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Late Breaking Oral Abstracts

1463
WFN15-1629
Late Breaking Abstract Session 1
Role of TNF-alpha-308G>A and IL-6 -174G/C polymorphisms in recurrent transient ischemic attacks

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Background: A transient ischemic attack (TIA) is a brief ischemic incident distinguished by rapid clinical improvement within 24 hours and no cerebral infarction.
Objective: To assess the role of proinflammatory cytokines specially TNF-α and IL-6 polymorphisms as clinical predictors of recurrent TIA and subsequent stroke.
Methods: one hundred and six participants (54 group 1 and 52 group 2) were enrolled with clinically resolved TIA and 34 (group 3) age-matched controls. DNA was extracted from blood samples of all subjects. Polymerase chain reaction for DNA amplification was done followed by digestion using NcoI and NlaIII restriction endonuclease enzymes for detection of promoter single nucleotide polymorphism (SNP) of TNFa-308G>A and IL-6 -174G/C respectively.
Results: Molecular analysis showed significant increase in TNFa-308G>A allele polymorphism in patients with high risk TIA (group 1) compared to both low risk (group 2) and control (group 3) groups [Odds ratio (95% confidence intervals): 3.3 (1.83–5.9), P = 0.0001 and 3.5 (1.85–6.79), P = 0.0001respectively] with significant increase of genotypes TNF-α − 308 AA [Odds ratio (95% confidence intervals): 10 (2.5- 3.2), p ≤ 0.05 and 8.5 (2.4- 30), p ≤ 0.05] was detected when compared to groups 2 and 3 respectively. IL6 allele polymorphism or genotypic distribution did not reveal any significance.
Conclusion: TNFa-308G>A but not IL-6 -174G/C SNP plays a significant role in evaluating and predicting recurrent TIA with subsequent high risk of actual stroke development and in this manner may contribute to primary stroke prevention.

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1465
WFN15-1644
Late Breaking Abstract Session 1
Computational analysis of single nucleotide polymorphisms in SCN1A gene of epilepsy, and implications in sodium voltage gated channel function

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Background: SCN1A is recognized as the most important epilepsy gene discovered to date. It encodes the alpha1 subunit of the voltage gated sodium channel.
Objectives: Single nucleotide polymorphisms (SNPs) are by far the most prevalent of all DNA sequence variations. Therefore this work focused on analysis of SNPs in coding and 3'untranslated regions (3' UTR).
Materials and methods: 1279 non synonymous SNPs in coding region of SCN1A gene were analyzed concerning degree of structural and functional impact in protein product, as predicted by SIFT and polyphen-2 server. Then protein modeling was performed according to each damaging SNP by using Raptor X and Chimera 1.10.1. PolymiRTS software was used to investigate SNPs in 3'UTR.
Results: Analysis with SIFT and polyphen-2 predicted 33 damaging SNPs out of total SNPs in coding region; nine of them are located in exon 30, and 20 SNPs were found in cytoplasm domains. These SNPs result in mutant amino acids which differ in their physiochemical properties from the wild type one. Among them 7 SNPs are located near highly conserved regions. Moreover, 14 of these SNPs affect the main activity of the protein. Analysis of 84 SNPs in 3' UTR resulted in no SNPs which could disrupt miRNA site.
Conclusion: The Large number of detected SNPs that could potentially affect SCN1A gene highlights the importance of such computational analysis, in order to contribute to the ability to recognize and diagnose epilepsy, and to find new treatment modalities.

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1466
WFN15-1655
Late Breaking Abstract Session 1
Clinico-radiological profile of childhood moyamoya disease - a study of 30 children from India

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Introduction: Childhood moyamoya disease, a vaso-occlusive disease, has myriad presentations. Early diagnosis is key to treatment and successful outcome.
Methods: 30 patients of childhood moyamoya disease diagnosed by MRI, MR-Angiography and DSA, were studied for various spectrum of clinico-radiological manifestations.
(Appropriate approval/consent taken)

0022-510X/$ – see front matter.

Results: Mean age: 6.71 yrs (6 months – 15 years); Female:Male ratio 1.15:1.

Clinically, headache, cognitive decline, bihemispherical TIAs, visual loss and radiologically, Boomerang sign, unilateral affection and posterior circulation involvement were the unusual presentations found in this study.

Conclusions: Knowledge of varied manifestations must be borne in mind for early diagnosis to achieve favorable prognosis of this potentially treatable entity.

