32nd European Neurology Congress

&

12th International Conference on

Vascular Dementia

July 22-24, 2019 London, UK

Special Session

Day 1

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Ann Ali Abdelkader Hanafy

Cairo University, Egypt

Basics of electrophysiological assessments of entrapment neuropathies

Nerve compression syndrome or compression neuropathy, is a medical condition caused by direct pressure on a nerve. It is known colloquially as a trapped nerve, though this may also refer to nerve root compression (by a herniated disc, for example). Its symptoms include pain, tingling, numbness and muscle weakness. I will discuss the clinical presentation of various entrapment nerve sites and the value of nerve conductions and electromyography in the diagnosis of each one.

Biography

Ann Ali Abdelkader Hanafy has completed her MD at the age of 30 years from Cairo University and postdoctoral studies from Cairo University School of Medicine. She is a Professor of Clinical neurophysiology & the President of Egyptian Clinical Neurophysiology Society. She has published more than 100 papers in local and international journals.

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Scientific Tracks & Abstracts

Day 1

SESSIONS

Neurology | Pediatric Neurology | Neurogenetic and Neurometabolic Disorders | Neuro Cardiology & Strokes Clinical Trails & Case Reports | Neuropathology | Clinical Neurophysiology | Neurological Diseases

Chair: William C. L. Stewart, The Abigail Wexner Research Institute at Nationwide Children's Hospital, USA
Co-Chair: Nihar Ranjan Haldar, Nobel Medical College Teaching Hospital, Nepal

SESSION INTRODUCTION

Title: Safety and efficacy of adjunctive perampanel in paediatric patients (aged 4 to <12 years) with partial-onset seizures (POS) or primary generalised tonic-clonic seizures (PGTCS): Final results from the 311 core study
Leock Y. Ngo, Eisai Inc., USA

Title: Clinical exome sequencing of patients from a highly consanguineous population: Novel pathogenic variants impacting neurological function
Shahid Aziz Mian, King Fahad Medical City, Saudi Arabia

Title: Diagnosing leprosy at its neuritic phase
Nihar Ranjan Haldar, Nobel Medical College Teaching Hospital, Nepal

Title: Malic enzyme 2 and genetic generalized epilepsy
William C. L. Stewart, The Abigail Wexner Research Institute at Nationwide Children's Hospital, USA

Title: Incidence of stroke among diabetic nephropathypatients: A meta-analysis
Abdulrahman O. Alharbi, Majmaah University, Saudi Arabia

Title: Study 506 – third interim analysis of a retrospective, phase IV study of perampanel in real-world clinical care of patients with epilepsy: Paediatric subgroup (aged <12 years)
Manoj Malhotra, Eisai Inc., USA

Safety and efficacy of adjunctive perampanel in paediatric patients (aged 4 to <12 years) with partial-onset seizures (POS) or primary generalised tonic-clonic seizures (PGTCS): Final results from the 311 core study

Leock Y. Ngo[1], Robert Flamini[2], Andras Fogarasi[3], Mathieu Milh[4], Steven Phillips[5], Shinsaku Yoshitomi[6], Anna Patten[7], Takao Takase[8] and **Antonio Laurenza[1]**

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[3]Epilepsy Centre, Bethesda Children's Hospital, Hungary
[4]La Timone-Enfants Hospital, France
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[8]Eisai Co., Ltd., Japan

Perampanel is a once-daily oral anti-seizure drug for POS and PGTCS. Study 311 (NCT02849626) is a global, multicentre, open-label, single-arm study assessing the safety, tolerability, pharmacokinetics and efficacy of once-daily adjunctive perampanel oral suspension in patients aged 4 to <12 years with POS (with/without secondarily generalised seizures [SGS]) or PGTCS. We report safety, tolerability and efficacy data from the 311 Core Study. This study included a 4-week Pre-treatment Period, 23-week Treatment Period and 4-week Follow-up Period. Primary endpoints were safety and tolerability. Secondary endpoints included median percent change in seizure frequency per 28 days from Baseline during the Treatment Period, and 50% responder and seizure-freedom rates during Maintenance (Core Study) and longer-term treatment (≤52 weeks). In total, 180 patients (POS, n=149; PGTCS, n=31) received ≥1 perampanel dose (mean age [standard deviation], 8.1 [2.09] years; female, 48.9%); 146 (81.1%) patients completed the Core Study and 34 (18.9%) discontinued. Adverse events (AEs) were the primary reason for discontinuation (n=14 [7.8%]). Median (minimum, maximum) dose of perampanel was 8.0 (2, 16) mg/day and duration of exposure was 22.9 (0, 27) weeks. Treatment-emergent AEs in ≥10% of patients were: somnolence, nasopharyngitis, dizziness, irritability, pyrexia and vomiting. Median percent reduction in seizure frequency per 28 days from Baseline, 50% responder rates and seizure-freedom rates, respectively, were: POS: 40.1%, 46.6% and 11.5%; PGTCS: 69.2%, 63.6% and 54.5%; SGS: 58.7%, 64.8% and 18.5%. Adjunctive perampanel was generally safe, well tolerated and efficacious in children aged 4 to <12 years with POS, SGS or PGTCS.

