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RESEARCH ARTICLE

Highlights of 2nd Taiwan International Congress of Parkinson's disease and Movement Disorders, Taipei, Taiwan (March 28-29, 2015): Parkinson's disease and movement disorder- An update

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Abstract:
2nd Taiwan International Congress of Parkinson's disease and Movement disorders was held at Taipei, Taiwan from 28-29th March 2015. Present report highlights various cellular and molecular aspects on the Parkinson's disease and related movement disorders. In brief, report provides a comprehensive overview to understand the etiopathogenesis of PD and related movement disorders. Neuroscientists and young researchers from all around the world presented their recent advancement with a wide range of scopes of neurodegeneration research, including new genes, molecular pathways, animal models, and potential therapeutics. Present conference report is mix blend of various deliberations consists of plenary lectures, symposia and oral and poster presentations.

Keywords:

Introduction

2nd Taiwan International Congress of Parkinson's disease and Movement disorders was held at Taipei, Taiwan from 28th to 29th March, 2015. It was organised by the Taiwan Movement Disorder Society, Taiwan. The congress was preceded by a one day symposium on movement disorders, held on 27th March 2015 at Howard civil service international house, Taiwan. This symposium was organized under the aegis of the 2nd Taiwan International Congress of Parkinson's disease and Movement disorders. The meeting was opened by the speech of Dr. Juei-Jueng LIN, chair of the local organizing committee and Dr. Ruey-Meei WU, chair of the scientific committee. Their speech highlight the overview of the entire meeting program, which consists of one day symposium, followed by two days congress, poster session and ended with the unforgettable valedictory ceremony. The aim of this conference was to bring together leading experts in the field of Parkinson's disease (PD) and movement disorders to provide a comprehensive overview of the molecular mechanisms and genetics underlying this disease with the intent of providing a clear direction for future research into early diagnosis and therapeutic interventions. Present report briefly addresses cellular and molecular aspects, recent developments and future challenges in the field of PD and movement disorders.

The congress was started with pre-congress symposium where back-to-back plenary lectures were delivered by renowned neuroscientists. Dr. Shen-Yang LIM from University of Malaya, Kuala Lumpur, Malaysia discussed the journey of levodopa in the treatment of PD and the other relevant aspects including the impact of age, progression of neuropathology, biomarkers and new technologies, along with the possible role of the gut in the disease progression. He also emphasized not dopa-responsive features which develop especially after a long duration of disease, such as axial motor disability and dementia.

Further, Dr. Eng-King TAN from National Neuroscience Institute, Singapore described the selection of the most appropriate drugs, dosing and the use of combination therapy that are believed to reduce or delay the onset of motor complications and other adverse events. Dr. Irene Litvan from University of California, USA reviewed the clinicopathological correlations as well as recently developed new clinical diagnostic criteria for corticobasal degeneration (CBD), which includes 4 recognized CBD phenotypes: corticobasal syndrome, frontal behavioral-spatial syndrome, nonfluent variant of primary progressive aphasia and progressive supra-nuclear palsy syndrome. However, she elaborated diagnostic criteria that need further validation to better recognize the various presentations of CBD. Further, Dr. Shoji TSUJI, University of Tokyo, Japan expanded understanding of the multiple system atrophy (MSA), which involves synucleinopathies. He classified MSA into MSA-C (characterized by cerebellar ataxia) and MSA-P (characterized by Parkinsonism). He also described that former one is more prevalent in Japanese population whereas the latter one is found to be more prevalent in Europe and North America. Dr. Pramod Pal from National Institute Of Mental Health and Neuro Sciences, India pointed out the second most common neurodegenerative movement disorder i.e. progressive supranuclear palsy (PSP) accounting for 1.4-3.9% of PD cases. Dr. Pal described the accuracy of clinical diagnosis at first evaluation is as low as 19% and the mean lag time between principal symptom onset and a clinical diagnosis of PSP is 2.8 and 4.8 years. He emphasized on clinical diagnostic criteria of National Institute of Neurological disorders and stroke (NINDS) for PSP (1), which has a high positive predictive value for a clinical diagnosis of PSP. On the basis of diagnostic criteria PSP can be described into several variants of PSP, viz. PSP-P, PSP-CBS, PSP-PAGE, PSP-PNFA and PSP-C. Dr. Pal also pointed out the importance of imaging techniques like MRI, VBM, DTI and 18F-FDG PET in the diagnosis of PSP.