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Table 1

Clinical Characteristcs	*Frequency*	*As Presenting Feature*
Ø Hemiparesis	22(73.33%)	22(73.33%)
Ø Recurrent TIAs	14(46.66%)	2(6.66%)
Ø Headache	12(40%)	2(6.66%)
Ø Seizures	8(26.66%)	1(3.33%)
Ø Cognitive Decline	2(6.66%)	1(3.33%)
Ø Bihemispherical TIA	2(6.66%)	1(3.33%)
Ø Visual Loss	1(3.33%)	1(3.33%)
Ø Recurrent stroke	12(40%)	

Table 2

MRI BRAIN	
A. Ischemic stroke	22(73.33%)
· Anterior circulation strokes	18(60%)
· Posterior circulation strokes	2(6.66%)
· Anterior + Posterior circulation strokes	2(6.66%)
B. Hemorrhagic stroke	1(3.33%)
C. Normal parenchyma(only abnormal flow voids)	7(23.33%)
ANGIOGRAPHY	
Ø Unilateral ICA	5(16.66%)
Ø Bilateral ICA with normal Posterior Circulation(PC)	21(70%)
Ø Bilateral ICA with PC involvement	4(13.33%)
Ø Isolated PC involvement	0

1467
WFN15-1720
Late Breaking Abstract Session 1

Electromyographic changes observed after combined treatment: autologous stem cell implant and intensive rehabilitation in patients with complete, chronic spinal cord injury

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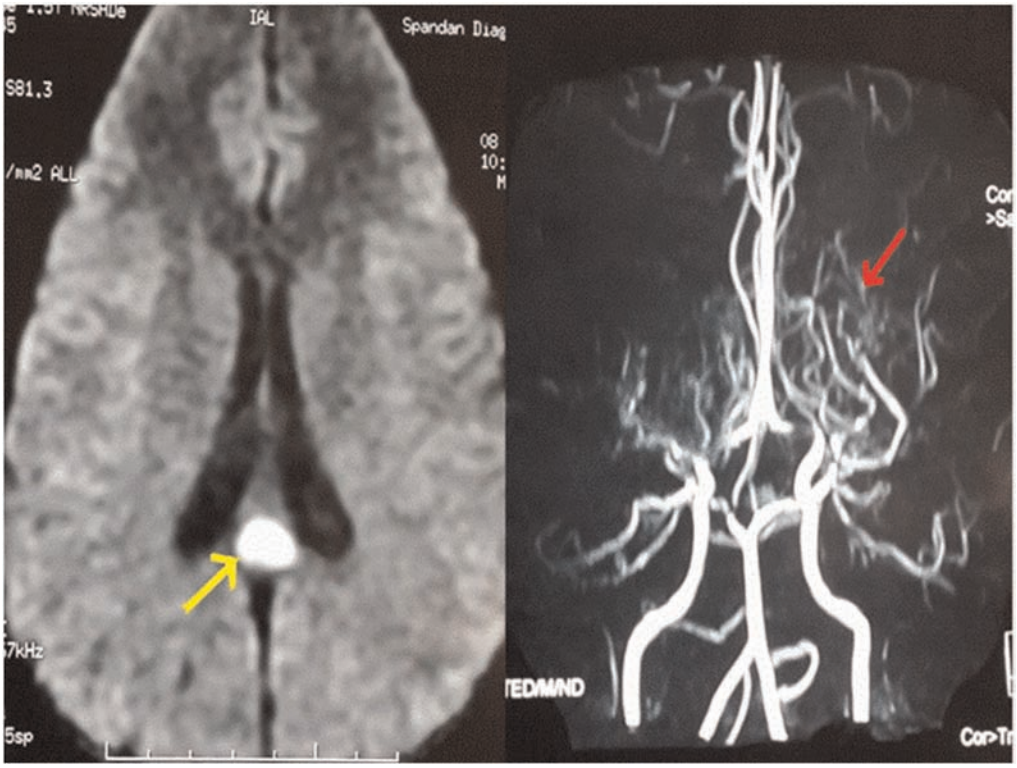
Background: Eight traumatic chronic spinal cord injury patients (ASIA A/Frankel A) received a combined treatment (**cell therapy:** endovascular implant with fat-derived autologous stem cells differentiated *in-vitro* from neural stem cells, and intensive, multidisciplinary **rehabilitation**), intending to recover the nervous functions lost due to the lesion. Electromyography was used as an objective method to assess the recovery of muscle electrical activity.

Objetive: Demonstrate through electromyography the recovery of muscles innervated by spinal roots below the lesion level.

Patients and methods: In June 2013, 8 patients with traumatic cSCI (ASIA A/Frankel A) 3/8 quadriplegic and 5/8 paraplegic began a combined treatment with cell therapy and rehabilitation. Before starting the treatment, none of them showed electrical activity in muscle groups innervated by nerve roots located 2 spinal levels below the lesion. Electromyography of the affected muscles was performed at 6, 12, and 24 months.

Results: After 24 months of treatment, the following changes were observed in 5/8 patients: onset of the response of motor unit potentials in muscles without previous electrical activity; a progressive increase in the voltage of motor unit potentials in the muscles explored and giant potentials (spinal potentials).

Conclusion: We confirmed the recovery of electrical activity in muscle groups affected by the lesion in patients with severe spinal cord injuries who received treatment. Electromyography is a sensitive and objective assessment method.