Biography

Leock Y. Ngo has a PhD in Pharmaceutical Sciences from the University of Alberta, Canada and was a Postdoctoral Research Fellow at the University of Washington in Seattle, Washington. Stella is Director in Clinical Research at Eisai Inc., responsible for the development of new anti-epilepsy drugs. She is the International Project Lead for Fycompa® and Inovelon®, and the Clinical Lead for new chemical entities in early development for epilepsy treatment. Before joining Eisai, Stella gained 19+ years' experience in clinical pharmacology and clinical trials across various therapeutic areas, including neurology (Alzheimer's disease and peripheral neuropathy), oncology, pulmonology and autoimmune/inflammatory disorders.

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Clinical exome sequencing of patients from a highly consanguineous population: Novel pathogenic variants impacting neurological function

Shahid Aziz Mian
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Saudi Arabia has a highly consanguineous population with specific geographical regions estimated to have rates exceeding greater than 80%. The downstream effect of such population dynamics is to significantly enrich the frequency at which recessive pathogenic variants occur and consequently their associated Mendelian disorders. This is evident at both a community and a family level. King Fahad Medical City (KFMC) is a tertiary care facility that diagnoses patients with inherited disorders through exome sequence analysis of germline DNA. Many of the biological pathways negatively impacted by these pathogenic changes manifest at a neurological level. These include for example intellectual disability, ataxia, epilepsy and white matter structural changes. The Department of Pathology and Clinical Laboratory Medicine (PCLM) has sequenced the exomes of over 1100 patients. Novel pathogenic variants in genes biologically and clinically linked to specific neurological conditions have been identified. Furthermore the PCLM knowledge base has also implicated novel genes with no known function, to a variety of neurological conditions. Evidence is presented at how genetic analysis of exome sequence data derived from patients orginating within highly consanguineous populations can lead to the identification of novel genes/genetic variants linked to neurological physiology.

Biography

Shahid Mian is a Consultant Clinical Research Scientist, PhD within the Department of Pathology and Clinical Laboratory Medicine (PCLM) at King Fahad Medical City (KFMC), Saudi Arabia. PCLM is a College of American Pathologists (CAP) accredited laboratory. He has responsibility for establishing bioinformatic and variant reporting pipelines for the clinical exome analysis of paediatric patients with suspected inherited disorders. He has reviewed over 500 clinical exome reports produced by third party laboratories for PCLM, independently reported over 100 patient exome results to KFMC physicians and analysed over 1300 exomes bioinformatically.

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Notes:

Diagnosing leprosy at its neuritic phase

Nihar Ranjan Haldar
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Introduction: Leprosy is known to common people including medical practitioners as a disfiguring contagious disease. Patients suffering from leprosy usually have ulcers, in the extremities loss of fingers, nasal tips and ear lobule if not treated. They go to dermatologist or laporalogists. Pathophysiology says it is an illness which affects nervous system i.e. peripheral sensory and motor nerves. Ulceration and disfiguring are secondary to trauma and later involvement of skin. It is a very common infective illness of nervous system if taken worldwide statistics (>200,000 new case per year). It is neglected by neurologist when patients present initially with neurological symptoms, it becomes obvious when skin is affected. Hypertrophic neuropathy is a common type of initial association which can be detected when first they present with minor neurological symptoms. Ultrasonography of nerves as a routine investigation tool is neglected in most of the places which could have identified the disease. Assessment of hypertrophy or other pathologic changes by ultrasonic examination was practiced by us to detect enlargement of any kind.