On the first day of the congress, Prof. Ruey-Meei WU, Professor at Department of Neurology, National Taiwan University Hospital, College of Medicine, Taiwan delivered a talk on the perspectives of genetics in hereditary PD (hPD). She reviewed about the biological pathways involved in hPD and the future direction for genetic study of hPD and translational medicine for new drugs discovery of PD (2). Next, Dr. Carolyn SUE, Professor Medicine, Northern Clinical School Kolling Institute of Medical Research, Sydney Medical School, Australia elaborated on pathology, progression and heterogeneity of PD. She discussed two distinct but interconnected disease pathways: a) disturbed mitochondrial function and; b) abnormal protein degradation. The lecture by Shu-Leong HO, Centre on Behavioral Health, Faculty of Social Sciences Department of Medicine, University of Hong Kong, China discussed on the role of mitochondrial and synaptic dysfunction in the pathogenesis of PD. He discussed the importance of neuronal uncoupling proteins (UCPs) in maintaining mitochondrial function and reducing oxidative stress. He also, presented a very interesting work where Leucine-rich repeat kinase 2 (LRRK2) knock-in mice found to be more susceptible to synaptic dopamine depletion and motor deficits associated with impaired dopamine uptake induced by reserpine. The higher susceptibility to reserpine in this knockin mouse indicates very early striatal synaptic dysfunction before any structural abnormalities could possibly be observed. Further, they found significant more toxic alpha synuclein aggregates in striatal presynaptic nerve terminals of these dopaminergic neurons of knock-in mice compared to wild type mice. In the parallel session, Dr. Irene LITNAN, director, Movement disorder center, Neuroscience, University of California, San Diego, USA discussed about the role of genetic as well as environmental/occupational factors in the etio-pathogenesis of PSP. Dr. Irene described about the involvement of genetic factor i.e. microtubule-associated protein tau hene (H1) as well as environmental toxins in the form of pesticides that lead to mitochondrial dysfunction, mainly complex I, which in turn could increase products of oxidative stress that may further leads to tau misfolding. Dr. Shoji TSUJI, Professor, Neurology, The University of Tokyo, Japan expanded the genomic factors responsible for multiple system atrophy (MSA). He showed that COQ2 might be a causative gene for familial MSA and the supplementation with coenzyme Q10 may be helpful in treating patients with MSA.

The post noon session was initiated with an introductory session by Dr. Chiung-Chih Chang, from Seoul National University Hospital, Korea. She comprehensively discussed about clinical diagnostic criterion and screening tools with their relative clinical impacts, for the identification of Parkinson's-disease related dementia (PDD).This was followed by a series of presentations targeting the role of genetic interplay involved in Parkinson's disease pathophysiology and management. While Dr. Yih-Ru Wu from Chang Gung Memorial Hospital, Linkou Branch, Taiwan focussed over targeting genetic as well as genome wide association studies (GWAS) for the studying of Parkinson's disease among Taiwanese patients. Dr. Chung from Asan Medical College, Korea discussed the process line where this GWAS could be used for the evaluation of motor and cognitive outcomes associated with PD. This was followed by a talk by Dr. Tan from National Neuroscience Institute, Singapore which was converged on the concept of targeting a common pathway that coincides both genetic and sporadic forms of PD as well as deciphering a patho-mechanistic link

between genetic and lifestyle interaction for the precipitation of PD.

Biomarkers in PD:

The next series of talks by various eminent speakers were focussed over biomarkers for PD. Dr. Yuk Fai, distinguished neurologist at Queen Elizabeth Hospital, China and Dr. Sheng-Di Chen from University School of Medicine, China emphasized over development of biomarkers in PD for not only its diagnosis at an early stage but also for predicting vulnerability and screening of various neuroprotectives for PD management (3). Also they forward a classification of biomarkers like the clinical biomarkers (hyposmia, depression REM sleep behavioural disorder, olfactory dysfunction), biochemical biomarkers (alpha synuclein, DJ-1, urate) and neuroimaging markers (SPECT, PET fMRI and TCS) which could be used for PD diagnosis. The next speaker for the session Dr. Yi-Hsin Weng from Chang Gung University, Taiwan elaborated the molecular imaging markers which could serve as a diagnostic to screen dopaminergic degeneration in PD (4). The markers included Vesicular Monoamine transporter type 2 (VMAT 2, and 18F-DTBZ (novel VMAT ligand) associated PET scanning. Dr. Serdar KOCER fromHopital du Jura, Porrentruy, Switzerland, put impetus on the application of botulium toxin (BoNTA) for the treatment of post-stroke spasticity. He described about the various techniques like EMG-electrical stimulation, ultrasonography and ultrasound to differentiate between normal muscle and neurological muscle (5).