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1468
WFN15-1728
Late Breaking Abstract Session 1
Clinical and genetic features of patients with glutaric aciduria type I (GA1)

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Introduction: GA1 is a metabolic disorder produced by a defect on glutaryl CoA dehydrogenase (GCDH) enzyme, in the GCDH gene localized in 19p13.2 chromosome.
Objective: To analyze clinical manifestations, neurologic evolution, imaging characteristics and type of mutations found in children with diagnosis controlled in our service.
Materials and method: Retrospective-descriptive study and prospective analysis of 11 children diagnosed in our center in the last 17 years, with positive genetic studies.
Results: Of a total of 11 patients, six were male. Eight debuted with an encephalitis-like episode at a mean age of 9,9 months. The three remaining patients debuted with psychomotor delay (mean age 4 months) with two of them presenting an encephalitis-like crisis later. Three patients progressed with macrocephaly. One patient presented mild, two moderate and eight severe disability. Cerebral RM in acute episode showed basal ganglia and white matter compromise, bifrontotemporal atrophy, progressing to striatal atrophy. Residual enzymatic activity was deficient in four patients who were studied. Mutations found were heterozygous to R161Q/R402W, Y133H/R161Q, Y133H/R402W, V133/A385V and homozygous to R402W/R402W, A293T/A293T, Y113H/Y113H. No relationship was found between neurologic severity and specific genotype.
Conclusion: In our series, the most frequent presentation was an encephalitis-like episode. The most invalidating symptoms were extrapyramidal and neuroimages were distinctive. The homozygous and heterozygous mutation in Y113H and R402W are frequent in chilean population, being Y113H exclusive in this population. The biochemical genotype and phenotype did not predict clinical course. In conclusion, presymptomatic diagnosis of this affection allows an appropriate management with a favorable evolution.

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1469
WFN15-1745
Late Breaking Abstract Session 1
Clinical and genetic characterization of patients with myotonic dystrophy type 1 (DM1) at San Borja Arriarán Hospital

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Background: DM1 is a multisystemic, autosomal dominant disorder, caused by an unstable expansion of CTG repeats in DMPK gene, and has a wide clinical spectrum.
Objectives: To describe and to correlate clinical and genetic findings from patients with DM1 of a Pediatric Neuropsychiatry Service.
Patients and methods: 22 patients with DM1 older than 6 years of age, without history of neonatal asphyxia, were subjected to a prospective protocol consisting of review of medical records, interrogation, physical examination and non-invasive cardiological evaluation.
Results: 36.4% had congenital DM1, 54.5% infantile DM1, and 9,1% juvenile DM1. 72,7% were maternally inherited. 100% of the patients with congenital DM1 had maternal inheritance, and 100% of the patients with juvenile DM1 had paternal inheritance, with a statistically significant relationship. The average of CTG repeats was 1300 in congenital DM1, 800 in infantile DM1 and 600 in juvenile DM1, with statistically significant relationship between larger expansions and early presentation. The first clinical manifestation was hypotonic syndrome in 36.6%, delayed language development in 31.8% and clubfeet in 22.7%. The average age of the sample was 16 years. All patients had muscle symptoms. 81.8% had cardiac arrhythmia, 33.3% bundle-branch block, and 22.7% atrioventricular block, all were asymptomatic. 50% of patients reported encopresis, 22.7% constipation and 3 patients had cataracts.
Conclusion: Early onset of the disease was related to larger expansions of CTG repeats and to maternal inheritance. The findings were multisystemic, all had muscle symptoms, most had heart rhythm disturbances, and half had gastrointestinal symptoms.

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1471
WFN15-1625
Late Breaking Abstract Session 2
Stroke epidemiology in a private tertiary hospital from Rio de Janeiro

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Introduction: Stroke is a leading cause of morbidity and mortality worldwide. Little information is available on the stroke epidemiology of patients in Rio de Janeiro. The disease epidemiology is an important tool to aid health care planning. Our objective was to describe risk factors, management in the acute setting, and outcome of our cohort.
Methods: All stroke patients data from Copa D'Or hospital (private tertiary hospital) have been collected prospectively since March 2012. The data was analyzed retrospectively from the data bank.
Results: Up to now we evaluated 193 patients (mean age 69; ±20,2 years; 55,4% females). Ischemic stroke was the most frequent subtype (84,4%), followed by intraparenchymal hemorrhage (8%), and subarachnoid hemorrhage (6%). Hypertension was the most common risk factor (66%). Twenty-three percent (23%) of our patients received intravenous thrombolysis. Time from symptoms onset to hospital admission was the leading contra-indication for IV rt-PA. The frequency of modified Rankin Scale ≤ 2 at discharge was 61,1%.
Conclusions: The aim of this study was the epidemiological data from our cohort. The outcome described and the frequency of IV thrombolysis is greater than other reports in Brazil. However, we are a private institution in a big and populated city of the state.

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1473
WFN15-1697
Late Breaking Abstract Session 2
Social anxiety level in adult patients with epilepsy and their first-degree cohabiting relatives

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Background: Epilepsy affects not only the patient, but also, it affects the cohabiting relatives of the patient to various degrees.
Objective: To investigate social anxiety levels in patients with epilepsy and their cohabiting relatives.
Patients and methods: State and trait anxiety, depression, and social fear and avoidance levels are investigated in 48 adult patients with epilepsy and 48 family members, compared with 43 healthy control subjects using the Beck Anxiety and Depression Inventories, State-Trait and Liebowitz Social Anxiety Scale.
Results: The results showed that the patients with epilepsy and their first-degree relatives had higher levels of depression, state and trait anxiety, and avoidance compared with the healthy subjects. The mothers had the highest level of depression and anxiety.
Conclusion: The patients with epilepsy and their cohabiting relatives have higher social anxiety levels, and if these problems could be recognized and treated they could be reintegrated into society.