Materials & Methods: We report 1200 consecutive cases, studied in patients from Nepal, India, Bhutan and Bangladesh. Patients were from neurology and dermatology clinics. High resolution Ultrasonography machine and 12 MHz linear transducer were used for the investigation. Hypoechoic nerves were subjected to aspiration cytology for detecting acid fast *bacilli*. Duration of study was from 2005 to 2018.

Results: From a total of 1200 cases, 75% had thickened nerves, 20% had doubtfully thickened nerves, 11% normal nerves, 3% cases showed nodularity and nerve abscess was seen in 2% cases; 43% had hypoechoic nerves; Aspiration cytology was done in 3323 hypoechoic nerves, of which 27% had granulomatous inflammation, 8% were AFB positive and 20% had no yield (Figures rounded).

Conclusion: It is true that all leprosy patients to start with are pure neuritic leprosy with sensory or motor symptoms. Early diagnosis is very important and obvious that doing ultrasonography of nerves is a very useful method to identify certain common group of peripheral nerves in medical practice, especially in tropical countries. Further development and practice in this regard needs to be done using high resolution ultrasonography machine and expert personnel generation. Awareness among general physician and neurophysician are very much needed for early identification of the disease.

Biography

Nihar Ranjan Haldar is 60 years old and a resident of Siliguri, Darjeeling, India. He completed his M.B.B.S from Calcutta University in 1982, MD (Medicine) in 1987 and DM (Neurology) in 1990 from PGIMER Chandigarh. He Practicing Neurology in India, Nepal, Bhutan & Bangladesh for 27 years. He presently works as a Professor in the Department of Neurology at Nobel Medical College Teaching Hospital & Research Centre, Biratnagar, Nepal. He is also Director of Tenovus Research & Diagnostic Centre and Founder Director of Mrigna Centre for Epilepsy. Nihar Ranjan Haldar engaged in patient care, neuroelectrophysiology and research work. He presented and published his work in various Conferences and Journals. He is also member of Neurology Society of India, Association of Neuroscientist of Eastern India, Indian Academy of Neurology and American Academy of Neurology.

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Notes:

Malic enzyme 2 and genetic generalized epilepsy

William C. L. Stewart, Meng Wang and **David A Greenberg**
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Genetic generalized epilepsy (GGE) is a highly heritable condition (h^2=66%) consisting of epileptic syndromes with overlapping symptoms. Previous studies (both linkage and association) identified malic enzyme 2 (ME2) as a candidate susceptibility gene for adolescent-onset GGE. To definitively test *ME2's* influence on GGE, we used three different approaches. First, we compared a newly recruited GGE cohort with an ethnically matched reference sample from 1000 genomes, using an efficient test of association (POPFAM+). Second, in a previously collected data set, we replaced the original controls with ethnically matched reference samples to minimize the confounding effect of population stratification and we used POPFAM+ in the re-analysis. Third, in a post hoc analysis of healthy human pre-frontal cortex, we identified single nucleotide polymorphisms (SNPs) influencing ME2 messenger RNA (mRNA) expression and then, we tested those same SNPs for association with GGE in a large case control cohort. In the analysis of our newly-recruited GGE Cohort, we found a strong association between an ME2 SNP and GGE (p =0.0006 at rs608781). In the re-analysis of previously collected data, we confirmed the Greenberg *et al.,* (2005) finding of a GGE associated *ME2* risk haplotype. Finally, in the post hoc ME2 expression analysis, we found evidence for a possible link between GGE and *ME2* gene expression in human brain. Overall, our research (and the research of others) provides compelling evidence that ME2 influences adolescent onset GGE susceptibility.

Biography

William C. L. Stewart has completed his PhD in Statistics from the University Washington in 2005, and finished his Postdoctoral studies in the Biostatistics Department at the University of Michigan in 2008. He is a Principal Investigator at the Abigail Wexner Research Institute of Nationwide Children's Hospital. He is an Assistant Professor of Statistics and Pediatrics at Ohio State University. He has published more than 30 papers in peer-review journals and has served on the Editorial Board of Frontiers in Genetics for nine years.

