the STICH II trial, which was coordinated by the Neurosurgical unit Newcastle, UK, and successfully recruited the highest number of patients within one year, for which an award was presented to Sri Lanka. He has special interest in skull base, spinal and vascular neurosurgery.

Prof. Roshitha Waduge

MD in Histopathology

Roshitha Nilmini Waduge graduated with MBBS from North Colombo Medical College with second class honours in 1992 and obtained her MD in Histopathology from the University of Colombo in 2002. She had her post MD overseas training at University of Sydney, Australia where she underwent a short training in interpretation of muscle biopsies.

She joined the Department of Pathology, Faculty of Medicine, University of Peradeniya as a senior lecturer in 2005 and has worked as an Honorary Consultant to the Teaching Hospital, Peradeniya since then. On her initiative, she developed a muscle biopsy service at the department under the guidance of her MD trainer Prof. NVI Ratnatunga. She was later promoted to Associate Professor in 2015 and Professor in Pathology in 2020.

In recognition of this service and the potential to expand the muscle biopsy service, Peradeniya was selected as the focal point for the establishment of the AOMC Sri Lankan chapter. She won the presidential award for scientific publications in 2014. She was a trainer to the PGIM, Sri Lanka and has trained postgraduate trainees in MD pathology, PhD and MSc. She is a member of many prestigious academic societies and has held

several administrative positions.

Her future interests are to develop the muscle biopsy laboratory to a state-of-the-art reference center.

Dr Dilum Palliyeguruge

MD, MRCP(UK), MRCP Neuro(UK)

Dr Palliyeguruge is currently attached to the Teaching Hospital Kurunegala and has been a board-certified Neurologist since 2011. During this decade as a Neurologist, he has served diligently in many hospitals across the island including Kandy, Batticaloa, Anuradhapura and Ratnapura. He obtained his MBBS in 2002 from the University of Ruhuna with second class honours. Dr Palliyeguruge received his MD from the University of Colombo in 2008 after completing his postgraduate training at the National Hospital of Colombo. He also received the S. E. Senevirathne Award at the Sri Lanka Medical Association Annual Academic Sessions 2007 for his research on "Efficacy of fresh frozen plasma (FFP) on thrombocytopenia in adults with Dengue Haemorrhagic Fever". His overseas training in neurology was at the Queens Hospital, East London during 2011 with further training at the Hull Royal Infirmary, Yorkshire in 2012. He completed his MRCP in 2011 and also qualified in MRCP Neurology Specialty in 2012.

Dr Palliyeguruge has special interests in treating headache disorders and epilepsy.

Dr Hasini Munasinghe

MBBS, MD

Dr Hasini Munasinghe is currently a Specialty Trainee in Clinical Neurophysiology at the National Epilepsy Center, National Hospital of Sri Lanka. She graduated with second class honours from University of Kelaniya in 2012 and obtained her MD Medicine in 2018. She is a member of the Association of Sri Lankan Neurologists, Sri Lanka Committee for Treatment and Research in Multiple Sclerosis and Related Disorders and the International Parkinson and Movement Disorder Society. She is actively involved in several research projects in neurophysiology and has several publications in peer-reviewed international journals.

Her research interests are Guillain-Barré Syndrome, epilepsy surgery, intraoperative neurophysiological monitoring and neuromodulatory devices.

Prof. Saman Gunatilake

MD, FRCP, Hon FRACP, FCCP

Professor Gunatilake was appointed the first Neurologist to Southern Sri Lanka and established the Neurology Unit in Galle. In 1994 he joined the University of Kelaniya and Colombo North Teaching Hospital as a Senior Lecturer and was promoted to Associate Professor in 1997. In 2007 he was appointed Professor of Medicine at the University of Sri Jayewardenepura and was the Head of the Department of Medicine. He retired from his post in December 2017.

His research interests have been in strokes in Sri Lanka, epilepsy, Alzheimer's disease and latterly, the problem of neurophobia, and teaching neurology. He has over 70 publications in national and international journals including the Ceylon Medical Journal, The Lancet, BMJ, Epilepsy Research, Seizures, Practical Neurology, BMC Medical Education and the JNNP and over 80 abstracts of scientific presentations made at national and international meetings.

He is a Past President of the Ceylon College of Physicians, the Epilepsy Association of Sri Lanka and the Association of Sri Lankan Neurologists. He is an overseas member of the Association of British Neurologists and a member of the World Stroke Organization. He was a co-editor of the Journal of the Ceylon College of Physicians, Chief Editor of the Sri Lanka Journal of Neurology and has been in the editorial board of the Ceylon Medical Journal. He chaired the Research Committee in the Faculty of Medical Sciences, USJP from 2010 to 2014. He is a council member of the Association of Sri Lankan Neurologists and national delegate of the Asian and Oceanian Association of Neurology.

QUIZ MASTERS (PRE-CONGRESS)

Dr Kishara Gooneratne

MD(Col), MRCP(UK)

Dr Kishara Gooneratne is a Board-Certified Neurologist with a special interest in epilepsy. Dr Gooneratne underwent overseas training at the National Hospital of Neurology and Neurosurgery, Queen Square, London and Oxford University, gaining valuable insights in tertiary care of epilepsy patients and patients with movement disorders. He is currently Consultant Neurologist at DGH Hambantota and is also part of the team providing tertiary care services in epilepsy at the National Hospital of Sri Lanka, which includes the surgical programme for epilepsy patients. He was former Joint Secretary of the CCP in 2020. He is also the Co-Editor of the Association of Sri Lankan Neurologists, Editor of the Epilepsy Association of Sri Lanka, and the Assistant Secretary of the Sri Lanka Clinical Trials Registry. He is a member of the editorial committee of the Journal of the Ceylon College of Physicians. He serves as a member of the Board of Study in Geriatric Medicine. He is a Council Member of the Sri Lanka Medical Association. He has published close to 40 articles in national and international medical journals (H index – 10, Google Scholar).

Dr Kumarangie Vithanage

MBBS, MD, MRCP

Dr Kumarangie Vithanage is a Senior Lecturer and Consultant Neurologist at the Faculty of Medicine, University of Colombo. She graduated from the University of Colombo in 2007 and was board certified as a specialist in Neurology in 2017.

Dr Shanika Nandasiri

MBBS, MD

Dr Shanika Nandasiri obtained her MD Medicine in 2016 and completed her overseas training in the Department of Neurology, Royal Melbourne Hospital, Australia. She is currently working as an Acting Consultant Neurologist at the Colombo North Teaching Hospital, Ragama. Her areas of special interest are motor neurone disease, epilepsy, and stroke rehabilitation.

Dr Manjula Caldera

MBBS, MD, MRCP

Dr Manjula Caldera obtained his primary medical qualification (MBBS) from the Faculty of Medicine, University of Colombo in 2004 and MD (Medicine) from the same university in 2010. He pursued a career in Neurology as a Senior Registrar at the Institute of Neurology, National Hospital of Sri Lanka, Colombo, and followed a neurology fellowship in King's College Hospital, London. Since 2016 to date, he serves as a Consultant Neurologist at the Teaching Hospital Anuradhapura. He has a special interest in cognitive neurology. He is currently the Secretary of the Association of Sri Lankan Neurologists and Assistant Secretary of the National Stroke Association of Sri Lanka. He was a member of the ASN team which became runners-up at the World Congress of Neurology "Tournament of Minds" in 2019, Dubai.

Dr AT Alibhoy

MBBS, MD, MRCP(UK), FCCP, FRCP(Lon)

Dr AT Alibhoy is a Consultant Neurologist in Colombo, Sri Lanka. He graduated from the University of Colombo in 1992 and was Board Certified as a Specialist in Neurology in 2002. He received his overseas training at the Royal London Hospital & St. Bartholomew's Hospital in London. He was awarded Fellowship of the Royal College of Physicians, London and Fellowship of the Ceylon College of Physicians in 2016.

He was the President of the Association of Sri Lankan Neurologists in 2016. He has special clinical interest in Movement disorders and Neuro-ophthalmology.

DR JB PEIRIS ORATION

Dr JB Peiris Orator 2021

Prof. Thashi Chang

MBBS, MD, MRCP(UK), MRCP(UK)(Neurol), DPhil(Oxon), FCCP, FRCP(Lond)

Thashi Chang is Professor in Neurology in the Department of Clinical Medicine of the University of Colombo and Honorary Consultant Neurologist at the Professorial Unit in Medicine at the National Hospital of Sri Lanka.

He was educated at DS Senanayake Vidyalaya, Colombo. He was the school Head Prefect from 1987 to 1988 and captained the First Fifteen Rugby team. He graduated from the Colombo Medical Faculty with first class honours with Distinctions in ten of the thirteen subjects. He was awarded 20 medals and awards including Gold medals in Medicine, Surgery, Paediatrics, Pharmacology, Parasitology, Physiology and Final MBBS. He was elected the 'Student of the Year' of the University of Colombo in 1996. He represented both the Medical Faculty and University Rugby teams and was awarded University Colours for Rugby.

He obtained his MD(Medicine) in 2002, MRCP(UK) in 2004 and MRCP(UK)(Neurology) in 2016. He was Board Certified as a Specialist in Neurology in 2005. He was elected as a Fellow of the Ceylon College of Physicians and a Fellow of the Royal College of Physicians of London in 2013. He was awarded the Commonwealth Scholarship in 2003 to read for the degree of Doctor of Philosophy in Clinical Neurology at the University of Oxford, UK which he completed in 2007. During this time, he also trained in Clinical Neurology at the Radcliffe Infirmary and the John Radcliffe Hospitals NHS Trust in Oxford. He served as the Clinical Lecturer in Neurology in the University of Oxford for a brief period and has been an Academic Visitor of the University of Oxford.