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1474
WFN15-1730
Late Breaking Abstract Session 2
Broad-spectrum frequency analysis of seizures of intracranial EEG in lesional and non-lesional pharamcoresistant epilepsy

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Background: Pharmacoresistant epilepsy often treated with surgical removal of the epileptogenic zone (EZ). Recently, there has be interest in using intracranial EEG (iEEG) for high-frequency oscillation (HFO) and infra-slow activity (ISA) with conventional frequency activity (CFA), though effects of lesion are unknown.
Objective: To evaluate difference in the rate and spatial distribution of CFA, HFO and ISA between lesional (L, lesion identified on MRI/pathology) and non-lesional epilepsy (NL).
Patients and methods: 17 patients (L = 10,NL = 7); who underwent iEEG were selected. Ten sec EEG sample was included from seizure onset (sustained rhythmic EEG changes associated with habitual seizure) for CFA, with preceding 2 min included for HFO and ISA. Maximum 5 seizures per patient were included. iEEG was reviewed under following setting: CFA (1-70Hz,30mm/sec), HFO (≥70Hz,60mm/sec) HFO and ISA (≤0.01Hz, with deflection ≥0.5 mV,10 mm/sec). Analysis was performed for presence of HFO and ISA and their spatial distribution using chi square statistic.
Results: 57 seizures (L = 35) were analyzed. Both HFO (NL = 83%, L = 74%, p = 0.730) and ISA (NL = 82%, L = 54%, p = 0.0474) were more common in NL group, though only ISA was significant. Analysis of spatial distribution of various frequency bands revealed CFA-HFO co-occur more frequently (25%) than HFO-ISA (20%), with CFA-ISA co-occurring in 10%. When analyzed between groups (NL vs L), CFA and HFO co-occur more frequently in NL (31% vs 22%,p = 0.001), while there was no difference when CFA compared to ISA (9% vs 11%, p = 0.352).
Conclusion: Broad-spectrum frequency analysis of iEEG shows that HFO and ISA is present in >80% seizures in NL. In the leisonal group, ISA was less common. Though majority of HFO zone doesn't overlap EZ defined by CFA, it tends to do so more often in NL group. Similar difference is not found for ISA.

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1475
WFN15-1742
Late Breaking Abstract Session 2
Characterization of cognitive disorders and neuroimaging of patients with myotonic dystrophy type 1 (DM1) from the San Borja Arriarán Hospital

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Background: DM1 is an autosomal dominant multisystem disorder. The clinical spectrum is broad and includes central nervous system abnormalities.
Objective: characterize the cognitive impairment and brain magnetic resonance imaging (MRI) of DM1 patients, and to correlate them with the form of inheritance and CTG repeats.
Patients and methods: 20 patients with DM1 over 6 years old, without history of neonatal asphyxia, were subjected to a prospective protocol consisting of review of medical records, assessment of cognitive function and MRI of the brain.
Results: 40% had congenital DM1, 50% infantile DM1, and 10% juvenile DM1. The average age was 15.6 years. 30% of the sample had mild intellectual disability (ID), 30% moderate ID, and 40% had IQ higher than 80. 83% of patients with moderate ID had congenital DM1. Juvenile DM1 patients had IQ higher than 80. Negative correlation was observed between the CTG expansion and IQ, which was statistically significant. 90% presented abnormalities on brain MRI. 30% had enlarged subarachnoid spaces, 50% ventricular enlargement, 45% white matter hyperintensities and 45% corpus callosum alterations. White matter abnormalities were located in frontal (100%) and parietal lobes (22%). The MRI abnormalities were more frequent in the group with maternal inheritance, without observing statistically significant association. All normal MRI were from patients with paternal inheritance.
Conclusion: More than half of patients had some degree of ID and a lower IQ was related to larger expansions of CTG repeats. MRI abnormalities occurred in most patients and only patients with paternal inheritance had normal MRI.

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1476
WFN15-1743
Late Breaking Abstract Session 2
Central nervous system hypomyelination related to PLP1 defects: clinical and imaging description

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Background: The proteolipid protein 1 (PLP1) gene encodes the two major proteins of the central nervous system (CNS) myelin: PLP and DM20. Aberrations in PLP1 gene result in altered CNS myelination and manifest as Pelizaeus-Merzbacher disease (PMD) or spastic paraparesis type 2 (SPG2).
Objective: To identify patients with a confirmed genetic defect in PLP1 and to characterize the phenotype and neuroimaging by mutation.
Material and methods: A retrospective/prospective study, analysis of clinical data and images and their relationship with the genetic defect in patients with impaired PLP synthesis.
Results: 19 patients. (I) 16/19 corresponded to PMD, all affected individuals and their mothers with PLP1 gene duplication. 10 with family history of PMD. Average age of symptoms onset was 3 months

(1–8 months). Most frequent initial symptoms were delay in motor development, nystagmus, head tremor. Other clinical signs were global development delay, cerebellar syndrome, bilateral pyramidal syndrome, dystonia. All neuroimaging showed diffuse hypomyelination. (II) 3/19 corresponded to SPG2, the study confirmed PLP1 gene mutation c.388C > T (p.His130Tyr) exon 3B in patient and mother. A 6 year old boy began at 7 months nystagmus, head tremor, motor development delayed, spastic paraparesis. Neuroimaging showed incomplete myelination regions. 2 cousins on mother's side had spastic paraparesis.