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Notes:

Incidence of stroke among diabetic nephropathypatients: A meta-analysis

Abdulrahman O. Alharbi[1], **Sultan Al Dalbhi** [2] and **Norah A .Alharbi** [3]
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[2]Prince Sultan Military Medical City, Saudi Arabia
[3]Qassim University, Saudi Arabia

Background and Objectives: The association of micro vascular complications (diabetic nephropathy) with the stroke is limited because it will require huge sample size of diabetic population with nephropathy and long follow-up period to see the association or development incidence of stroke among these patients. So, we conducted out this meta-analysis of the existing studies to find out the incidence/ risk of stroke among diabetic nephropathy patients and and, explore the association between stroke and proteinuria in a diabetic nephropathy population and to describe Does Degree of Proteinuria a Clinical Matter!! ?

Methods & Materials: We searched the existing databases from the year 1995 to August 2018 by using the MeSH terms. All cohorts, cross sectional studies were searched for, fulfilling the inclusion criteria and as per operational definitions. The quality assessment criterion for quality of studies was already predefined.

Study Result: Seven studies were found to be eligible for inclusion in the meta- analysis. The hazards or risk of stroke development among diabetic patients was 3.25 times higher in patients with nephropathy as compared to patients without nephropathy.

The pooled hazards ratio of 1.46 (95% CI=0.81-2.60) and of 1.65 (95% CI=0.53-5.11) among diabetic patients with micro albuminuria and macro albuminuria respectively.

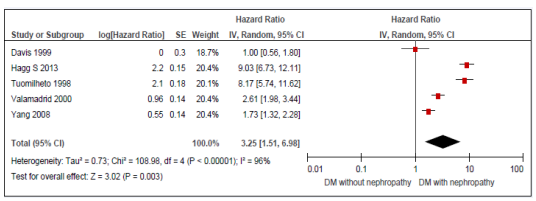

Figure1: Forest plot- Hazard ratio of stroke among diabetic patients with and without nephropathy

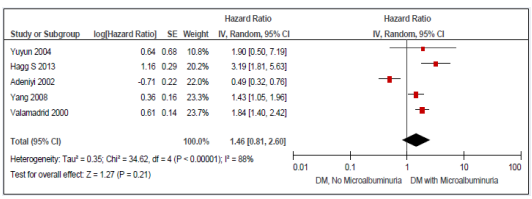

Figure2: Hazard ratio of stroke among diabetic patients with and without Micro albuminuria

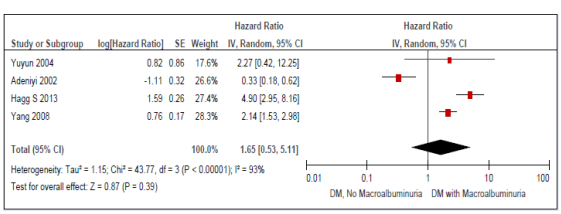

Figure3: Hazard ratio of stroke among diabetic patients with and without Macro albuminuria

Conclusion: Diabetic nephropathy patients have a higher incidence and risk of stroke compared to diabetic patients without nephropathy. This is the first meta-analysis which has included studies from January 1995 to August 2018 to the best of our knowledge which has tried to compare the risk/ hazard of stroke among diabetic patients with and with out nephropathy and sub-group analysis for micro and macro albuminuria.

Biography

Abdulrahman Alharbi is currently working as an Assistant Professor of Neurology & Consultant Stroke Neurologist in the College of Medicine at Majmaah University.

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Notes:

Study 506 – third interim analysis of a retrospective, phase IV study of perampanel in real-world clinical care of patients with epilepsy: Paediatric subgroup (aged <12 years)

Manoj Malhotra[1], Katherine Moretz[2], James Wheless[3], Eric Segal[4], Marcelo Lancman[4], Anna Patten[5] and **Betsy Williams[1]**
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[2]Meridian Clinical Research, USA
[3]University of Tennessee, USA
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Perampanel is given daily once orally as anti-seizure drug for partial-onset seizures (POS) and primary generalised tonic clonic seizures. We report second interim results for paediatric patients from the multicentre, non-interventional, Phase IV, retrospective study 506 (NCT03208660), to assess retention rate, safety and dosing experience of perampanel administered to patients with epilepsy during routine clinical care. Data were obtained from medical records of patients initiating perampanel after 1 January 2014. Primary endpoint is retention rate (proportion of patients in Safety Analysis Set [SAS] remaining on perampanel). Safety, efficacy and dosing experience are secondary objectives. Interim SAS comprised 605 patients; 68 were aged <12 years (mean age [standard deviation (SD)], 6.7[3.0] years). Seizure types included: complex partial, n=33 (48.5%); POS with secondary generalization, n=11(16.2%); generalized tonic-clonic, n=21 (30.9%). Mean (SD) cumulative duration of exposure to perampanel was 14.3(11.5) months and mean (SD) maximum perampanel dose was 5.4(3.2) mg. At data cut-off (5 March 2018), 34(50.0%) paediatric patients remained on perampanel 33(48.5%) had discontinued, primarily due to adverse event (AE; n=15 [22.1%]) and inadequate therapeutic effect (n=11 [16.2%]). Retention rates at 3, 6, 12, 18 and 24 months were 82.4% (n=56/68), 66.2% (n=43/65), 61.0% (n=36/59), 53.2% (n=25/47) and 48.6% (n=17/35), respectively. Treatment emergent AEs occurred in 39.7% of patients; most common were abnormal behavior, aggression and irritability (all 5.9%). This subgroup analysis suggests that daily oral doses of adjunctive perampanel are generally well tolerated, with favorable retention rates for ≤2 years in pediatric patients (<12 years) with epilepsy.