Professor Chang has the distinction of being appointed as the first Professor in Neurology of the University of Colombo in 2016. He has been awarded several President's Awards for research, the award of Excellence in Research of the University of Colombo and the Graduate Scholarship of St Hugh's College of the University of Oxford. He has delivered the Ceylon College of Physicians' EV Peiris Memorial Oration in 2011 and the SLMA Oration in 2015.

He has served as the Secretary of the Specialty Board in Neurology and the Board of Study in Sports Medicine of the Postgraduate Institute of Medicine coordinating the development of curricula and prospectuses in postgraduate training in Neurology, Neurophysiology and Medical Rehabilitation.

He is a founder member of the Sri Lanka Committee for Treatment and Research in Multiple Sclerosis and Related Disorders, the Movement Disorder Society of Sri Lanka and the National Stroke Association of Sri Lanka. He has been a Council Member, Co-Editor and Joint Secretary of the Association of Sri Lankan Neurologists and is its immediate past-President.

Dr JB Peiris Oration 2021

Autoimmune encephalitis: A Sri Lankan experience

Prof. Thashi Chang

MBBS, MD, MRCP(UK), MRCP(UK)(Neurol), DPhil(Oxon), FCCP, FRCP(Lond)
Professor in Neurology
Department of Clinical Medicine, University of Colombo

Among an estimated annual incidence of approximately 5 to 8 cases of encephalitis per 100,000 persons, autoimmune encephalitis (AIE) has emerged as the third most common cause after infections, and acute disseminated encephalomyelitis. More importantly, AIE associated with autoantibodies directed against neuronal cell surface/synaptic proteins have emerged as the most treatment responsive encephalitis with the greatest potential for complete recovery. Among the antibody-mediated encephalitides, NMDAR-antibody-encephalitis is the most common followed by limbic encephalitis mediated mostly by antibodies directed against LGI1 and CASPR2. Although rare, a number of other AIE associated with antibodies to AMPAR, GABABR, mGluR5, D2R, DPPX, GABAAR and Neurexin-3α have been recognised. These forms of AIE are distinguished from paraneoplastic encephalitides, which are associated with antibodies directed against intracellular proteins, and are poorly responsive to antibody-depleting immunotherapy.

NMDAR-antibody-encephalitis is characterised by a female predominance (4:1), younger onset (median age 21 years), associated tumours (ovarian teratoma) and a multi-phenomenological syndrome that evolves over time with seizures, abnormal movements, insomnia, and irritability more frequent in children, and psychosis, abnormal behaviour, dysautonomia and coma more frequent in adults. By contrast, limbic encephalitis is characterised by an older age of onset (>45 years), amnesia, confusion, seizures, hyponatraemia, increased signal of medial temporal lobes on magnetic resonance imaging and variable association with tumours determined by the associated antibody. The recent description of diagnostic criteria and the recognition of specific clinical syndromes have enabled the diagnosis of AIE even in settings with limited access to diagnostic antibody assays.

The key points that will be discussed in the oration are:

- History and nosology of AIE
- Pathogenesis of antibodies against neuronal cell surface/synaptic proteins
- Clinical approach to the diagnosis of AIE
- AIE in Sri Lanka
    - First reports
    - Clinical characteristics
    - Prevalence of autoantibodies
    - Comparison of antibody-positive with antibody-negative AIE
    - Typical and atypical presentations of AIE among children
    - Autoimmune versus infectious aetiologies of encephalitis
    - Immunotherapy and outcomes
    - Establishment of cell-based assays at the Medical Research Institute, Colombo

PRE-CONGRESS QUIZZES

DAY 1

Friday 12 February 2021

QUIZ 1 – 09:00

NEUROLOGY CHALLENGE

Quiz masters

Dr Kishara Gooneratne, Dr Kumarangie Vithanage and Dr Shanika Nandasiri

This will be an open quiz where anyone can take part and check their neurology knowledge. All are encouraged to participate.

QUIZ 2 – 11:30

INTER-NEUROLOGY UNIT MEDICAL OFFICER'S QUIZ

Quiz master

Dr Manjula Caldera

This team quiz is restricted to Medical Officers attached to neurology units. Each team will consist of 4 members. Others can follow the event virtually.

The objective of the quiz is to improve the knowledge in neurology of junior doctors who are the real workforce in wards as well as outpatient clinics. While being an educational activity, this will also be a fun event.

QUIZ 3 – 13:00

REGISTRAR'S NEUROLOGY QUIZ

Quiz master

Dr AT Alibhoy

This quiz is restricted to Medical Registrars and Senior Registrars (Non-Neurology), pitched at a more advanced competency level. Participants will compete individually.

ABSTRACTS OF PLENARY LECTURES

DAY 2

Saturday 13 February 2021

11:30 Parkinson's Disease: Update on non-motor aspects

Prof. Shen-Yang Lim

Non-motor features are an integral part of the "Parkinson's complex" and encompass a broad array of clinical and subclinical elements in the neuropsychiatric, sleep, autonomic, and sensory domains. Proper understanding of these non-motor aspects of the disease is needed for more holistic and patient-centred care, and for advancing PD research.

In this talk, I will provide an update on the non-motor aspects of PD, with a focus on important observational studies and key clinical trials.

14:00 Understanding Parkinson's: where has genetics got us?

Prof. Nicholas Wood

The technological progress in genetics has been extremely rapid. We now have available unprecedented depth and breadth of data to understand human disease. Here, I will address some of the major breakthroughs and challenges facing neurology; How do we use these approaches to understand neurological disease? What are the next challenges and how will we meet them, how best to translate genetic discoveries to clinical practice, and can we use genetics to drive therapeutic innovations?

14:30 Update on stroke thrombolysis and thrombectomy

Dr Anthony Pereira

This is a very exciting time in the management of acute stroke. New technological advances are providing insights into early stroke pathophysiology and well-targeted trials have demonstrated that they can assist the delivery of safe and effective acute stroke treatment beyond the traditional narrow timeframes. This lecture will provide an update on the evidence for the use of mechanical thrombectomy and thrombolysis in the acute management of stroke as well as guidance on how this may be implemented in routine clinical practice.

15:00 Emerging therapies in migraine

Dr Manjit Matharu

Globally headaches are second only to back pain as a cause of years lived with disability. The acute and preventive treatment available for migraine until recently had limited efficacy and were often poorly tolerated in patients. In the last few decades, considerable advances in our understanding of migraine and its pathophysiology have paved the way for the development of targeted treatment options. Calcitonin gene-related peptide (CGRP) plays an integral role in the neurobiology of migraine, and new classes of drugs that target the CGRP pathway have included gepants and CGRP pathway monoclonal antibodies. Serotonin 5-HT1F receptor agonists-namely ditans-have also been developed to treat acute migraine. Lastly, non-invasive and invasive neuromodulation offers another treatment option for migraine patients who prefer treatments that have fewer side effects and are well tolerated. This lecture will address the emerging treatment options for migraine.

15:30 The impact of epilepsy in the elderly: challenges and opportunities

Prof. Ley Sander

Globally, as populations age, there will be challenges and opportunities to deliver optimal health care to older people with Epilepsy. Epilepsy, a condition characterised by spontaneous recurrent seizures, is very common in people over the age of 65 and has received comparatively little attention in this age group. There are several issues in ascertaining the underlying causes of epilepsy in older people, particularly difficulties in establishing a diagnosis of epilepsy in this population. The choice of appropriate antiseizure medications is also an issue, particularly in terms of availability and drug affordability. Cognitive, psychological, and psychosocial comorbidities and the effect that epilepsy might have on an older person's broader social or care network is another challenge, particularly in middle-income and low-income countries. The need for clinical trials to be more inclusive of older people with epilepsy, to help informed therapeutic decision making, and discuss whether measures to improve vascular risk factors might be an essential strategy to reduce the probability of developing epilepsy.

Day 3

Sunday 14 February 2021

09:00 Arterial ischaemic stroke: a paediatric perspective

Dr Pyara Ratnayake

Arterial ischaemic stroke in childhood, although not common, is a condition with a high percentage of mortality and morbidity. The aetiological variability is also greater than that seen in adults. Management principles are the same as in older people although clinicians find decision making regarding exact interventions and their optimal timing difficult due to the lack of reliable data. This presentation would be case based and will discuss varied approaches to management as decided by precise aetiology.

09:30 Principles and methods in CSF diversion

Dr Stravinsky Perera

CSF diversion is used to treat acute and chronic hydrocephalus, be it diverting the CSF into an externalized bag, another cavity of the body or even bypassing the obstruction. External ventricular drainage, shunts and endoscopic ventriculostomy are the methods used in diverting the CSF. Indications and complications for each method is hereby discussed.

10:00 Clinicopathological features of muscular dystrophy in Sri Lanka. Are we missing chronic autoimmune inflammatory myopathy?

Prof. Roshitha Waduge

Muscle diseases are not an uncommon problem in children and adults. Accurate diagnosis of the disease is important in the proper management of patients. An important diagnostic tool is the muscle biopsy, where the pathologist examines the muscle tissue using routine and special techniques. This service was not readily available in the country. However, the Department of Histopathology at the Teaching Hospital, Peradeniya has successfully developed a specialized muscle biopsy service. We have already given our services regarding the interpretation of muscle biopsies to many hospitals from the year 2010 onwards. There were several audits done on these biopsies with interesting outcomes in terms of demography and pathology.

Muscular dystrophy is a commonly inherited group of muscle diseases that progressively weakens the muscular skeletal system and hampers locomotion. The disease has more than one form with different inheritance patterns. For instance, the X-linked recessive

forms of muscular dystrophies such as Duchenne Muscular Dystrophy (DMD) are caused by mutations in the dystrophin gene on chromosome Xp21.2.