Conclusion: In our series, patients with PLP1 gene duplication exhibit PMD with symptoms and CNS´s hypomyelination more diffuse and severe. Those with a mutation in the gene expressed SPG2.

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1478
WFN15-1771
Late Breaking Abstract Session 2
Frequency of epilepsy in patients with neurocysticercosis, in the Service Neurology and Neurosurgery Mother-Child Hospital

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Abstract

Cysticercosis of the central nervous system is the most important of human neurological diseases of parasitic origin. It generates considerable morbidity, is known to be one of the major causes of epilepsy, with serious social, physical and psychological consequences.

Objective: To assess the frequency of epilepsy in patients with neurocysticercosis, treated at the Mother and Child Hospital.

Type of study: Study descriptive. Study Area: Neurology and Neurosurgery Department of the Mother and Child Hospital the city of La Paz – Bolivia.

Subjects: work was done with the total of the universe, 352 patients diagnosed with epilepsy treated in the during the period from 2007 to 2010.

An instrument to collect: Workbook statistics and medical histories.

Results: Of the total 115 patients had such as base diagnosis neurocysticercosis in 43 cases (37%) were asymptomatic patients and in 72 cases (63%) patients had epilepsy.

Conclusion: Of 352 patients diagnosed with epilepsy in 72 cases (20%) epilepsy was secondary to neurocysticercosis, followed by 31 cases (9%) secondary epilepsy to AVC.

Key words: Neurocysticercosis and epilepsy.

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1479
WFN15-1661
Late Breaking Abstract Session 3
Early ultra-high-dose methylcobalamin treatment prolongs survival in amyotrophic lateral sclerosis patients

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Background: Amyotrophic lateral sclerosis (ALS) is an intractable neurodegenerative disease lacking effective treatment, except for riluzole, which modestly prolongs survival with little functional improvement. Ultra-high dose methylcobalamin is neuroprotective and a potential candidate for ALS therapy. We conducted a randomized, double-blind, placebo-controlled, phase 2/3 clinical trial to evaluate the efficacy and safety of ultra-high dose methylcobalamin in ALS patients and its efficacy with respect to disease duration before entry.

Methods: Patients with ALS (El Escorial definite or probable; laboratory-supported probable; duration ≤36 months) were randomly assigned to placebo or 25 or 50 mg of intramuscular methylcobalamin groups. Primary endpoints were time to primary events (death or ventilation support) and change in ALSFRS-R score from baseline to week 182. Efficacy was also evaluated in the patient subgroup with an earlier diagnosis (duration ≤12 months) using post-hoc analyses. Adverse events were recorded to evaluate safety.

Results: Significant differences were not detected in either primary endpoint (minimal P value = 0.087). However, post-hoc analyses of patients with an earlier diagnosis demonstrated a significant dose–response prolongation in the time to a primary event (P linear and saturated = 0.01 for both) and an inhibition of the decrease in the ALSFRS-R score (P linear and saturated = 0.003 and 0.01, respectively). The incidence of treatment-related adverse events was similar and low in all groups.

Conclusions: Ultra-high-dose methylcobalamin administration significantly prolongs survival and inhibits functional deterioration in patients with ALS without major adverse effects when treatment is started early (≤12 months from symptom onset).

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1480
WFN15-1664
Late Breaking Abstract Session 3
Association between phosphodiesterase 4D (PDE4D) gene polymorphism and ischemic stroke in North Indian population: a case–control study

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Background: Stroke is a multi-factorial disease and is influenced by both genetic and environmental factors.

Objective: The aim of this study was to investigate the association of SNP 83 (rs966221) of PDE4D gene with the ischemic stroke risk in North Indian Population.

Methods: In this study, 250 patients and 250 age-and sex-matched controls were recruited from Outpatient Department and Neurology ward of All India Institute of Medical Sciences (A.I.I.M.S.), New Delhi. The study was approved by Department of Biotechnology (DBT), New Delhi, India. Genotyping was performed by using Polymerase chain reaction– Restriction fragment length polymorphism (PCR-RFLP) method. PCR results were confirmed by DNA sequencing. Frequency distribution of genotypes and alleles were compared between cases and controls by using conditional logistic regression.