Biography

Manoj Malhotra received his Medical Degree from Wayne State University in Detroit, Michigan. He completed his Neurology residency and two fellowships at The Cleveland Clinic in Cleveland, Ohio. He is the Vice President, Head of Medical Affairs for the Neurology Business Group at Eisai Inc. He is responsible for Medical Affairs activities for the Americas and is Global Medical Lead for Epilepsy. He holds six neurology board certifications (neurology, epilepsy, sleep medicine, clinical neurophysiology, vascular neurology and electrodiagnostic medicine) and has extensive experience in neurodegenerative diseases, rare diseases and epilepsy. His industry experience includes working at Novartis, Takeda and Mallinckrodt.

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Notes:

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Scientific Tracks & Abstracts

Day 2

Day-2

SESSIONS

Pediatric Neurology | Central Nervous System | Neuro Cardiology & Strokes | Neuropathology | Vascular Dementia and Stroke | Neuropathology | Neurogenetic and Neurometabolic Disorders | Dementia

Chair: Alper İbrahim Dai, Gaziantep University, Turkey
Co-Chair: Kasid Ahmed Nouri, Mohammed Bin Rashid University of Medicine and Health Sciences, UAE

SESSION INTRODUCTION

Title: Efficacy of stem cell therapy in ambulatory and non-ambulatory children with Duchenne muscular dystrophy: Phase I–II
Alper İbrahim Dai, Gaziantep University, Turkey

Title: *Varicella-zoster*
Kasid Ahmed Nouri, Mohammed Bin Rashid University of Medicine and Health Sciences, UAE

Title: Silent brain infarction and metabolic syndrome in middle aged Egyptian ischemic stroke patients
Wael Osman M Amer, Al Azhar University, Egypt

Title: Giving a voice to neurological disorders in Sub-Saharan Africa
Chika Okwuolisa, Brain and Spine Foundation, Africa

Title: Alanine rich dipeptide repeat proteins sequester arginine rich dipeptide repeat proteins in a cellular model of *C9orf72*
Katherine Radcliffe, University of Manchester, UK

Title: Two homozygous *KIF1C* mutations in a Turkish family with cerebellar dysfunction and spastic paraparesis
Güllü Tarhan, Gaziosmanpasa Taksim Training and Research Hospital, Turkey

Efficacy of stem cell therapy in ambulatory and non-ambulatory children with Duchenne muscular dystrophy: Phase I–II

Alper Ibrahim Dai[1], Osman Baspinar[1], Ahmet Yeşilyurt[2], Eda Sun[3], Çigdem Inci Aydemir[4], Olga Nehir Öztel[4], Davut Unsal Capkan[5], Ferda Pinarli[2], Abdullah Agar[6] and **Erdal Karaöz[3, 4, 7]**
[1]Gaziantep University, Turkey
[2]Diskapi Yildirim Beyazit Research and Education Hospital, Turkey
[3]Istinye University, Center for Stem Cell Research and Application, Turkey
[4]Liv Hospital – Center for Regenerative Medicine and Stem Cell Research and Manufacturing, Turkey
[5]Deva Hospital, Turkey
[6]University of Travnik, Bosnia and Herzegovina
[7]Istinye University, Turkey

Purpose: Duchenne muscular dystrophy (DMD) is an X-linked recessive paediatric disorder that ultimately leads to progressive muscle degeneration. It has been known that cell-based therapies were used to promote muscle regeneration. The main purpose of this study was to investigate the effects of allogeneic Wharton jelly-derived mesenchymal stem cells therapy in Duchene muscular dystrophy.