The consecutive parallel session was inclined towards the novel perspectives in the treatment of PD, where Prof. Louis Tan National Neuroscience Institute, Singapore in his presentation, highlighted the various non-conventional therapeutic approaches (other than neuroprotectives, early symptomatic therapies and main symptomatic therapies) for the management of PD along with the future prospects of therapeutic research as well as novel targets that can be researched upon. Dr. Takahashi from department of neurology, Kyoto University Graduate School of medicine, Japan explained novel animal and cellular iPD experimental models developed by him, which could be harnessed for screening PD pathomechanisms and drug discovery. The animal model was developed by targeting the SNP in the alpha-Syn gene. To enhance alpha-Syn toxicity, the alpha-Syn mutant mice were crossed with IRP2 trangenic mice in which oxidative stress was produced by aberrant iron metabolism, to create a novel animal model for iPD. Similarly the cellular model was developed by using induced pluripotent stem cells (iPSCs) from genetic PD patients and differentiating these into mDA (midbrain dopaminergic neurons). Dr. Hideki Mochizuki, from department of neurology, Osaka University graduate School of medicine, Japan focused on the importance of gene therapy in the treatment of PD. He classified the gene therapy into two groups; 1) symptomatic molecules that relieve the movement difficulties in PD; 2) regenerative proteins that slow down or reverse the process of degeneration of affected dopaminergic neurons.

Non-motor symptoms in PD

The objective of the next session was to review the Non-motor Symptoms associated with PD. The first speaker Dr. Jong Fuh from Department of Neurology, Taipei Veterans General Hospital, Taiwan emphasized on various clinical approaches for identifying PD with dementia (PDD) like deficits on tests of executive function, visuospatial abilities, and verbal fluency as well as behavioural symptoms like hallucinations, delusions and depression. The succeeding speaker Dr. Shen-Yang from division of neurology, University of Malaya, Kuala Lumpur, Malaysia talked about the clinical aspects and etiology of gastrointestinal dysfunctions associated with PD. The discussion included details about symptoms like constipation, dysphagia, sialorrhea and features of gastroparesis associated with PD and involvement of etiologies like alpha-synuclein pathology, gut infections like Helicobacter pylori, bacterial overgrowth as well as impaired absorption of antiparkinsonian medications and caffeine intake for GI morbidities associated with PD. Dr. K Ray Chaudhuri, Director Parkinson's Centre of Excellence, Kings College London, focussed on pathophysiology as well as types and prevalence of impulse control disorders (ICDs) in PD populations. The talk included discussion over examples of ICDs associated with PD like pathological gambling, compulsive buying, compulsive sexual behaviour or compulsive eating, punding as well as the therapeutic strategies used for its management like apomorphine infusion and IJLI (intrajejunal levodopa infusion) therapy (6).

Day 2 started with the plenary session entitled "New Insight in the pathophysiology of Dystonia by electrophysiology. The first orator Dr. Ulf Ziemann from University of Tubingen, Germany reviewed various TMS (transcranial magnetic stimulation) strategies like MEP (motor evoked potentials), CSP (the corticalsilent potential), SICI (short interval intracortical inhibition) as specific tools to understand the cortical pathophysiology involved in focal hand dystonia (FHD) (7). These strategies supported the notion that hyperexcitability, disturbed inhibition, altered sensorimeter integration and abnormal regulation of synaptic plasticity contribute extensively to clinical manifestations of dystonia. The consecutive speaker Dr. Ying-Zu Huang from Medical school, Chang Gung University, Taiwan discussed about the role of premotor cortex in motor plasticity associated

with dystonia. Through his study results concluded that reduced connectivity and excessive motor plasticity seen in dystonia are likely to be compensatory adaptations due to premotor hyperactivity. Thus, targeting the pre-motor cortex area could serve as pivotal therapeutic target for dystonia management. On the other hand, Dr. Mark Hallett, chief, Human Motor Control Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, USA delineated the role of brainstem in dystonias like blepharospasm, cervical dystonia, cranial dystonia, and spasmodic dystonia. He discussed how the loss of inhibitions identified in many brainstem circuits could lead to precipitation of dystonia and thus more emphasis should be placed on evaluating braisnstem mechanisms in dystonia.