Due to the limited number of patients and lack of demographic and clinicopathological data regarding muscular dystrophy, many challenges exist regarding the establishment of the diagnosis, combating the progression and complications of the disease. Furthermore, the presence of overlapping clinical features and biochemical abnormalities in different types of muscular dystrophies and chronic autoimmune inflammatory myopathy can result in misdiagnosis. This hampers the process of halting the disease progression, timely interventions, and genetic counselling.

For example, muscular dystrophy and autoimmune inflammatory myopathy can present with similar clinicopathological profiles such as proximal muscle weakness, similar histological features such as atrophy, fibre necrosis, regenerating fibres, endomyseal and perimyseal inflammation and similar electromyographic and biochemical findings such as elevated creatine phosphokinase levels. Sadly, wrong genetic advice will be given to a carrier if mistakenly diagnosed. This problem of overlapping clinical and routine histological features of these two conditions can be solved by accurate diagnosis of a muscle biopsy using immunohistochemistry which helps to visualize the presence or absence of sarcolemmal labelling of dystrophin and other associated proteins in muscular dystrophy. MHC labelling of sarcolemma and C5b-9 complement staining of capillaries will help differentiate between autoimmune inflammatory myopathy from dystrophinopathy, especially when there is minimal endomyseal and perivascular inflammatory infiltrate.

However, due to the constraints faced by pathologists in having a continuous supply of immunostains and the costs incurred, one has to depend on the experience of handling similar cases in the context of history, clinical features and the demographic variation of the said diseases following an audit or research.

10:30 Use of mobile apps in clinical neurology

Dr Dilum Palliyeguruge

With the evolution of mobile technology into a widespread global industry and invention of smartphone devices and wearables, a majority of us have started using these devices to facilitate our everyday tasks. Using smartphone apps to resolve otherwise complicated activities have not only become common but also an essential function of our lives. The health related smartphone apps segment crossed the 12 billion mark in 2018 and out of these there are quite a few neurology-related smartphone apps available to facilitate clinical neurology. More than 50% of clinicians have embarked on using smartphone apps for a number of reasons. Saving time and paper and improving precision are a few reasons contributing to the popularity of mobile health apps. Finding solutions for simple as well as complicated clinical problems have become smooth and streamlined with numerous databases available through apps in your smart handheld device which can also be used to bridge any gaps in knowledge. This presentation focuses on identifying areas which a smartphone app can be helpful in day-to-day neurology practice and also to demonstrate the use of recommended top-rated apps under four different segments including enhancing efficiency, accuracy, communication, and keeping up-to-date with the latest knowledge.

11:30 Alzheimer's disease: past, present and future

Prof. Emeritus Amos Korczyn

The term Alzheimer's disease (AD) has been initially used to describe the occurrence of dementia in presenile, otherwise healthy individuals. Over the years the term has been borrowed to also include elderly individuals. Early onset AD (EOAD) is frequently familial, transmitted as a dominantly inherited mutation, whereas the late onset form (LOAD) is sporadic and associated with a large number of predisposing factors. Thus, these are different entities. While EOAD is a disease, LOAD is actually a syndrome, with a multitude of causative factors and a heterogeneous phenomenology. The role of beta amyloid in the pathogenesis of LOAD is still unknown.

12:00 Mild cognitive impairment - is it a legitimate diagnosis?

Prof. Emeritus Amos Korczyn

Mild Cognitive Impairment (MCI) is an artificial term, describing the period of initial mental decline preceding the occurrence of dementia. As such, it does not have an accurate time of onset nor termination. Its significance is unclear since it does not have precise inclusion or exclusion criteria. It does not have specific underlying pathology and its evolution is unpredictable. The use of the term causes unnecessary stress and therefore should not be employed as a diagnosis.

12:30 Recent advances in paediatric MS and its mimics

Dr Cheryl Hemingway

My 20-minute talk will review the spectrum of acute demyelinating syndromes in paediatrics, highlight the importance of accurate and early recognition of the different clinical syndromes and discuss the arguments for early aggressive treatment.

Using clinical case vignettes, I will discuss the key features of diagnosis, the application of the McDonald 2017 to paediatrics and the associated long-term consequences of MS. I will also review the range of different phenotypes associated with MOG antibody associated demyelination and briefly review treatment.

14:00 Epilepsy surgery at the National Epilepsy Center: where we are and the future

Dr Hasini Munasinghe

An epilepsy center is a specialized institution where patients with epilepsy are diagnosed and treated through a comprehensive team approach. The level of epilepsy care provided by epilepsy centers vary according to the expertise and facilities available.

The National Epilepsy Center of Sri Lanka which was founded in 2017 at the National Hospital of Sri Lanka, is the center of excellence for epilepsy care for patients across the country. As it is the prime center for the management of epilepsy, it is equipped with many modern facilities required for care of patients with refractory epilepsy. At the National Epilepsy Center, patients with pharmaco-resistant epilepsy are being evaluated by an expert multidisciplinary team on a regular basis. The selected patients undergo either resective or palliative surgeries. Poor surgical candidates are offered other treatment options such as ketogenic diet and immunotherapy. Out of all epilepsy surgeries, 90% are lesionectomies while the rest of them are disconnection procedures. According to current observations, the post-operative unexpected complication rate is minimal. The expertise in intraoperative neurophysiological monitoring guides adequate and safe surgical resections.

Neuromodulatory therapies are the other treatment options for patients with drug-resistant epilepsy. Even though the neurosurgical team has the required knowledge and skills in implanting these devices, these are sparsely available owing to their high cost, thus having limited usage in Sri Lanka.

While the main aim of this presentation is to improve awareness among health care personnel on different treatment modalities available for pharmaco-resistant epilepsy, it is hoped that some of these techniques still not available including neuromodulatory therapies will be made available for the benefit of Sri Lankan patients in the near future.

ABSTRACTS OF ORAL & POSTER PRESENTATIONS

ABSTRACTS OF ORAL PRESENTATIONS

OP-01

Bladder dysfunction in multiple sclerosis: A preliminary data from an ongoing hospital based study

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Introduction: Bladder dysfunction in multiple sclerosis (MS) is common and is often overlooked. This could be due to dysfunction in bladder storage, voiding or both. The location of demyelinating plaques is an important determinant of bladder dysfunction.

Methodology: Patients with multiple sclerosis diagnosed according to 2017 McDonald criteria who attended the MS clinic at the National Hospital of Sri Lanka, with bladder symptoms were evaluated. Details on bladder symptoms, disease course and magnetic resonance imaging were obtained. The Actionable questionnaire on quality of life in MS patients with bladder dysfunction was administered. An USS KUB with pre- and post-void bladder volumes and free flow uroflowmetry were performed. Treatment was commenced according to the type of bladder dysfunction and the same questionnaire and QOL assessment was repeated after 10 weeks. Antimuscarinics and alpha blockers were used in the treatment (Tolterodine, Tamsulosin)

Results: Eleven diagnosed MS patients with urinary symptoms were evaluated out of 100 MS patients who were screened. Three were (27%) males and 8 (72%) were females. Age distribution was from 23 to 57 years (SD = 11.8). The mean duration of disease until the onset of bladder symptoms was 10 years and the average EDSS of the group was 4 (SD=2.3) Three patients had hypotonic non-compliant bladder without outlet obstruction, five had hypotonic non-compliant bladder with outlet obstruction, two had normal detrusor with sensory urgency and one had bladder outlet obstruction only. Majority of the patients had bladders with no risk to upper tracts. The mean actionable symptom screening scores pre- and post-treatment were 5.1 and 2 respectively. 91% patients had demyelinating plaques in the cervical and dorsal spine on MRI.

Conclusion: Bladder dysfunction in MS should be actively evaluated in order to determine the type, severity and complications. Both storage and voiding dysfunction were observed in combination where storage dysfunction was predominant. Targeted treatment according to the type of bladder dysfunction significantly improved the symptoms and the quality of life.

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OP: 02

Effects of long-term meditation on cognition and electroencephalography derived brain dynamics: a cross-sectional comparative study

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Objective: To compare the dynamics of EEG wave patterns and measures of cognitive domains of long-term meditators (LTM) with non-meditators (NM).

Method: Ten experienced LTM (regular long-term meditational practice of over 3 years) were selected using a validated intake-interview. Nine age, gender, ethnicity, and educational-level matched non-meditators were recruited from the community. Montreal cognitive assessment (MoCA) score of >26 was one eligibility criterion.

The validated Sinhala version of repeatable battery for the assessment of cognition (RBANS) was used to assess immediate memory, visuospatial/constructional, language, attention, and delayed memory in both groups.

EEG was recorded according to the 10-20 system. In LTM, EEG was recorded with one-minute eyes-closed followed by 19 minutes of meditation while in controls, the total 20 minutes of EEG recording was in an eyes-closed relaxed state of mind. EEG wave frequencies during meditation/relaxed state were analysed from 4 regions: F7-T3, T5-O1, F8-T4, T6-O2.

Results: The mean score of RBANS among LTM (mean age 39.78; SD=9.27 years) was 440.8; SD=109.4 while among NM (mean age 40.44; SD=8.39 years) it was 331.2; SD=101.91 (p=0.139).

Cognitive scores were higher among LTM compared to the NM: immediate memory, LTM=45.44±SD, NM=36.13±SD (p=0.09); visuospatial, LTM=37.78±SD, NM=28±SD (p=0.013); language, LTM=39.67±SD, NM=35.13±SD (p=0.08); attention, LTM=67.22±SD, NM=60.63±SD (p=0.39); delayed memory, LTM=54.78±SD, NM=47.13±SD (p=0.019).

In EEG, a higher percentage of alpha activity was observed among LTM (25.31%) compared to NM (7.75%) (p=0.001) in all the EEG regions. Delta activity predominated in the fronto-parietal region of NM (NM=61.38%; LTM=30.54%; p= 0.003) while alpha activity predominated among LTM (LTM=24.23%; NM=9.63%; p= 0.011).