Patients & Methods: Four ambulatory and five non-ambulatory male patients were assessed as having acceptance criteria. Gene expression and immunohistochemical analysis were performed for *dystrophin* gene expression. The fluorescent *in situ* hybridization method was used for detection of chimerism and donor–recipient compatibility. Complement dependent lymphocytotoxic crossmatch test and detection of panel reactive antigen were performed. All patients were treated with 2 × 106 cells/kg dose of allogeneic Wharton jelly derived mesenchymal stem cells via intra-arterial and intramuscular administration. Stability was maintained in patient follow up tests, which are respiratory capacity tests, cardiac measurements and muscle strength tests.

Results: The vastus intermedius muscle was observed in one patient with MRI. Chimerism was detected by fluorescent *in situ* hybridization and mean gene expression was increased to 3.3-fold. An increase in muscle strength measurements and pulmonary function tests was detected. Additionally, we observed two of nine patients with positive panel reactive antigen result.

Conclusion: All our procedures are well tolerated and we have not seen any application related complications so far. Our main purpose of this study was to investigate the effects of allogeneic mesenchymal stem cell therapy and determine its suitability and safety as a form of treatment in this untreatable disorder.

Biography

Alper İbrahim Dai, graduated from Istanbul University, School of Medicine. He completed his paediatric residency at Jackson Memorial Hospital, University of Miami / Florida and Children's hospital of West Virginia at Morgantown. As a fellowship he had 3 years of paediatric neurology at SUNY, Children's Hospital at Buffalo / New York. He had 1 year of EEG / Epilepsy fellowship at Vanderbilt University, Nashville / Tennessee. He has been working at Division of Pediatrics Neurology at Gaziantep University Hospital, Gaziantep / Turkey for 15 years. His major interests are, phase III clinical trial in anti-epileptic medications and phase I and II clinical trials in stem cell therapy in children with neurological problems..

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Varicella-zoster

Kasid Ahmed Nouri
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Varicella- zoster virus (VZV) is an exclusively human neurotropic, double-stranded DNA alpha herpesvirus, primary infection causes varicella (chickenpox), with a decline in VZV-specific cell-mediated immunity in elderly and immunocompromised individuals and VZV reactivates to cause *herpes zoster* (shingles) with its complications. *Varicella-zoster* virus infections can produce multiple disorders of the central and peripheral nervous system and often without rash. The most common complication of *herpes zoster* is post herpetic neuralgia, pain that persists months to years after rash resolved. *Herpes zoster* may cause serious chronic complications, including PHN, cerebral arteritis and *herpes zoster* ophthalmicus. The important of early diagnosis with a rapid virology verification and early prompt treatment with antiviral agents can lead to complete recovery; even in patients with protracted disease. *Herpes zoster* vaccination significantly reduces the incidence of both *Herpes zoster*, post herpetic neuralgia and other complications. Given the limitations of existing *zoster* therapies, the prevention of VZV infection has gained overriding importance. VZV vaccinations can reduce both the health and economic effects of *Herpes zoster*. Although relatively costly, vaccinations not only reduce the risk of infection but may also preserve health-related QOL in the geriatric population. With seven cases studies presentation there will be a high light on different *Varicella- zoster* clinical presentations.

Publications:

1. 1. N Nciri , A Yousafzai , A Safi, S Nawaz , S Kundanpally and K Nouri (2012)- Adult H1n1 A Viral cerebritis revealed by homonymous hemianopsia – Am. J Respir. Crit. Care Med. 185:A3180.
2. 2. Mohammed Abdulelah Mezaal, Kasid A Nouri, Shareefa Abdool, Khalid Al Safar and Ahmed S M Nadeem (2009) Cerebral Palsy in Adults Consequences of Non Progressive Pathology - Open Neurol. J. 3:24-26.
3. 5. Mohamed A Al-Zaidi and Kasid A Nouri (2002) – Guillain–Barré syndrome: Pattern of Muscle Weakness. Neurosciences (Riyadh) 7 (3): 176-178

Biography

Kasid Ahmed Nouri is an adjunct Clinical Assistant Professor of Neurology at Mohammed Bin Rashid University of Medicine and Health Sciences (MBRU) College of Medicine, Dubai, UAE. He is also working as a consultant neurologist at Mediclinic City and Welcare Hospitals, Dubai, UAE.