Results: Hypertension, Diabetes, Dyslipidaemia, Low Economic Status and Family Stroke History were found to be independent risk factors for ischemic stroke risk. Multivariate logistic regression analysis suggested an independent association between PDE4D 83 gene polymorphism and risk of ischemic stroke under dominant model of inheritance (OR, 1.59; [95%

CI, 1.02 to 2.50]; $P = 0.04$). Subgroup analysis was done as per TOAST classification and independent association was found with Large Vascular Disease (OR, 2.73; [95% CI, 1.16 to 6.39]; $P = 0.02$). All the observed genotype frequencies were in Hardy- Weinberg equilibrium in both cases and controls.
Conclusion: The present study suggests that SNP 83 of PDE4D gene is an independent risk factor of ischemic stroke risk in the North Indian population. Further large prospective studies are required to confirm these findings.

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1481
WFN15-1668
Late Breaking Abstract Session 3
Association of transforming growth factor-β1 gene C509T, G800A and T869C polymorphisms with ischemic stroke in North Indian population: a case–control study

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Background: Stroke is a multi-factorial polygenic disease which is influenced by both genetic and environmental factors. Transforming growth factor-β1 (TGF-β1) is a multifunctional cytokine involved in inflammation and pathogenesis of atherosclerosis. There is limited information on the relation between variations within the TGF-β1 gene polymorphisms and risk of ischemic stroke (IS).
Objective: The aim of this present study is to investigate the association of the TGF-β1 gene (C-509 T, G800A and T869C) polymorphisms with the risk of IS in North Indian population.
Methods: In a hospital based case control study, patients with IS and control subjects were recruited from inward and outward of Neurology department, All India Institute of Medical Sciences, New Delhi. Genotyping was performed by using SnaPshot Method. Stroke was classified using Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification. Frequency distributions of genotypes and alleles were compared between cases and controls using multivariate logistic regression.
Results: In this study, 250 IS patients and 250 age-and sex-matched control subjects were recruited. Mean age of cases and controls were 52.8 ± 12.5 and 50.9 ± 12.7 years respectively. Multivariate logistic regression analysis showed significant association between TGF-β1 gene polymorphisms with the risk of IS; for C509T (Odds ratio [OR], 2.19; 95% CI, 1.25 to 3.83), for G800A (OR, 4.43; 95% CI, 2.10 to 9.32) and for T869C (OR, 2.63; 95% CI, 1.52 to 4.54) under dominant model of inheritance.
Conclusions: Our findings suggest that TGF-β1 gene polymorphisms are significantly associated with the risk of IS in a North Indian population.

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1482
WFN15-1690
Late Breaking Abstract Session 3
Association between CYP 450 gene polymorphism and ischemic stroke in North Indian population: a case control study

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Background: Stroke is a multifactorial disease. Its etiology is diverse, and is affected by both genetic and environmental factors.
Objective: The purpose of this case control study was to determine the relationship of Cytochrome P450 (CYP4F2) G1347A polymorphism with ischemic stroke in North Indian Population.
Methods: In this study, 250 patients and 250 age-and sex-matched controls were recruited from Outpatient Department and Neurology ward of All India Institute of Medical Sciences, New Delhi. Genotyping was performed by using Polymerase chain reaction–Restriction fragment length polymorphism (PCR-RFLP) method. PCR results were confirmed by DNA sequencing. Frequency distributions of genotypes and alleles were compared between cases and control by using conditional logistic regression.
Results: Hypertension, Diabetes, Dyslipidemia, Low Economic Status and Family History of Stroke were found to be an independent risk factor for Ischemic stroke. Mean age of cases and controls were 52.83 ± 12.59 and 50.97 ± 12.70. Multivariate logistic regression analysis showed independent association between CYP4F2 G1347A polymorphism and ischemic stroke under dominant model of inheritance (OR, 2.05; 95% CI, 1.18 to 3.56; $P = 0.01$) but not under recessive model of inheritance (OR, 1.72; 95% CI, 0.84 to 3.49; $P = 0.13$). All the observed genotype frequencies were in Hardy- Weinberg equilibrium in both cases and controls.
Conclusion: The findings of present study suggest that polymorphism in G1347A position of CYP450 gene might be a risk factor for ischemic stroke in North Indian population. Further studies are required to confirm these findings.

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1484
WFN15-1704
Late Breaking Abstract Session 3
Clinical presentation, epidemiology, treatment and outcome of nonconvulsive status epilepticus: a 3- year prospective, hospital based study

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Background: This study has been made possible by a grant from Qatar National Research Fund. An IRB approval from our Institution has been obtained. Continuous EEG monitoring (cEEG) remains an essential tool in detecting Nonconvulsive Status Epilepticus (NCSE) in patients with Altered Mental Status (AMS).8 to 48% of ICU patients may have NCSE which may be fatal.
Objective: Report the prevalence, describe the clinical presentation, etiology, treatment and outcome of NCSE.
Methods: This is a 3-year, prospective, hospital based study involving patients admitted to Hamad Hospital, Doha, Qatar. cEEG was performed in all patients with AMS.
Results: Patients with AMS is 240. Patients with clinical/EMG findings consistent with NCSE is 95 (m54,f41). Number of controls (in which cEEG was not consistent with NCSE) is 145 (m77,f68). Rate of occurrence of NCSE in patients with AMS is 40%.24 patients with NCSE (25,3%)and 27 patients (18,6%)in the control group died ($p < 0.22$). NCSE group was younger ($41,9 \pm 23$ vs $52,3 \pm 15,7$ys,$p < 0.001$),had more previous seizures (20 vs 4,$p < 0.001$)and a longer hospital stay ($15,2 \pm 7,7$ vs $12,7 \pm 5,5$ ys, $p < 0.008$). Complete recovery occurred in 47 patients