Conclusion: Long-term meditation enhances all cognitive domains assessed using RBANS with a significant improvement in visuospatial and delayed memory. Long-term meditation also produces changes in EEG frequencies suggesting the possibility of long- and short-term effects of meditation on CNS functions.

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OP: 03

A study on clinical profile of drug resistant epilepsy in children

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Introduction: Epilepsy is a common neurologic disorder with 20-40% remaining drug resistant despite continuous development of antiepileptic drugs. Understanding types and spectrum of drug resistant epilepsy will facilitate targeted investigations and therapy.

Objectives: Study on clinico-etiological features of drug resistant epilepsy in children.

Methods: Observational prospective study was conducted at the Lady Ridgeway Children's Hospital. Children aged 1 month to 16 years with refractory epilepsy based on the ILAE definition of, "failure of adequate trials of two tolerated and appropriately chosen and used Anti-Seizure Medication (ASM) schedules to achieve sustained seizure freedom" were included.

Results: Total number of children were 50. Median age of cohort was 9.5 years (SD of 4.6); majority were in 6-12 years category. Mean age of epilepsy onset was 29.2 months (SD of 35.5 months). Onset of epilepsy was mainly in early infancy (44.0% between 0-6months). Aetiology according to ILAE framework, majority remained unknown (46.0%) structural (40.0%), genetic (2.0%), infections (4.0%), immune (6.0%) and metabolic (2.0%).
The refractory epilepsy types were focal (58.0%), generalized (16.0%), mixed (18.0%) and undetermined (8.0%). Syndrome was diagnosed in 24 (48.0%); West syndrome was commonest. Developmental status at seizure onset, majority was age appropriate (62.0%). Out of them, 9 subsequently slowed in development while 5 showed regression. Based on effect on development and EEG characteristics, these 14 children were categorized to have Developmental Epileptic Encephalopathy (DEE). Commonest epilepsy type in all age groups was focal epilepsy. All these children were trialed on multiple ASDs. Some received other modalities of treatment such as immune therapy (32.0%), ketogenic diet (10.0%) and surgery (16.0%).

Conclusion: Majority of refractory epilepsies in childhood begin in infancy. Majority of this group had no identifiable aetiology based on investigations offered. Detailed genetic testing in this group will help identification of underlying cause of their epilepsy.

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ABSTRACTS OF POSTER PRESENTATIONS

PP: 01

Causes for delay in care seeking in stroke victims admitted to a provincial thrombolytic center in Southern Sri Lanka

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Introduction: Early interventions improve the outcome of patients with stroke. We analysed the data on delay in care seeking among stroke victims in 2018. To find out the causes for delay and to suggest needed improvements.

Method: Data on the time taken to arrive at Teaching Hospital Karapitiya (THK) and causes for the delay in arrival for more than 270 minutes were collected from the responses given by the stroke victims or their family members in 2018. Biodata, level of education, distance to the thrombolytic centre, first place of care seeking and their knowledge on stroke symptoms and the need to seek medical care urgently was analysed. Univariate analysis and multivariate analysis were done with $\chi 2$ statistics and logistic regression, respectively.

Results: Of 168 subjects 59.5% were males and 86.9% were above 50 years. Ninety-seven (66.07%) had reached the thrombolytic center within 270 minutes. The percentage coming directly to a thrombolytic centre was 68(40.5%). In univariate analysis, education above ordinary level, ($p<.001$) living within 20 km of THK (56 (82.3%) $p<.001$) and directly admitting to THK ($p<001$) was significantly associated with early arrivals. Significantly more patients who developed a stroke while working arrived at THK earlier. Other causes mentioned as a cause for delay by the patients were delay in finding a vehicle (57/73), road traffic congestion and poor road conditions and poorly accessible routes. However, the delay in arriving at the THK was not significantly affected by the Level of education, knowledge of the stroke symptoms or urgent nature of the illness. Ninety-seven patients were unaware of the availability of free ambulance service. However multivariate analysis showed that only the distance was an independent predictor.

Conclusions: Although 66% reached the thrombolytic centre within 270 minutes, this can further be improved. The reason for delay was more due to logistic reasons than the knowledge of the patient and care giver regarding the illness or its urgency by the patient or bystanders. Educating public on the availability of the free ambulance service, training the ambulance staff to recognize strokes, and transporting them directly to a thrombolytic centers would further reduce the time delays when admitting stroke patients to hospital.

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PP: 02

The clinical profile of amyotrophic lateral sclerosis patients presenting to the National Hospital of Sri Lanka

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Objectives: Amyotrophic lateral sclerosis (ALS) is a devastating neurodegenerative disease. Published data on ALS in Sri Lanka is scarce. The aim of this study was to describe the clinical profile of ALS patients presenting to the National Hospital of Sri Lanka (NHSL).

Methods: A descriptive cross-sectional study was carried out in 33 consecutive patients with "clinically definite ALS" according to the revised El-Escorial and Awaji criteria, who presented from May 2018-May 2020 to two neurology units at NHSL.

Results: The mean age was 59.7 (±12.3) years while 57.6%(n=19) were males. The average duration for diagnosis was 15.4 (±12.4) months. The commonest first symptom was upper limb weakness 39.4%(n=13) followed by lower limb weakness 33.3%(n=11) and bulbar symptoms 27.3%(n=9). According to King's Staging System, 18.2%(n=6) were in stage 1, 39.4%(n=13) were in stage 2, 36.4%(n=12) were in stage 3 and 3%(n=1) each were in stage 4A and 4B at the time of diagnosis. None had a family history of ALS. Four were treated with Riluzole. Out of all, 45%(n=15), 24.2%(n=8) and 6.1%(n=2) were referred for physiotherapy, occupational therapy and psychiatry assessment respectively. Seventy five percent (18/24) patients with bulbar symptoms, were referred to speech therapists and one had gastrostomy. Advance directives were not discussed with any of the patients.

Discussion: The ALS patients presenting to the NHSL had a mean age of 59.7 years with a slight male preponderance. The average duration taken for diagnosis was 15.5 months while the commonest first presentation was upper limb weakness. Most presented in King's stage 2 or 3 and only a few were treated with disease modifying agents. Multidisciplinary care provided and management of disease related complications were suboptimal. Discussing about advance directives with ALS patients, is yet not part of Sri Lankan practice. This emphasizes the need to enhance standards of care for ALS patients to improve their quality of life.

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PP:03

The rare coexistence of trigeminal autonomic cephalalgia (TAC) with trigeminal neuralgia (tic doloroux) in the same patient: Tac-tic headaches, a case series

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Background: TAC headaches are strictly unilateral, short lasting headaches with ipsilateral cranial autonomic symptoms. Trigeminal neuralgia, also known as tic-doloroux (Tic) involves brief shock-like pain along the distribution of the trigeminal nerve. The occurrence of TACs and Tics ipsilaterally in the same patient is rare. We describe a series of Sri Lankan patients with TAC-tics with an emphasis on clinical significance, pathophysiological conjectures and a literature review.

Case Series: Four patients having TAC-tic combination were evaluated. Two males had CH-tic and CPH-tic respectively whilst one female had SUNA-tic & the other had Probable CPH-tic. Patient with CPH-tic had Tics involving all 3 branches of trigeminal nerve while others had V1 Tics. Male CPH patient had Tic first, followed by CPH 5 months later, whereas the other 3 patients developed both headache types simultaneously. Except in the CH-tic patient, others had both types of headaches occurring simultaneously as well as separately. CH-tic patient had Tics for about 3 days prior to a bout of CH, around the same time of the day as the CH attacks. MRI brain with CISS sequence demonstrated stretching of the ipsilateral trigeminal nerve by vascular loops in all except CH-tic patient, who had a vascular loop in close proximity to the trigeminal nerve without impingement. All patients had satisfactory response to combination treatment targeting both TACs and Tics.

Discussion: Overlapping clinical features in TACs & Tics could indicate a common underlying aetiology giving rise to a spectrum of manifestations. Strictly unilateral nature, the combination of ipsilateral TACs and Tics and the finding of trigeminal neuro-vascular conflict in our patients as well as in the available literature raises the possibility of a common structural aetiology. Further studies involving TACs, Tics and TAC-tic combinations with dedicated trigeminal nerve imaging would be beneficial to further evaluate this phenomenon.

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PP:04

Paediatric multiple sclerosis – case series from Sri Lanka

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Introduction: Paediatric-onset MS is generally defined as MS with an onset before the age of 16 years and it comprises approximately 3 to 10% of patients with the disease. Paediatric MS is more likely to relapse in comparison to the adult-onset cases. However, data from large paediatric cohorts are lacking and no large placebo-controlled studies are yet to be published.

Case series: Six patients with clinically definite paediatric MS, based on McDonald and MAGNIMS diagnostic criteria, were evaluated. All had a relapsing and remitting course. The sex distribution was equal with mean age of onset being 11.5 years (17,16, 15, 9, 6, and 6 years). Three patients initially presented as unilateral optic neuritis and other three as stroke like episodes (acute/ subacute hemiparesis or hemisensory loss). Mean EDSS was 1.83. CSF Oligoclonal bands was checked using the isoelectric focusing method in all patients and was positive only in one patient. Out of the total 6 patients in this group, 3 patients were on DMT. Two patients were started on interferon and one on fingolimod whilst the rest are awaiting DMTs. Patients mean EDSS Score was 1.88. Hence, in this group of Pediatric MS patients outcome was favourable. None Of them had progressive symptoms.

Conclusion: Childhood MS though rare can present with a wide variety of manifestations including optic neuritis, sensory, brainstem-cerebellar and motor symptoms. In our study they presented predominately with optic neuritis and stroke like episodes. Overall, children with MS have a favorable outcome and a slower disease progression. The current first-line treatment for MS in children is either interferon beta or fingolimod.