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Notes:

Silent brain infarction and metabolic syndrome in middle aged Egyptian ischemic stroke patients

Wael Osman M Amer, Mahmoud M Abdel Sayed, Tarek I Meneci, Sayed A El Zayat, Mohamed F Abdel Mouaty and **Mohamed Fathy Abd Alsalam**
Al Azhar University, Egypt

Objective: To investigate the relationship between silent brain infarcts and metabolic syndrome in middle aged patients with ischemic stroke.

Methods: We studied fifty middle aged patients between 40-59 years, (mean, 52.26 ± 5.29 years) with ischemic stroke who admitted to Al-Azhar University Hospitals or followed up in outpatient clinic. Metabolic syndrome was diagnosed according to the National Cholesterol Education Program Adult Treatment Panel III definition. Silent brain infarct was diagnosed using magnetic resonance imaging of the brain and without a history of corresponding neurologic symptoms or signs.

Results: Silent brain infarcts was diagnosed in 24(48%) patients (18 men and 6 women) while metabolic syndrome was diagnosed in 31(62%) patients of the 50 patients including (22 men and 9 women). There was a strong association between metabolic syndrome and silent brain infarction in middle aged subjects with ischemic stroke. Among metabolic syndrome components elevated blood pressure and impaired fasting glucose were strongly associated with the presence of silent lacunar infarction while hyper triglyceridemia, low high density lipoprotein cholesterol and large waist circumference were not significantly associated with silent brain infarction. Although, only elevated blood pressure and impaired fasting glucose were found to be significant components, subjects with a greater number of metabolic syndrome components showed more prevalent silent brain infarctions.

Conclusions: Metabolic syndrome was significantly associated with the prevalence of silent brain infarction in middle aged patients with ischemic stroke. Independent risk factors for silent brain infarctions include elevated blood pressure and impaired fasting glucose. The clustering of metabolic syndrome components tends to increase the prevalence of silent brain infarctions as there is a dose response relationship between the number of metabolic syndrome components and the risk of having silent brain infarction.

Biography

Wael Osman M Amer has completed his MD Al- Azhar University and Postdoctoral studies in Neuro intervention from School of Medicine, REIMS University, France. He is the Director of the stroke unit at Al-Azhar University School of Medicine. He has published more than 15 papers in reputed journals.

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Notes:

Giving a voice to neurological disorders in Sub-Saharan Africa

Chika Okwuolisa
Brain and Spine Foundation, Africa

It's a common knowledge that Africa has the least neurological services and African neurologists and neurosurgeons cannot match the rapid progress in the developed world. And this I write with extreme sadness. As the founder of The Brain and Spine Foundation Africa, it is no gain saying the fact I am coming from a background of diametric ramifications of health emergencies- ours is a harrowing experience and indeed, a sordid reality that no one can be proud of. Prominent among the challenges in the Nigerian health sector for example, is the escalation of various degrees of neurological emergencies. The world, especially European and American institutions/organizations must rise to this urgency, and bring their technological edges to bear on this very challenging bracket, vis-à-vis the deployment of alleviative fiscal policies. Brain and Spine Foundation, Africa is from the grassroots; we are close to these unfortunate ones who are going through agonies everyday, never as a result of any fault of theirs. We hear their cries everyday, we see their despairs, because the cost of treatment is usually beyond their reach or there is no Neuro-specialist available to attend to them. We render our helps which are usually grossly inadequate. Most importantly, we stay by their sides intentionally to be available for them, and serve as psychological reinforcements for their survival instincts. We watch them die sometimes owing to the lack of adequate facility for treatment and precarious volume of resources available to us, which often empty into paucity sooner or later. Neurological disorders as we all know are associated with high mortality, prolonged hospital stay and socioeconomic burden for the families. Unfortunately, in Sub-Saharan African countries, there's a huge disconnect between where disease is and where experts are. We need you, most especially those of African descents to come and help us make changes to our national policies that could help our people. We need your help in working to build up the resources and networks necessary to conduct clinical trials in Africa, and to create education and training programs for health care providers and researchers. We as a Charity Organization are creating awareness, educating the public, providing support and hope, but we also work with the health care structure to ensure there are providers and treatment.