Advancement in Pathophysiology in Parkinson's disease

Prof. Robert CHEN from Department of medicine, university of Toronto and Toronto western hospital, Canada has discussed about the "Transcranial Magnetic Stimulation (TMS) in Parkinson's disease". According to Prof. Robert TMS is a non-invasive way of stimulating the human brain. It works by passing a large, brief current through a wire coil placed on the scalp. The transient current produces as large and changing magnetic field, which induces electric current in the underlying brain. In Parkinson's disease (PD), single and paired pulse TMS studies showed decreased cortical inhibition measured by cortical silent period and short-interval intracortical inhibition (SICI), decreased presynaptic inhibition and exaggerated by cortical facilitation (short-interval intracortical facilitation, SICF). TMS can also be used to induce and measure cortical plasticity. Cortical plasticity induced by a protocol known as paired associative stimulation is deficient in PD, particularly in patients with levodopa-induced dyskinesias. TMS studies also provided insights into the mechanisms of deep brain stimulation (DBS). Another technique known as repetitive TMS (rTMS) has been studied as a treatment for Parkinson's disease. Several meta-analyses have found beneficial effects of rTMS as treatment for motor symptoms of PD. In the same session Dr Ming-Kuei LU, Physician from Taiwan discussed about "The differences of motor related cortical activation between Parkinson's disease and essential tremor". He elaborated that the PD and essential tremor (ET) are two the most common movement disorders and motor related cortices are the targets for both diseases. He also showed that the differences of the motor related cortical activation between PD and ET that can be assessed by non-invasive methods such movement related cortical potential (MRCP), TMS and functional brain imaging. Dr. Chiung-Chu Chen, from the department of neurology, Chang Gung Memorial Hospital, Taiwan, has discussed about the "Synchronized neural oscillations and the pathophysiology of Parkinson's disease". She discussed that excessive synchronization of the basal ganglia neuronal activity in the 13-35 Hz frequency band, so-called beta activity has been associated with the motor deficits of Parkinson's disease. Studies has also demonstrated that beta activity may be suppressed by treatment with dopaminergic medication and sub thalamic nucleus (STN) Deep Brain Stimulation (DBS), with degree of suppression correlated with clinical improvement (8).

Dr. Jie-Yuan Li, from division of neurology, Kaohsiung veterans general hospital, Taiwan, has discussed about "Psychogenic (Functional) movement (PMD) disorders". He discussed that PMD are involuntary movements that cannot be attributed to known organic causes, and which commonly have a significant psychological or psychiatric contribution. Typical clinical features suggesting PMD include an abrupt onset with rapid progression to maximum severity. Dr Mark Hallett from Human Motor Control Section, National Institute of Neurological Disorders and Stroke, USA has discussed about "Electrophysiological study in functional movement disorders (FMD)". He discussed that in most situations, the movement is involuntary, but in a minority, when the disorder is malingering or factitious, in which the patient is lying and movement is voluntary. He discussed about the Bereitschafts potential (BP) which is indicative of certain brain mechanisms for generating movement that are voluntary in nature. Prof. Pramod Kumar Pal from National Institute of Mental Health and Neuro Sciences, India discussed about "Case-based video presentation of psychogenic movement disorders". He discussed that psychogenic neurological disorders are disorders which cannot be fully accounted for by any known organic syndrome and which appear, as based on available clinical evidence, to have significant psychological and/or psychiatric contributions. The disorders include psychogenic hemiplegia, paraplegia, blindness, seizures, pain syndromes and variety of movement disorders (psychogenic movement disorders, PMD).

Dr. Nobutaka Hattori from department of neurology, Juntendo University, School of medicine, Japan has discussed about the "Long-term effectiveness of monoamine oxidase-B inhibitors (MAOBI) as initial treatment for Parkinson's disease". The study discussed that the MAOBI as initial levodopa sparing therapy was at least as effective as dopamine agonists. Dr. Erle C. H. Lim from department of medicine, National University of Singapore, Singapore has discussed about the use of Botulinum toxin in the neurology clinic. Botulinum neurotoxin (BoNT) is an effective treatment for conditions associated with over activity of glandular, smooth or skeletal muscle. He also discussed about use of BoNT injections in numerous neurological conditions, including dystonia, headaches and sialorrhea and

drooling.

Dr. Roongroj Bhidayasiri from Centre of Excellence on Parkinson's Disease & Related Disorders, Chulalongkorn Centre of Excellence on Parkinson's Disease & Related disorders, Thailand gave lecture on the "Long-term effectiveness of levodopa as initial treatment for Parkinson's disease (PD)". He discussed that levodopa provide better short-term control of the motor symptoms of newly diagnosed PD and fewer side-effects that do dopamine agonists (DAs) or monoamine oxidase B inhibitors (MAOBIs). He also elaborated that for the practical purposes, low –dose levodopa therapy (up to 400 mg/day) remains a very effective initial treatment of choice for the majority of patients.

Moving further in the session next session was on what's new in Movement disorders. In this session Dr. Chiung-Mei Chen from department of neurology, Chang Gung Memorial Hospital, Taiwan has delivered talk on topic entitled as "From biomarkers to pathophysiology in Huntington's disease (HD)". She discussed about the causative gene mutation for HD which is an unstable CAG tri-nucleotide repeat sequence encoding a polyglutamine (poly Q) tract in the huntingtin (HTT) protein. And this polyQ expansion can cause a conformational change in the mutant protein leading to intranuclear and intracytoplasmic aggregates, which may lead to impaired proteasome activity, transcriptional dysregulation, oxidative stress and mitochondrial and metabolic dysfunction. She highlighted various omics techniques, transcriptomics, proteomics and metabolomics that have been integratively useful in not only identifying biomarkers, but also revealing the pathophysiological abnormalities in HD. In the same session Dr. Zhi-Ying WU from Institute of Neurology, Huashan Hospital, China delivered talk on topic "From genetics to pathogenesis in Paroxysmal Kinesigenic Dyskinesia (PKD)**"**. It is an autosomal dominant movement disorder with characteristics of involuntary attacks precipitated by sudden movements. They have identified the gene using whole-exome sequencing followed by Sanger sequencing encoding the proline-rich transmembrane protein 2 (PRRT2) as a causative gene for PKD. In the same session Dr. Raymond Rosales from department of neurology and psychiatry, faculty of medicine & surgery, University of Santo Tomas, Philippines gave lecture on "Dystonia-parkinsonism (DP) Syndrome: from genetic to treatment". They discussed that DP is named under the Combined Persistent Dystonia classification belonging to the heading of Monogenic Dystonias. DP is hereredo-degenerative accompanied by pyramidal tract involvement or other neurological deficits.