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PP: 05

Type 3 Gaucher Disease manifest as isolated progressive myoclonic epilepsy in two siblings

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Introduction: Gaucher disease (GD) is a rare autosomal recessive metabolic disorder, caused by a deficiency of beta glucocerebrosidase enzyme resulting in accumulation of glucosylceramide in different organs. It has been classified as type 1, without neuropathic findings, type 2 with acute infantile neuropathic signs and type 3 or chronic neuropathic form. Here, we are reporting two cases of type 3 Gaucher disease in the same family without evidence of other organ involvement. This has not previously been reported in Sri Lanka and rarely reported in current medical literature.

Case 1: A 14-year-old girl who was well up to age of 12 years was referred to us with frequent seizures and progressive cognitive regression. Symptoms started with infrequent episodes of generalized tonic clonic convulsions which did not respond to usual antiepileptics. Examination revealed generalized resting myoclonus, oculomotor apraxia, distal muscle wasting of upper and lower limbs, spasticity, and mild degree of ataxia. General nonverbal skills were affected with relative sparing of verbal skills. There was no organomegaly. Full blood count, blood picture, liver enzymes, serum LDH, HIV screening, X-ray limbs, ultrasound scan abdomen and echocardiogram were normal. Bone marrow biopsy revealed presence of 20% of Gaucher cells without presence of any other atypical cells. EEG showed multifocal epileptiform activity with photosensitivity. MRI brain, nerve conduction study and CPK were normal.

Case 2: Her 17 years old sister also had a similar presentation but with a much slower progression since age of 10 years. There was evidence of generalized resting myoclonus and slowing of horizontal saccade on eye examination. Spasticity, ataxia and organomegaly were absent. Investigations findings were similar to her sister.

Discussion: Even though it is rare, clinicians should consider the rare possibility of type 3 Gaucher diseases in the setting of progressive myoclonic epilepsy, especially without evidence of systemic involvement

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PP: 06

Heroin induced acute myelopathy, rhabdomyolysis and chronic globus pallidus ischemia: uncommon complications of heroin abuse

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Introduction: Heroin or diacetylmorphine is a widely consumed illegal opioid. In addition to systemic complications of heroin, number of neurological manifestations have been described. They include spongiform lekoencephalopathy, vascular ischemic manifestations, wound botulism, transverse myelopathy, neuropathy and cerebral atrophy. Heroin induced myelopathy is a very rare manifestation mostly seen in relation to the first episode of heroin usage or following consumption after a period of abstinence. Neurovascular ischemia is the most common complication of heroin and occurs due to vasospasm, vasculitis or embolism involving globus pallidus, periventricular and subcortical regions. We describe rare case of acute myelopathy, rhabdomyolysis and globus pallidus ischemia in a chronic heroin user.

Case: A 41 year old Sri Lankan man presented with acute bilateral lower limb weakness, numbness and urinary retention followed by unconsciousness four hours after using inhaled heroin. He has consumed heroin first time after two years of abstinence. Examination revealed reduced limb power, hypotonia, hyporeflexia with a sensory level at T7. Joint position sense was absent. Haematology was abnormal with high a very high CPK (10431 U/L), hyponatremia (128 mmol/l), high creatinine (13.2 mg/dl) and positive urine myoglobin level. MRI brain showed T2 FLAIR hyperintensities in C2/3, C4/5 and C5/6 regions involving anterior cord and central cord respectively. There were T2 hypo intensities in the globus pallidi with a central hyper intensity, mimicking a 'tiger's eye appearance' along with contrast enhancement and blooming artifacts in SWI. T2 hyper intensities were also noted in paraspinal muscles due to rhabdomyolysis. Given the history and examination findings the patient was diagnosed as heroin induced acute myelopathy along with rhabdomyolysis. He also suffered from acute kidney injury and chronic ischemia in Globus pallidus. Patient improved with hemodialysis and methylprednisolone.

Conclusion: Suggested mechanisms of acute heroin myelopathy include direct toxicity, hypersensitivity, vasculitis and hypotension. Globus Pallidus is the commonest brain structure to be affected in 5-10% chronic heroin users.

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PP: 07

Neurofibromatosis type 1 presenting as progressive proximal muscle weakness: An uncommon presentation mimicking proximal myopathy

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Introduction: There are three clinically and genetically distinct forms of neurofibromatosis (NF), NF type 1, type 2 and schwannomatosis. The hallmarks of NF type 1 are the multiple café-au-late macules and associated cutaneous neurofibromas. There are various causes for generalized muscle weakness in neurofibromatosis. But progressive proximal muscle weakness is rare in NF type 1. We present an unusual case due to neurofibromas in multiple nerve roots and nerve plexuses.

Case: A 27-year-old female presented with progressive proximal muscle weakness in both lower limb and upper limbs over one year. She did not have features of connective tissue disorders or features of endocrine abnormalities. Her family history was unremarkable. Examination revealed waddling gait, MRC grade 4 proximal muscle weaknesses in both upper and lower limbs without sensory impairment. She has multiple café-au-late macules on her back and axillary freckles. No cutaneous neurofibromas were detected anywhere in the body.

Basic blood and urine investigations and the inflammatory markers were within normal values. The EMG was normal with no myopathic changes. NCS showed patchy asymmetrical sensory abnormalities with relatively preserved motor responses. She was subjected to MRI pan spine which showed multiple neurofibromas and plexiform neurofibromas involving almost all neural nerve roots in varying sizes involving both brachial and lumbosacral plexuses. MRI brain did not show bilateral cerebellopontine angle shwannomas. Her pure tone audiogram was normal. The diagnosis of NF type 1 was made in the presence of multiple plexiform neurofibromas and café-au-late macules with absent bilateral acoustic neuromas.

Conclusion: NF 1 presenting as progressive proximal muscle weakness without sensory signs mimicking a myopathy is exceptionally rare. Neurofibromas only involving the nerve plexuses and roots were the cause and appropriate neuroimaging confirmed it.

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PP: 08

Extensive cerebral venous thrombosis secondary to essential thrombocythaemia treated with lumbo-peritoneal shunting

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Introduction: Essential thrombocythaemia (ET) is a myeloproliferative disease with megakaryocytic hyperplasia in bone marrow. Even though thrombo haemorrhagic complications are common in ET, cerebral venous thrombosis (CVT) is an unusual presentation. We describe a Sri Lankan female with severe CVT due to ET treated with lumbo peritoneal shunting.

Case: A 35-year-old female presented with chronic headache for four months. Examination revealed papilloedema without other focal neurological signs. She did not complain of visual obscurations or diplopia. MRI brain was normal. MRV confirmed severe CVT involving bilateral superior sagittal and transverse sinuses extending up to the jugular bulb. A lumbar puncture revealed a CSF opening pressure of 330mm H_2O and therapeutic removal of 30cc CSF was performed. She was started on oral acetazolamide and anticoagulation. However, her symptoms persisted with recurrent hospital admissions hence a lumbo-peritoneal shunt was inserted, after which she made a significant recovery as evidenced by the decrease in the headache (pain score 8/10 to 2/10) and the number of symptomatic hospital admissions. FBC revealed thrombocytosis with normal haemoglobin level and WBC count. Bone marrow examination revealed megakaryocytic hyperplasia. These findings were in keeping with a myeloproliferative disease, essential thrombocythaemia. Genetic test for myeloproliferative neoplasm (MPN) was positive for JAK2p.V617F mutation. Rest of the thrombophilic screening was negative. Hydroxyurea was added as treatment.

Conclusion: CVT as the first presentation of ET is very rare with only a few cases reported in literature. This case emphasizes the need for extensive evaluation of patients with CVT to find an underlying cause. Lumbo-peritoneal shunting is a treatment option in extreme cases of CVT

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PP: 09

Acute onset generalized weakness: A rare variant of multifocal motor neuropathy

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Introduction: Multifocal motor neuropathy (MMN) is a slowly progressive asymmetrical focal demyelinating neuropathy involving one or few motor nerves commonly in the distal upper limbs. We present a patient consistent with acute multifocal motor neuropathy with conduction blocks (AMMNCB), a rare variant of MMN.

Case: A 69-year-old woman presented with acute onset generalized weakness in 2009, 2013, 2016 and responded to intravenous immunoglobulins (IVIg) without residual weakness. In 2020, she presented with acute onset bilateral lower limb weakness, evolved very rapidly causing generalized weakness within a day. On examination she had asymmetrical lower limb weakness on the left more than right, with proximal and distal power MRC grade 1-2/5. Upper limbs were symmetrically affected with proximal and distal power MRC grade 2/5 with global areflexia. Cranial nerves, sensory, autonomic or sphincter involvement was not present. Nerve conduction study (NCS) disclosed widespread motor CBs in non-compressive sites with diffuse F wave abnormalities with normal sensory conduction. Serology revealed positive anti-GM1 antibodies without cyto-protein dissociation in CSF done after 2 weeks. She responded to IVIg. Follow up NCS after a month revealed persistent motor CBs despite clinical improvement.

Discussion: Acute motor axonal neuropathy (AMAN), acute inflammatory demyelinating polyradiculopathy (AIDP) and chronic inflammatory demyelinating polyradiculopathy (CIDP) were considered in this clinical context. Definite CB is not usually seen in AMAN apart from rare form, AMAN with reversible conduction block variant. AIDP shows definite CBs which disappear with IVIg and also upper limb sensory involvement with sural sparing is classical. Sensory sparing and motor CBs observed alone without any other features of demyelination are against the diagnosis of CIDP. Therefore, the presentation with acute onset generalized weakness, past history of three similar episodes with above-described electrophysiological evidence and serological confirmation are consistent with AMMNCB, a rare presentation of MMN

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PP: 10

Young adult with acute disseminated encephalomyelitis manifesting with myoclonus

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Introduction: Acute disseminated encephalomyelitis (ADEM) is a multifocal demyelinating disease of central nervous system (CNS) which is common in children and rare in young adults. Here we report a case of a young adult diagnosed as ADEM manifesting with myoclonus, a rare manifestation of ADEM.