with NCSE (49%),and in 90 (62%) in the control group ($p < 0.01$). Death was more often in comatose NCSE compared to NCSE proper [15(45,5%) vs 9 (14,5%),$p < 0.002$], and with the control group [15(45,5%) vs 27(18,6%),$p < 0.003$]. Among NCSE group, 21 patients (31%) presented refractory NCSE: 12 patients (58%) survived; 9 patients (43%) died.
Conclusion: This is the first, prospective study reporting a high number and prevalence of NCSE in the Middle East and North African (MENA) countries. NCSE patients did not perform better than the controls. NCSE is a serious emergency condition and should be treated promptly.

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1485
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Late Breaking Abstract Session 3
Glial fibrillary acidic protein (GFAP) plasma levels distinguish intracerebral hemorrhage from cerebral ischemia in the early phase of acute stroke

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Background: Recent studies suggested that GFAP plasma concentrations distinguish between intracerebral hemorrhage (ICH) and ischemic stroke (IS) within the first hours after symptom onset. This prospective multicenter study validated GFAP in a broader patient cohort and assessed the relationship between GFAP release and bleeding size and localization.
Methods: We included patients suspected of having acute stroke who were admitted to 10 stroke centers in Germany within a time window of 6 hours after symptom onset. Patients had to have a persistent neurological deficit with a NIHSS score of at least 4 points. A blood sample was withdrawn at hospital admission, and GFAP plasma levels were measured by an electrochemiluminometric immunoassay. The primary endpoint was the final diagnosis established at hospital discharge (classified as ICH, IS or stroke mimic).
Results: The study included 214 patients (45 with ICH, 157 with IS, 12 stroke mimic). GFAP concentrations were significantly increased in ICH patients compared to IS patients [median (interquartile range) 0.2 ng/ml (0.0-3.3) vs. 0.0 ng/ml (0.0-0.0, $p < 0.001$). Diagnostic accuracy of GFAP for differentiating ICH from IS and stroke mimics was high [area under the curve 0.873 (95% CI 0.802-0.943), $p < 0.001$]. A GFAP cutoff of 0.03 ng/ml provided a sensitivity of 77.8% and a specificity of 94.3% for differentiating ICH from IS and stroke mimics. GFAP levels correlated with ICH volume.
Conclusion: Plasma GFAP has the potential to function as a biomarker in patients with acute stroke. Small ICH may be missed due to little tissue destruction. Future studies may evaluate GFAP together with other promising candidate biomarkers, in order to increase diagnostic accuracy for the development of valid point-of-care-systems.

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1486
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Late Breaking Abstract Session 3
Pet imaging of myelination in the central nervous system

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Objectives: Destruction or changes associated with myelin in the CNS play a key role in the pathogenesis of multiple sclerosis (MS) and related neurodegenerative disorders. Novel therapies are currently under development to prevent demyelination and promote remyelination. For efficacious evaluation of these myelin-targeted therapies, a major challenge is assessing and quantifying changes in myelin content *in vivo*. To meet this unmet need, we have developed a PET probe ([^{11}C]-MeDAS) that readily enters the CNS and selectively binds to myelin membranes. Here we reported its application in image-guided myelin repair therapies in an animal model of MS.
Methods: 1) Lysolecithin (LPC) was administered to the brain and spinal cord via stereotactic injection; 2) The LPC rats were treated with a mesenchymal stem cell-based hepatocyte growth factor (HGF) to promote remyelination. 3) The time course of myelin changes were quantitatively monitored by longitudinal microPET imaging in the brain and spinal cord and correlated with histological analysis.
Results: 1) Focal demyelination in the brain and spinal cord was induced by LPC; 2) HGF treatment showed significant remyelination; 3) Quantitative imaging analysis showed that the uptake and retention of [^{11}C] MeDAS correlated well with the level of demyelination/remyelination in the brain and spinal cord.
Conclusions: [^{11}C]MeDAS-PET is a promising imaging marker to monitor the changes in myelination in vivo, which is capable of monitoring myelin-targeted drug effects.