In this session speakers discussed about involvement of movement disorders in sleep medicine. Dr. Carlos H. Schenck form department of psychiatry University of Minnesota, medical School, Minneapolis, USA delivered a talk on "Update on REM sleep behaviour disorder". He discussed that RBD is a strong harbinger of alpha-syncelinopathy neurodegenerative disorders. RBD is situated at a strategic and very active crossroads of clinical sleep medicine, neurology and the neurosciences. In the same session Dr. Meng-Han TSAI from department of neurology, Kaohsiung Chang Gung Memorial Hospital, Taiwan delivered talk on topic "From Paroxysmal Hypogenic Dyskinesia to Nocturnal Frontal Lobe Epilepsy: a Review of Clinical Spectrum and Genetics". He discussed that paroxysmal hypogenicdyskenesia (PND) or nocturnal paroxysmal dystonia was characterized by complex behaviour with dystonic, dyskinetic or ballic movements arise from NREM sleep. These nocturnal episodes are usually brief (<1minute) can occurred many times a night and almost every night. Also they discussed that further molecular evidence came from the studies of families with frontal lobe epilepsies in Australia. Mutations in nicotinic acetylcholine receptor (nAChR) subunit genes were found in ~20 % patients with a positive family history and <5% of sporadic patients. Recent studies using whole-exome sequencing have identified mutation in KCNT1, encoding a sodium gated potassium channel subunit, in severe ADNFLE. In the same session another speaker Dr. Shih-Bin Yeh from department of neurology, Changhua Christian hospital Yun Lin Branch, Taiwan delivers talk on "Advance in Sleep related Rhythmic movement disorders (RMD)". He discussed that RMD consists of various subtypes of sleep related RMD like headbanging, headrolling, body rolling and body rocking. RMD mainly affects infants and children and involves large muscle groups (especially neck and trunk muscles) that engage in repetitive, stereotyped and rhythmic movements that emerge predominantly during drowsiness or sleep with a frequency of 0.5-2 sec and with clustered episodes usually lasting less than 15 min.
During the congress workshop was held on "Evaluation of movement disorders by clinical history and features". During this workshop Dr. Han-Cheng Wang from department of neurology, Shin Kong Wu Ho-Su Memorial Hospital, Taiwan delivered a talk on topic "Hyperkinetic movement disorders". He discussed that hyperkinetic movement disorders (dyskinesias) consist of tremor, chorea, dystonia, myoclonus and tics. He also discussed about the etiology of hyperkinetic movement disorders as it can be primary or secondary or are associated with some heredo-degenerative disorders. Primary movement disorders tend to have involuntary movements as their pure clinical manifestations, and lack of other neurological or systemic features. Secondary movement disorders are caused by identifiable secondary causes within nervous system. Heredo-degenerative movement disorders occur as part of a generalized degenerative of the nervous system.

Dr. Roongroj Bhidayasiri from Centre of Excellence on Parkinson's Disease & Related Disorders, Chulalongkorn Centre of Excellence on Parkinson's Disease & Related disorders Thailand talked on the topic parkinsonian disorder which is recognized by the presence of red flags in the earlier stage of illness. In the 1960s, the many original case report of atypical Parkinsonism was described in the medical literature. Upto1980s the recognition of this disease was limited. Then the atypical parkinsonian disoders (APDS) was recognized as the diagnostic category. He demonstrates that a correct diagnosis of a parkinsonian syndrome will improve the management of individual patients. So it is important to differentiate the PD from the APDs . He suggested that physicians must recognize the different forms of APDs by identifying their idiosyncratic clinical hallmarks or "Red flags".

Dr. Juei-Jueng LIN from department of neurology, Chushang Show-Chwan Hospital, Natou and Chung-Shan University Hospital Taichung, Taiwan described about the paroxysmal dyskinesias which are aheterogenous group of disorders characterized by sudden attack of unilateral and bilateral involuntary movements also included the dystonic postures, chorea, ballismus without loss of consciousness. In his talk he described about the clinical presentation and different diagnosis of paroxysmal dyskinesias. He also demonstrate movement disorders for different diagnosis and most important diagnosis are the non-epileptic psychogenic, non-epileptic organic and epileptic attack disorders especially nocturnal frontal lobe epilepsy.