Case: A 29-year-old male presented with sub-acute onset left lower limb weakness followed by right lower limb and bilateral upper limb weakness with deterioration of cognition with a preceding diarrheal illness. Examination revealed spastic quadriplegia without involvement of cranial nerves or sensory modalities. Later he developed myoclonic jerks involving right and left upper limbs and lower limbs alternately. His basic investigations were within normal range. Brain MRI showed diffuse white matter T2 hyperintensities involving bilateral fronto-parietal-occipital area, with T1 contrast enhancement without diffusion restriction or meningeal enhancement. There was a T2 hyperintensity in C4-C6 spinal segments in the spinal MRI. His cerebrospinal fluid (CSF) analysis was normal with protein 37mg/dl. In EEG, there were generalized slow waves without seizure activity. His infectious, demyelination and autoimmune screening was negative. He improved after high dose intravenous (IV) steroids, plasmapheresis and IV immunoglobulins.

Discussion: In our patient, ADEM was diagnosed with exclusion of infectious, demyelinating, and autoimmune screening with supportive radiological evidence. Movement disorders such as ataxia, myoclonus, dystonia, and chorea have been rarely reported in ADEM and most are among the paediatric population. Myoclonus which manifested in our patient, is encountered as a rare manifestation of ADEM.

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PP: 11

Sensory ganglionopathy showed a good response to immunotherapy

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Introduction: Sensory ganglionopathy/ sensory neuronopathy (SN) is a disease of sensory neuronal cells in dorsal root ganglia (DRG). Autoimmune, paraneoplastic, infectious, toxic and idiopathic causes are notable for etiopathogenesis. As the treatment, immunotherapies have been tried with limited success. We present a case of SN showed a good response to intravenous immunoglobulin (IVIg), a rare response related to treatment of SN.

Case: A 56-year-old male presented with sub-acute onset progressive difficulty in walking associated with numbness of bilateral upper limbs, lower limbs and face for 3 weeks duration. He was ataxic and areflexic with impaired joint position sense and vibration in upper and lower limbs. Pain and temperature sensation were diminished in both hands and feet. Romberg sign was positive. Rest of the neurological examination was normal. He denied sicca symptoms and had a normal systemic examination. Nerve conduction study was suggestive of SN with absent sensory nerve action potentials with normal motor conduction studies. His haematological, biochemical, and radiological investigations were normal including MRI spine and contrast enhanced CT abdomen, chest, pelvis. CSF was normal with normal protein level. Evaluation of autoimmune panel is pending. He had a good response to IVIg within 2 weeks with estimated qualitative improvement of disability with Evaluation of Sensory Ataxia Rating Scale (SEARS) 20/41 to 10/41 and Scale for Assessment and Rating Ataxia (SARA) 8/40 to 4/40.

Discussion: According to the recent data, variety of immunotherapies (glucocorticoids, immunosuppressive agents, plasma exchange, intravenous immune globulin, and rituximab) have been tried in different forms of SN with little success. The response is limited by destruction of the cell body of the sensory neuron. In our patient, there was a good improvement for immunotherapy. We speculate that some of the remaining DRG neurons may have had impaired function due to smouldering inflammation which regained function after IVIg.

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PP: 12

Acute haemorrhagic encephalomyelitis after amphetamine overdose

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Introduction: Acute haemorrhagic encephalomyelitis (AHEM) is a rare variant of acute disseminated encephalomyelitis (ADEM). It is usually after bacterial or viral infection which results in a fulminant and rapid inflammation of myelin due to antigen mimicry. These patients can present with fever, headache, rapidly progressive encephalopathy, and focal neurological signs with a high risk of mortality and morbidity. Although there are reported cases of ADEM following Amphetamine, there are no case reports with AHEM. We present this case report to enlighten the possibility of developing AHEM due to Amphetamine in an era of increased accessibility to street drugs in society.

Case report: A 15-year-old boy, presented with fever for 1-day and seizures after taking few tablets of amphetamine. Although his seizures were settled with IV diazepam, GCS remained persistently low (10/15). Patient was normotensive throughout the hospital stay. WBC was 24.66*103 with neutrophil predominance. Markers of metabolic, infective, and inflammatory were normal. ECG and 2D echocardiogram were also normal. NCCT brain showed haemorrhagic lesions with perilesional oedema on the left posterior frontal and temporal region. Bilateral Periodic Epileptiform discharges were noticed in the EEG. Initially he was treated with IV dexamethasone to reduce cerebral oedema. CSF and autoantibody screening were negative. MRI confirmed haemorrhagic lesions with focal cerebritis on the left fronto-temporal and parietal regions. EEG showed generalized epileptiform discharges with background asymmetrical slowing. Screening for HIV was negative. The patients' GCS returned to normal within 5 days of IV methylprednisolone. The patient was later discharged with no residual neurological deficits and was seizure-free up to date without being on antiepileptics.

Conclusion: Diagnosis of AHEM due to amphetamine toxicity is favoured by patients' clinical history, radiological findings, and exclusion of other common aetiologies together with excellent response to steroids. Timely diagnosis and early initiation of immunosuppressive treatment in AHEM lead to favourable outcome despite high mortality and morbidity. Amphetamine toxicity should be considered as a cause for AHEM in the appropriate clinical setting

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PP: 13

An unusual cause of recurrent lacunar strokes

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Introduction: Lacunar strokes contributes to a quarter of all ischaemic strokes. In most lacunar strokes, the vascular pathology is diffuse. Cumulative disability and cognitive impairment are the major concerns with each stroke recurrence and warrants extensive evaluation for an underlying aetiology. Screening for rarer causes of stroke recurrence should be performed in patients without vascular risk factors and for recurrences despite optimal medical management. Hyper-eosinophilic syndrome (HES) is one rare cause for recurrent strokes and is characterized by peripheral blood eosinophilia with end organ manifestations.

Case report: A previously healthy 56-year-old right-handed male, was admitted outside the thrombolytic time window with sudden onset right hemiparesis sparing facial and bulbar muscles. He had intermittent diffuse headache and generalized pruritus for 3 months preceding the event. Clinical examination was unremarkable except for the motor weakness of right upper and lower limbs. The non-contrast CT brain on admission was normal. Basic investigations revealed a high white cell count with absolute eosinophil count of 6800 cells/cc. Rest of the risk factor evaluation including cardio-vascular assessment and thrombophilic screening were normal. He had mild hepato-splenomegaly in abdominal ultrasound.

His MRI brain was consistent with multiple bilateral subacute white matter infarctions particularly in watershed zones. The MRA and MRV was normal. Digital subtraction angiogram did not show evidence of cerebral vasculitis.

Incidentally detected hyper-eosinophilia was investigated with serology of parasitic infections and anti-neutrophil cytoplasmic antibody which were all negative. Empirical anti-helminthic treatment for 21 days failed to show a decline in eosinophil count. Within 3 months of presentation, patient developed two episodes of transient ischaemic attacks.

Subsequent bone marrow biopsy was suggestive of hyper eosinophilic syndrome, upon which he was started on oral steroids, hydroxyurea and aspirin. He is awaiting PDGFR mutation analysis for further management of the condition.

Conclusion: HES is a potentially treatable cause of recurrent lacunar strokes although rare. Early recognition is beneficial to prevent complications.

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PP: 14

Asymmetrical scapular winging, bilateral facial muscle involvement and distal myopathy with normal CPK; unusual features in anti Jo-I positive polymyositis

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Introduction: Polymyositis is an idiopathic inflammatory myopathy which present with typical symmetrical proximal muscle weakness with the evidence of muscle inflammation. The acute inflammation of muscle results in high levels of CPK and has a relapsing remitting course with a good response to anti-inflammatory treatment. We present here a patient who presented with a chronic myopathy with relapsing remitting fashion, with asymmetrical scapula winging, and bilateral facial weakness, with predominant distal muscle weakness which was supported by EMG.

Case: 65-year-old woman presented with 6-year history of progressive muscle weakness. Her initial symptoms were associated with involvement of lower limb proximal muscles and then she noticed difficulty in walking mainly due to slapping of her feet on the ground. There were episodes of worsening of weakness which was responsive to IV methyl prednisolone. She never experienced muscle pains, muscle cramps or positive or negative sensory symptoms. On examination, she had bilateral foot drop with limb girdle weakness. Additionally, she had asymmetrical scapula winging (R> L) and bilateral facial muscle weakness. Her CPK was found to be mildly elevated. (300). EMG was in favour of myopathy (distal > Proximal) and her nerve conduction was normal. Muscle biopsy revealed inflammation. Specific antibody testing with Jo-1 results was found to be positive.

Conclusion: Long standing idiopathic inflammatory myopathy can be a diagnostic challenge when presented with atypical features. High degree of suspicion is needed as these are treatable conditions with good response to immuno-suppressive therapy.

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PP: 15

Spinocerebellar ataxia (SCA) 38 with evidence of excessive brain iron accumulation: A neurodegeneration with brain iron accumulation (NBIA) look alike

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Introduction: Neurodegeneration with brain iron accumulation (NBIA) is where early deposition of iron in the basal ganglia and the other deep grey matter is characteristically associated with movement disorders. Spinocerebellar ataxia (SCA) is characteristically a neurodegenerative disorder predominantly affecting the cerebellum and the corticospinal tracts. Rarely excess iron accumulation is reported in long standing SCA patients. We report a case of genetically proven SCA 38 with evidence of excess iron deposition in the basal ganglia similar to a NBIA.