Biography

Chika Okwuolisa is the Founder and Executive Director of Brain and Spine Foundation, Africa. Brain & Spine Foundation (BSFA) is an operational registered non-Governmental Organisation (NGO). It was established it following a first-hand traumatic experience during a harrowing pathological Brain event that has changed their lives forever. BSFA is founded to create awareness on the burden of Brain and Spinal conditions and all the variants there are of these in Nigeria and Africa. We also focus on ways to prevent, manage and reduce the burden of these illnesses which thrive on the general ignorance of individuals.

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Notes:

Alanine rich dipeptide repeat proteins sequester arginine rich dipeptide repeat proteins in a cellular model of *C9orf72*

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A hexanucleotide repeat expansion in the *C9orf72* gene is the most common genetic cause of frontotemporal dementia and motor neuron disease. Carriers present with both conditions concurrently, so they are considered a continuum. The RNA from the hexanucleotide expansion is translated by repeat associated non-ATG (RAN) translation; producing five dipeptide repeat (DPR) proteins. These are the alanine rich polyAP and polyGA, the arginine-rich polyGR and polyPR, and polyGP. Previous *in vitro* studies show that the arginine-rich DPRs are toxic because they localise to the nucleolus, causing nucleolar stress. However, preliminary research showed sequestration of polyGR by polyGA when they were co-expressed in HeLa cells, Drosophila and human neurons. Our aim was to determine whether alanine-rich DPR proteins sequester arginine-rich DPR proteins when the two are co-expressed *in vitro*. This was investigated using alternative coding sequences for the DPR proteins, cloned into mCherry-tagged plasmids. Next, two DPR proteins with different fluorescent tags were co-transfected into HeLa cells and the subcellular locations of the DPR proteins were visualised using immunofluorescence. Our results showed cytoplasmic co-localisation of the arginine-rich DPRs with polyGA. This was replicated 3 times in HeLa cells and once in SH-SY5Y cells. Co-transfection, but no colocalisation, was seen when two alanine-rich or two arginine-rich DPR proteins were co-expressed. Overall, our findings suggest that the arginine-rich DPRs are sequestered by polyGA in the cytoplasm, meaning they are unlikely to cause nucleolar stress. This demonstrates that single transfections of DPR proteins may not be a good model to study DPR protein function or toxicity.

Biography

Katherine Radcliffe conducted this original research whilst incalating and studying MRes Translational Medicine at the University of Manchester. She has just graduated from Manchester Medical School and is due to start as an FY1 doctor in August. This research earned her the best poster prize within her course and she has presented posters at multiple conferences including a European Congress whilst studying.

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Two homozygous *KIF1C* mutations in a Turkish family with cerebellar dysfunction and spastic paraparesis

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KIF1C mutation is genetic defect which is observed with hereditary spastic paraparesis and cerebellar dysfuntion. In this report, we analyzed the Turkish family who had spastic paraparesis and cerebellar dysfunction with KIFC mutation. There were paraparesis, ataxia, dysarthria, tremor in brothers whom parents were second degree relatives and asymptomatic. Father and mother's ages were 65 and 62 and brothers were 44, 42, 36. Their complaints appeared with tremor in their hands in childhood. In the following terms, the other complaints began to reveal. There were no point in family history. Patients' neurologic examination: their speech were dysarthric, biletaral dysmetria and dysdiadokokinezi in all. Intention tremor could be seen in all brothers' heads and extremities. The patient's, who were 44 years old, spastic paraparesis is more serious than the other two and he were walking by using one crutch. The other two brothers', who are 42 and 36 years old, spastic paraparesis were less serious and they could walk without using any crutch. General medical tests such as routine biochemistries, hemograms, hormones, B12, VDRL, HIV, vitamin A and E, thyroid hormones, plasma ceruloplasmin and copper were normal. In Cranial-Spinal MR, there were remarkable cortical and cerebellar atrophie. Electrocardiographies, electromyographies and odiographies were normal. They had adequate IQ scores, which were among 80-100 scores. In whole exome sequencing two variant mutation were identified in their *KIF1C* genes. The parents are heterozygote and brothers are homozygote. On the basis of clinical and genetical analyzies, autosomal recessive spastic paraparesis and ataxia were considered due to mutation in *KIF1C*.

Biography

Güllü Tarhan is a 26 year old and working as a Neurology resident in the Gaziosmanpasa Taksim Training and Research Hospital. She has completed Istanbul Faculty of Medicine two years ago.

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