Dr. Chin-Song LU from neuroscience research centre, Chang Gung Memorial Hospital, Taiwan talked about the treatment of motor complication in Parkinson's disease, as after the chronic levodopa treatment motor fluctuation and dyskinesia are very common. By increasing the dose of levodopa and dividing the dose into smaller but more frequent and also with the addition of dopamine agonist inhibit the breakdown of levodopa and its effect can be prolonged. Pramipexole, ropinirole and transdermal rotigotine reduce off time compared to placebo. Subcutaneous apomorphine reduce the impulsive control disorders and dopamine dysregulation syndrome. Levodopa reduced strategies reduce the levodopa induce dyskinesia (LID) but it worsen the parkinsonism. Amantadine, clozapine and deep brain stimulation (DBS) might improve the LID. Levodopa carbidopa intestinal gel (LCIG) and DBS are helpful in the management of patients with motor complications. Dr. K. RAY Chaudhuri Director, Parkinson's Centre of Excellence, Kings College London from clinical neuroscience Kings college, London described about the non motor symptoms (NMS) in Parkinson's disease. These NMS impair the quality of life throughout the all stages of PD. A recent international survey showed that 62% of NMS might remain undeclared to health. With the PET imaging he described that NMS origin is non-dopaminergic.

Dr. Beom S. Jeon from department of neurology, Seoul National University, Korea discusses about the novel neuroprotective therapeutic options for PD. Although disease modifying therapies have no way in the clinical practice but number of compounds have been identified which shoe neuroprotection in preclinical studies. These neuroprotective drugs can be used in the patients with early signs of disease and even prior to the appearance of disease.

Poster Session:

Yi Ching WENG from department of neurology, LinKou Chang Gung Memorial Hospital, Taiwan presented poster on the case report and literature review on Segawa disease. At low dose levodopa showed the good response in the Segawa disease. Male patients have older age of onset and milder symptoms than female. Pure parkinsonism with excellent response to levodopa is common feature in older male patients with GCH1 mutations. This further expands the understanding of heterogeneity of Segawa disease.

Chai-Hung Chein from Institute of Life Sciences, College of Life Sciences, National Taiwan University, Taiwan represented the upregulation of micoglia-derived cytokines/chemokines which cause the death of nigrostraital dopaminergic neuron in DJ-1 knockout mice. The data which he showed indicate that the INF-gamma derived from DJ-1 deficient microglia is responsible for the lipopolysaccharide-induced neuronal loss and suggest the interaction between the genetic and inflammatory factors contribute for the development of PD.

Dr. Mahmaud Lotfinia from Shefa neuroscience Research Center, Iran presented poster on the decreased spreading depression susceptible in Parkinson rat model. Spreading depression is known as an evoked neuronal activity and change in the ionic, metabolic and hemodynamic characteristics of the brain.

Melatonin is a neuroprotective against iron-induced oxidative stress. From the experiment it came in front that it will attenuate the iron-induced expression of TfR, LfR, DMT1 and FPN1 suggest that the regulatory role of melatonin in the neuronal iron homeostasis. It was presented by Kheun Yen NG. There is an association between the climbing Fibre –Purkinje cell (CF-PC) synaptic pathology and tremor severity in essential tremor (ET). CF-PC synaptic pathology could be an important neuropathological substrate for tremor in ET by the Sheng-Han KUO Colombia University, USA.

Chin-Hsien LIN form National Taiwan University

Hospital, Taiwan present a poster on the repeat expansion in C9orf72 which is not common cause of PD, parkinsonism syndrome or dementia. Spreading depression induced stress to neurons and energy failure in astrocytes by the inhibition of the metabolism also decreased ATP level, energy failures of astrocytes and neurons induction of apoptosis cascades and vulnerability of cells. It was presented by Milad Ahmadi from Shefa Neuroscience Research Center, Iran.

Yu-Hsuan Wu from Taichung Veterans General Hospital, Taiwan presented poster on the risk of premotor symptoms in patients with newly diagnosed PD: A nation-wide population–based study. In which he concluded that the prevalence of premotor symptoms was higher among PD patients than the control. Babak KHODAIE from Shefa NRC-Khatam Hospital, Iran presented the novel therapeutic strategy for movement disorders by alteration of brain waves..

Chi-Feng Tang from the department of neurology, China Medical University Hospital, Taiwan showed that the Taiwanese patients with PD are at higher risk of developing brain tumour than the general population. The exact underlying aetiologies require further investigations.