Case report: A 38-year-old woman presented with difficulty in walking and maintaining her balance for 3 years. She did not complain of any sensory symptoms and denied any defect in the sense of smell or hearing. On examination she was ataxic with prominent cerebellar signs. There was gaze evoked horizontal nystagmus. Appendicular rigidity was prominent with exaggerated reflexes and extensor planter responses. She did not have any evidence of pes cavus. She did not have KF rings. Ceruloplasmin, serum cooper, TSH, Serum Ferritin were normal. MRI showed evidence of brain iron accumulation with prominent cerebellar atrophy. Her genetic studies showed heterozygous ELOVL5 gene mutation due to c.304C>T mutation characteristic of SCA 38.

Conclusion: SCA 38 is characterized by slowly progressive ataxia with prominent cerebellar signs. Evidence of brain iron accumulation which normally occurs with NBIA and Wilson's disease (WD) is unusual in SCA. Brain iron accumulation was previously reported only in cases of SCA 15. It has never been reported before in SCA 38. A genetic evaluation in such cases is of paramount importance to make an accurate diagnosis distinguishing SCA from NBIA and WD.

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PP: 16

Tuberculosis of the parotid gland associated with lower cranial neuropathy

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Introduction: Parotid gland involvement as a form of extra pulmonary tuberculosis is a very rare manifestation even in countries where tuberculosis is endemic. Extension of tuberculosis to skull base with multiple cranial nerve involvement is even rarer. We report a case of atypical presentation of tuberculosis with multiple cranial nerve palsies.

Case: A previously healthy 40-year-old woman presented with progressive dysphagia for one-week without a history of fever, chronic cough, or headache. Later she developed deviation of mouth to the left. On examination she had bilateral nontender asymmetrical parotid enlargement without cervical lymphadenopathy. Nervous system examination revealed right sided lower motor type of facial nerve palsy, right palatal palsy and weakness of left sternocleidomastoid and trapezius with wasting. ESR was 40mm/1st hour with normal CRP. Cerebrospinal fluid analysis showed elevated protein of 143mg/dl with lymphocyte count of 10/cumm. Contrast enhanced CT (CECT) of head, neck and chest showed enlarged mediastinal lymph nodes and bilateral asymmetric parotid enlargement. Sputum for AFB and Mantoux test were negative. Tuberculosis PCR was negative. Serum ACE level and calcium were normal. MRI brain showed enhancing thickened right CN VII and left CN XII with bilateral parotid enlargement without meningeal thickening and enhancement. Excision biopsy of left parotid gland showed caseating granulomatous inflammation characteristic of tuberculosis. Retroviral and VDRL antibodies were negative. She was empirically started on fixed dose combination of anti-tuberculosis treatment (FDC4) for one year without steroids. During the first three months of treatment parotid swelling regressed and dysphagia and facial nerve palsy also improved remarkably.

Discussion: Granulomatous diseases such as tuberculosis and sarcoidosis were the main differentials. Even though the caseating granulomatous inflammation is characteristic of tuberculosis, sarcoidosis also can rarely give rise to it. Our patient responded well to anti tuberculosis treatment without steroids clearly supported the diagnosis of tuberculosis. Diagnosis of tuberculosis with atypical presentation needs a high degree of clinical suspicion.

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PP: 17

Extensive tractopathy with anterior cord involvement and "inverted V sign" in B_{12} deficiency due to pernicious anemia

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Introduction: Subacute combined degeneration of the cord (SACD) due to vitamin B_{12} deficiency typically causes demyelination of the dorsal and lateral spinal cord sparing the anterior portion. B_{12} deficiency due to pernicious anemia is common amongst Caucasian populations, but rarely seen in south Asia. We present a Sri Lankan lady who developed severe SACD due to B_{12} deficiency with pernicious anemia. Her radiology demonstrated involvement of all tracts of the spinal cord including the anterior with the inverted V sign.

Case report: A 49 years old non vegetarian woman presented with marked upper and lower limb weakness and numbness for 2 years. She was pale and pigmented. She had flaccid paraparesis of the lower limbs. Distal motor weakness was noted in the upper limbs. All sensory modalities of the lower limbs were impaired. Blood picture reveled severe macrocytic anemia with hyper-segmented neutrophils. Bone marrow examination demonstrated a megaloblastic marrow with giant metamyelocytes and erythroid hyperplasia. Serum vitamin B_{12} was low 67 (140 -650***μmol/l***). MRI spine demonstrated significant high signal intensities in anterior, lateral and dorsal columns with white matter changes in brain. Dorsal column hyperintensity specifically showed the inverted V sign. Both intrinsic factor and parietal cell antibodies were positive. Severe SACD due to pernicious anemia was diagnosed. Treatment with vitamin B_{12} partially alleviated her symptoms.

Conclusion: Tractopathy in vitamin B_{12} deficiency is rare. Involvement of the anterior column has been previously reported only in a single case study. Inverted V sign "rabbit ear sign "is a classic finding in SACD. Severe vitamin B_{12} deficiency if not detected early and treated could result in severe neurological sequel.

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PP: 18

Neurocysticercosis in a young Sri Lankan presenting with adult onset epilepsy

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Introduction: Neurocysticercosis (NCC) is a rare neurological disorder caused by the larval stage of the pork tapeworm *Taenia solium* (TS). According to the WHO, neurocysticercosis is the most frequent preventable cause of epilepsy worldwide. Even though cysticercosis is endemic in India it has not been reported from Sri Lanka in an adult.

Case Report: A 29-year-old man presented with chronic headache and adult-onset epilepsy. He was well before headache began 18 months ago. He also suffered 2 episodes of generalized tonic-clonic seizures within the last 2 months. Neurological examination including fundi was normal.

His haematological and biochemical evaluation was normal. There was no peripheral eosinophilia. His CSF too was normal with normal protein, cells, sugar etc. NCCT brain had multiple hypodense areas in bilateral frontal and left parietal regions. His MRI brain revealed multiple focal lesions in bilateral cerebral hemispheres. On T2 MRI images these lesions had a target appearance due to multiple alternating signal intensities. All lesions enhanced with gadolinium. The MRI appearance was characteristic of parasitic infection due to NCC with lesions of different stages.

TS antibodies were negative in both serum and CSF. Repeat MRI brain imaging showed new lesions appearing whilst some old lesions were disappearing. With the clinical history and neuroimaging findings suggesting NCC, the patient was started on Albendazole 15 mg/kg daily dose with steroids, dexamethasone 0.1 mg/kg/day. The treatment was continued for 14 days. Within 2-3 days of initiation of treatment, the patient had a significant improvement in headache. Post treatment MRI showed near complete resolution of the previous lesions without any new lesions.

Conclusion: Even though serological diagnosis of cysticercosis may be difficult, a proper clinical evaluation together with imaging findings led to the diagnosis of NCC. It is interesting as to why an infectious disease endemic in neighbouring India is so uncommon in Sri Lanka.

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PP: 19

Symptomatic Cluster-tic syndrome due to a meningioma

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Background: The combination of TAC headaches with trigeminal neuralgia (tic-doloroux) is a rare occurrence. Several such cases have been reported in literature including from Sri Lanka. Some were symptomatic TAC-tics, whilst a definite cause was not identified in others. We report a case of a Sri Lankan female with cluster-tic attacks occurring symptomatically due to a meningioma.

Case: A 52-year-old female presented with right side-locked headaches for 6 years. She had 1-2 attacks lasting about 20 minutes every day, associated with agitation, and ipsilateral cranial autonomic symptoms including red eye, partial ptosis, tearing, nasal congestion and rhinorrhoea. Attacks were very severe, and patient had a poor response to migraine treatment which had been previously prescribed. She developed brief shock-like stabbing pain in the right periorbital region 8 months prior to presentation to us. These occurred in bouts lasting about 15 minutes, several times a day, and were comparatively less severe and were not associated with autonomic symptoms. There were no cutaneous triggers. Two types of pains occurred simultaneously as well as sequentially. Acute attacks showed significant improvement with high flow oxygen, however, different combinations of preventive medication failed to reduce the frequency of attacks. MRI of the brain with gadolinium enhancement showed a meningioma along the lateral wall of the cavernous sinus with impingement of the right trigeminal nerve. Patient underwent surgical excision of the meningioma, with significant improvement of symptoms.

Discussion: This patient had features suggestive of chronic cluster headache and right-sided probable trigeminal neuralgia, attributable to the meningioma. Available literature has descriptions of many cases with this combination, and some of these were due to structural causes such as pituitary adenoma, demyelination plaques, arachnoid cysts and vascular loops. An extensive search for an underlying structural lesion should be done in all patients with TAC-Tics and isolated TACs.

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PP: 20

Dural and bone metastasis presenting as Gradenigo syndrome.

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Introduction: The clinical triad of ipsilateral abducens nerve palsy, facial pain in the trigeminal nerve distribution, and suppurative otitis media is known as Gradenigo syndrome. Giuseppe Gradenigo first described these classic symptoms of petrous apicitis in a case series published in 1904.

Case report: A 82-year-old female patient presented with left side lateral rectus palsy, retro orbital and facial pain, left ear serous discharge, left side trigeminal nerve sensory loss with absent corneal reflex and left side trigeminal nerve motor weakness with elevated ESR and CRP suggesting Gradenigo syndrome. MRI revealed metastatic deposits on apex of the left side petrous bone. CT chest and abdomen revealed metastasis in liver, lung and kidney. Primary was found to be a breast carcinoma.

Discussion and conclusion: Infectious causes of Gradenigo syndrome have become exceedingly rare in the modern antibiotic era. However, Gradenigo syndrome has been reported in association with other non-infectious etiologies, like granulomatous diseases, tumours and growths such as cholesteatoma and venous sinus thrombosis. Gradenigo syndrome due to metastatic deposits is rarely reported.

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PP: 21

Anti-NMDAR encephalitis; a great mimicker?