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1487
WFN15-1714
Movement Disorders 3
MT-1303, a novel selective s1p1 receptor modulator in RRMS - results of a placebo controlled, double blind phase II trial (momentum)

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Background: We assessed efficacy and safety of three doses of MT-1303, a novel selective sphingosine 1-phosphate 1 (S1P1) receptor modulator in patients with RRMS.
Methods: 415 patients with RRMS were randomized to once daily oral MT-1303 0 · 1, 0 · 2, or 0 · 4 mg, or placebo for 24 weeks. Primary endpoint was the total number of gadolinium (Gd)-enhanced T1-weighted lesions on monthly brain MRI scans from Weeks 8 to 24. Secondary endpoints included the total number of Gd-enhanced lesions (Weeks 4 to 24), new or enlarging T2-weighted lesions, percent change in brain volume, and annualised relapse rate (ARR). Safety assessments included intensive Holter assessments was also evaluated.
Findings: The mean total number of Gd-enhanced T1-weighted lesions at Weeks 8 to 24 was lower in the MT-1303 0 · 1 mg (5 · 5; estimated incident rate ratio [EIRR] 0 · 53; $p = 0 · 008$), 0 · 2 mg (2 · 3; EIRR 0 · 39; $p < 0 · 001$) and 0 · 4 mg (1 · 7; EIRR 0 · 23; $p < 0 · 001$) groups compared with placebo (8 · 3). Dose-dependent reductions were also observed in the total number of Gd-enhanced T1-weighted lesions and of new or enlarging T2-weighted lesions at Weeks 4 to 24. In the 0 · 2 and 0 · 4 mg doses less change of grey matter volume occurred ($p = 0.013$ and $p = 0.002$, respectively). MT-1303 0 · 4 mg reduced ARR by 82% (0 · 10; EIRR 0 · 18; $p = 0 · 005$) compared with placebo (0 · 44). The incidence of treatment-emergent adverse events (AEs), including infections and cardiac disorders, was comparable between treatment groups; the

most common AEs were headache and nasopharyngitis. No serious AE was reported for more than one patient in any group. No clinically significant heart rate reductions were observed at any MT-1303 dose. **Interpretation:** The favourable efficacy and safety profile of MT-1303 warrants further investigation in large-scale phase III studies in the RRMS population.

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1488
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Pain 1
In vivo confocal microscopy in patients with symptomatic diabetic polyneuropathy compared to controls

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Diabetic neuropathy (DN) is a common clinical condition. The currently recommended diagnostic tests have low sensitivity, such as electromyography or invasive tests, such as skin biopsy. Additional researches of new techniques have been developed in order to identify the early involvement of the peripheral nerve. With the advent of corneal confocal microscopy (CCM) in vivo, it was observed a reduction of corneal innervation in patients with DN. We compared the morphological changes of the sub-basal corneal epithelial plexus through the in vivo CCM in 35 diabetic patients with symptomatic distal symmetric polyneuropathy (DSP), compared to 55 control subjects. Furthermore, we sought to determine a pattern of change between severity stages of DSP, comparing clinical, laboratory, and nerve conduction (NC) variables. Differences between control and diabetic groups were observed for the following variables: age (44.9 ± 13.24 vs 57.02 ± 10.4, p-value < 0.001), fiber density (29.7 ± 10.2 vs 16.6 ± 10.2, p-value < 0.001), number of fibers (4.76 ± 1.30 vs 3.14 ± 1.63, p-value < 0.001), number of Langerhans cells (4.64 ± 8.05 vs 7.49 ± 10.3, p-value $= 0.035$), tortuosity (p-value < 0.05) and thickness (p-value < 0.05). Furthermore, inverse relationship was found between fiber density and age (p-value <0.01) and fiber density and clinical level (p-value <0.05). We also observed a positive relationship between conduction velocity and peroneal nerve fiber density (p-value < 0.05). We therefore conclude that the CCM is a fast, noninvasive and reproducible method for diagnosis, staging and monitoring of diabetic DSP.

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1489
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Pain 2
Contact heat evoked potentials (CHEPs) in painful autoimmune neuropathy (pain)

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CHEPs latency and amplitude is being considered of clinical utility for the assessment of neuropathic pain. It has been recently reported that prolonged latency and decreased CHEPs amplitude correlates with the intraepidermal nerve fiber density observed in skin biopsy performed in the area involved in the neuropathic symptoms. Painful auto-immune neuropathy (PAiN) encompass a painful clinical condition presenting with neuropathic painful symptoms, multifocal or nearly symmetrical (polyneuropathy pattern), involving almost exclusively A-delta and C small sensory fibers of cryptogenic nature. Conventional electrodiagnostic tests are unremarkable. In these cases extensive laboratory investigation do not disclose any specific underlying disease, and there is no family history. Increased protein levels can be seen in CSF examination and there is an inflammatory process with demyelinating features in skin or fascicular sensory nerve biopsy. These patients have a dramatic reduction of painful symptoms with immunoglobulin, not obtained with current neuropathic pain treatments. Eight patients with PAiN clinical and laboratorial characteristics were included and underwent CHEPs examination, according to standard technique. There were 5 women, and the median age was 38.7 years old (range, 18–79). All patients complained of burning pain.. CHEPs latencies obtained in Cz were absent or prolonged in the lower limbs in all patients, but asymmetric in four. CHEPs potentials amplitudes were not representatively reduced in our cases. All patients responded to intravenous immunoglobulin treatment, with complete resolution of neuropathic pain in six. CHEPS seem to be a useful and non-invasive tool in the diagnosis support of small fiber compromise in PAiN cases.

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