An inflammatory molecule Nur77/Nurr1 contributes the pathogenesis of PD. But the memantine rescues 6-OHDA-lesioned PC12 partially through the novel mechanisms by regulating the cross talk of Nur77/Nurr1. So his study strongly recommends that the Nurr77 may modulate the mitochondrial impairment in 6 –OHDA-lesioned PC12 cells and the modulation of Nurr 77 and mitochondrial impairments by memantine represent the novel approach and inhibit DA neuronal death by Xiaobo WE.

Aiko matshusima from the Sapporo Medical University, School of medicine, Japan suggested that the specialists of healthcare and administrative bodies need to provide information about public welfare services, supplements and nursing care or delivery meals to prevent families of PD patients from being exhausted with the long-term care giving.

Arindam Bhattacharya from the University of Calcutta, India demonstrated that PQ induces ROS production and differential α-synuclein expression that promotes different patterns of dopaminergic neurotoxicity in three different regions of mouse brain.

Huang Guang from Fu Xing Hospital, China showed that the PD patients with rapid eye movement sleep behavioural disorders (RBD) have more different in REM-SL, RBD period (%) and ERP latency than those patients without RBD and normal control group. The cognitive functional impairments may be correlated with the change of sleep structure.

Sabin Katpattil from Yenepoya Dental College, India discuss the socioeconomic impact on oral health related quality of life of parkinson's disease patients and suggested that the clinicians should routinely check patients oral health in order to maintain the high quality of life of parkinson's disease patients.

Meclizine provide neuroprotection against 6-OHDA in both SH-SY5Y cells and rat primary cortical culture model by the Chientai HONG. The protection was associated with increased glyosis –related mitochondrial hyperpolaization.

In the patient with Parkinson's (pwp), mobility remains the primary concern. Speech and voice changes have a negative impact on the quality of life of pwp as they spin-off a whole range of speech and voice disorders which in turn affect the activity of daily living and social interaction of pwp and those living around them.

Kia-Hsiang Chen from the department of neurology, National Taiwan University Hospital, Taiwan explained the adult onset neurodegenration with brain iron accumulation present with motor tricks and paradoxical RTMS responses.

Asmin SHA from the Al-Iqbal Hospital, India present the poster in which explain that the affected patients had poor oral health and general status and requires restorative and prosthetic therapy.

Dr Anna Chang & Dr HC Wang from Shin Kong Memorial Hospital, Taiwan discussed clinical correlations between gait and balance dysfunction in PD patients. They observed that mentality and frontal lobe functions such as walking, falling and posture are better determinants of actual gait and balance functions rather than motor performances of patients. Dr Kuo-Hsuan Chang and his group from Chang Gung University college of Medicine, National Taiwan Normal University and Chang gung memorial Hospital-Linkou Medical Centre, Taiwan discussed the impairment of proteasome and oxidative pathways in the IPSC model for sporadic PD. They generated PD-induced Pluripotent Stem Cells (PD-iPSC) from a sporadic early onset PD patient carrying a heterozygous deletion of exon 5 in the PARKIN gene. The neurons derived from this demonstrated abnormal α-synuclein accumulation and downregulation of the proteasome and anti-oxidative pathways. Proteasome inhibitor MG132 and H2O2 markedly induced cell death and while the proteasome enhancer benzamil and anti-oxidant genipin rescued from these abnormalities. Dr Ryul Kim and group from Seoul National University Hospital, Korea discussed a

case report wherein the use of dopamine agonist Pramipexole proved to be more effective than levodopa in a post-encephalitic Parkinsonism case. Dr. Yu Ching Huang and group from Tao-Yuan General Hospital, Dayeh University, China Medical university, and Chung-Shan Medical University hospital, Taiwan evaluated the early cognitive impairment prevalence in PD patients in Taiwan. They demonstrated that early cognitive impairment in PD is not rare and PD per se is one of the most contributed risk factors for early dementia. Dr Yuan-Hao Chen and Hsin-I Ma from Tri Service General Hospital/ National Defence Medical Centre, Taiwan discussed the role of dopamine in corticostriatal synaptic plasticity and the consequences of dopamine denervation of the striatum on synaptic plasticity. They explained that dopamine plays a role in supporting several forms of striatal plasticity, the impairment of which related to the symptomology of PD. Dr Kai-Cheng Hsu and Dr Feng-Sheng Wang from Hsu Kai Cheng Neurology Clinic and National Chung Cheng University, Taiwan explained the simulation of dopamine metabolism by mathematical models to reveal the behavior of biomedical system with defected enzymes. They showed that the fuzzy optimization method can compute the satisfaction grades of main metabolites & side products and that the simulation of human metabolic disorders is helpful to realize the synergistic effects of different drugs. Dr Yu Aoh and Dr Chon-Haw Tsai from China Medical University Hospital, Taiwan discussed a case of Frontotemporal Lobar Degeneration overlapping syndrome (FTLD-os). They highlighted the occurrence of all the clinical features of frontotemporal dysfunction, parkinsonism and motor neuron disease in the same subject of FTLD-os. Dr Jin Siang Shaw and his group from National Taipei University of Technology, Taiwan discussed the development of a knee type laser cueing device for PD patients to overcome abnormal gait. They showed that the knee type laser cueing device improved gait patterns, decreased events of freezing of gait and increased mean walking velocity. Dr Ya-Ting Chang and Dr Cheng-Hsien Lu from Chang-Gung Memorial Hospital, Taiwan discussed the risk factors associated with Frozen Shoulder Syndrome in a prospective cohort study with 34 patients with a definitive diagnosis of idiopathic PD who were followed up at the Neurology Out-patient clinic for more than 6 months after titration of their daily anti-Parkinsonian agents to a steady dose in accordance with their clinical symptoms. They concluded that higher mean ipsilateral UPDRS III and sub-scores are associated with higher risk of frozen shoulder syndrome in PD patients.