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Introduction: Stroke in young is rare with a published incidence varying from 5 to 15 per 100 000 person-years in many European studies up to 40 per 100 000 person-years in some African countries. Presentation of young onset stroke in close temporal profile with NMDR encephalitis is scarcely described. This case report describes an unusual case of anti NMDAR encephalitis presenting as a stroke like episode which was later complicated with stress induced cardiomyopathy.

Case: We report a 17-year-old male who presented with acute onset right sided hemiparesis. This was proceeded by insidious onset, progressively worsening behavioral changes for 2 weeks. Patient had CT and MRI evidence of left fronto-parietal cerebral infarction with normal vasculature. On evaluation for young onset stroke, patient was noted to have severe left ventricular dysfunction with apical ballooning of heart. He subsequently developed seizures, autonomic instability, and oro-facial dyskinesia. A diagnosis of anti-NMDAR encephalitis was made based on clinical features and positive CSF and serum anti-glutamate receptor antibody (NMDA). Patient responded to immunosuppression and had complete recovery of neurological disability but with residual MRI abnormalities. His cardiac function was normalized once the autonomic instability recovered.

Discussion: This case highlights stroke like presentation as an atypical manifestation of anti NMDA receptor encephalitis. The possibility of neurogenic stunned myocardium as a potential source of embolism should also be considered. High degree of suspicion is necessary, as early diagnosis and intervention may result in a successful outcome.

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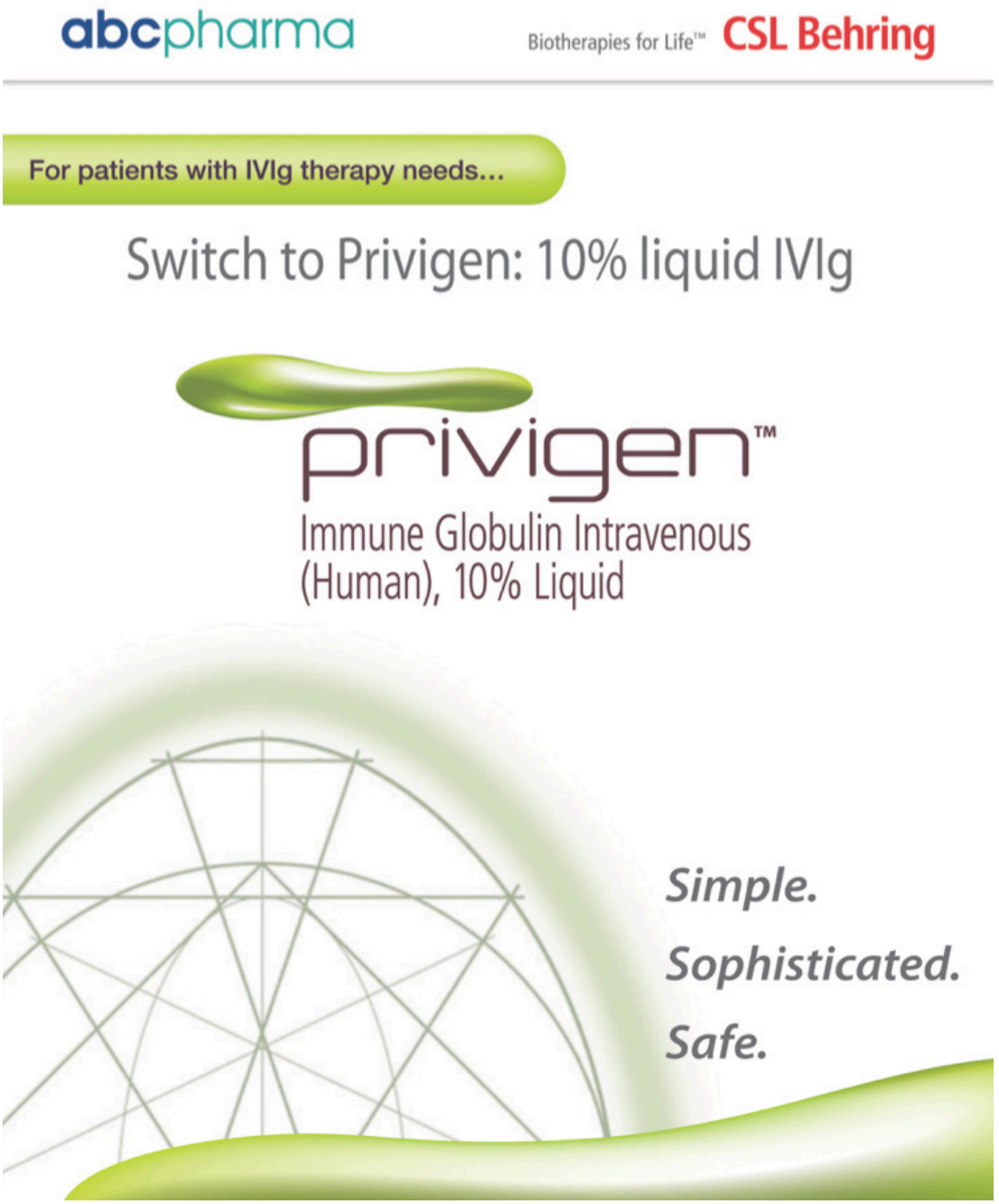
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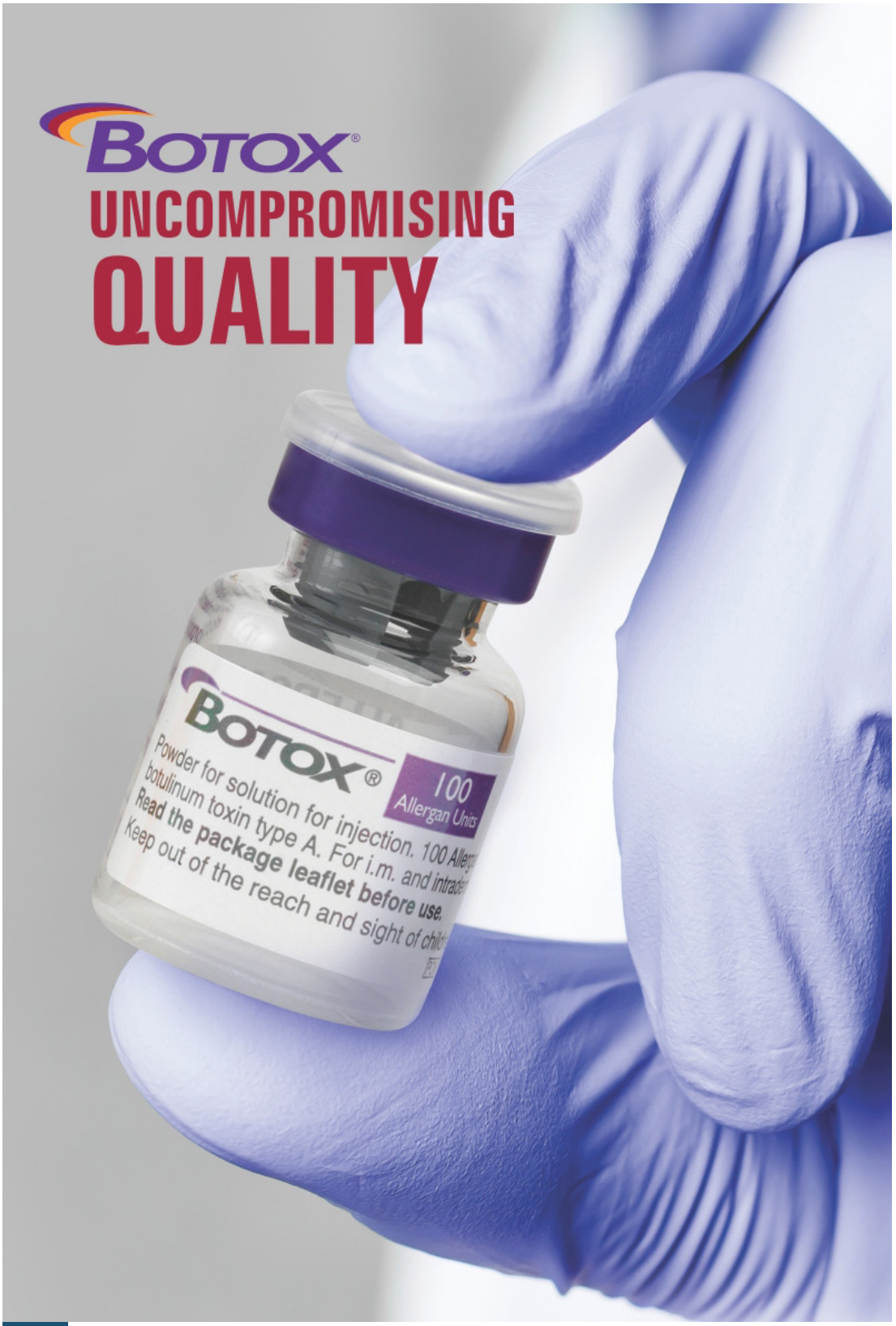
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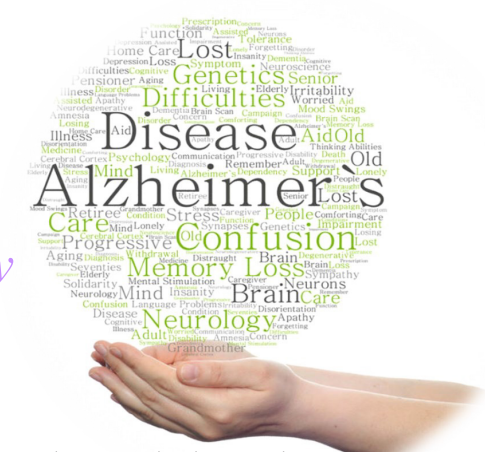

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