Puneet Kumar and his group from ISF college of Pharmacy, Moga, India discussed the involvement of Hemeoxygenase-1 and GSK-3β in the pathophysiology of HD. They showed that a combination of Hemin and Lithium chloride synergistically exerted a protective effect in reversal of oxidative and inflammatory abnormalities caused by 3-NP induced HD. Dr Wei-Chung Wang from China Medical University Hospital and Chang Gung Memorial Hospital, Taiwan discussed a case of unusual mutation in Dopa Responsive Dystonia. They observed a mutation of c.-152T>C at Exon 1a in the GCH1 Gene. Dr Hung-Ju Chen and Dr Chih-Shan Huang from Wan Fung Hospital, Taipei Medical University, Taiwan discussed a case report of a 20 Yr old woman who developed subacute combined degeneration presenting Dystonia and Pseudoathetosis after chronic administration of N2O for about 2 years. This condition is treatable with vitamin B12. Dr Junghwan Shin and his group from Seoul National University Hospital, Korea explained the search for cut-off value of SCA-17 which is an autosomal dominant cerebellar ataxia with expansion of CAG/CAA trinucleotide repeats in TATA-binding protein gene. Dr Han-Tao Li and Dr Yih-Ru Wu from Chang Gung Memorial Hospital-Linkou Medical Centre and Chang Gung University College of Medicine, Taiwan presented a case report of HIV associated myelopathy induced spinal cord disease. They showed that the spastic paraplegia in this condition was improved by treatment with highly active anti-retroviral therapy (HAART) combivir and baclofen. Dr Yi Jung Lee and group from Neurological Institute, Taipei Veterans General Hospital, Taiwan presented a case report of patients of Blateral Striopallidodentate calcinosis (BSPDC) presenting with hyperkinetic movement disorders. The hyperkinetic movement disorders included ballism, chorea, dystonia and oro-facial dyskinesia. Dr Tsung-Pei Yang and Dr Tsu-Kung Lin from Chang Gung Memorial Hospital, Taiwan discussed a case report showing hot cross bun signs after Pontine infarction. They demonstrated that Hot Cross Bun signs are not specific to Multiple System Atrophy and were observed in this case after pontine infarction. Dr Wey Yil Lin and his group from Chang Gung Memorial Hospital, Taiwan reported a case of delayed encephalopathy after carbon monoxide intoxication presenting generalized chorea. In this case hypokinetic movement disorder was observed and the neuroimaging studies disclosed bilateral pallidal or striatal lesions. Dr Han-Chieh Hsieh from National Cheng Kung University Hospital, Taiwan discussed a case report of a middle aged woman with concomitant Systemic Lupus Erythematosus and Huntington's disease. They raised an attention that the patients who have both cognitive impairment and chorea should receive genetic test of Huntingtin gene despite of previous history of Systemic Lupus Erythematosus. Dr Yu-Ting Wu and his group from Taichung Veterans General Hospital, Taiwan presented a case report showing cerebellar variant of Adrenoleukodystrophy with ABCD1 Gene Mutation. The mutation was reported to be frameshift, c.1866-10G>A identified in intron of8

of the ATP-binding cassette D1 gene (ABCD1). Dr Yi-Chien Yang and his group from China Medical University Hospital, Taiwan discussed a case of NeuromyelitisOptica relapsing with complex movement disorders. The subject had a history of Neuromyelitis Optica with positive AQP$ antibody and had recently developed acute tremor like movements in the left limbs. Methylprednisolone 1gm for 3 days resolved the tremors gradually.

Concluding Remarks:
Overall, the 2nd Taiwan International Congress of Parkinson's disease and Movement Disorders was a huge success and advancement in the research activities in PD and related disorders all around the world showed great promise for better understanding of these diseases as well as the possible future therapies.

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