Neurorestoration Workshop

The 3rd Scientific Meeting Updates in Neurology

Event Report
I. **Mild Cognitive Impairment in Parkinson Disease “Early Markers of Cognitive Decline and Therapeutic Development in Parkinson** (dr. Silvia F. Lumempouw, Sp.S (K))

Idiopathic Parkinson’s Disease (PD) is a progressive neurodegenerative disease that is typically characterized by symptoms of motor features: bradykinesia, tremor, rigidity, and postural instability. However, it has been increasingly clear and significant presence of non-motor features such as cognitive impairment, constipation, bladder dysfunction, sleep disorder, depression, anxiety, and psychosis. These symptoms are often less well known and treated, but can dominate PD symptoms at an advanced stage with the occurrence of significant disability, impaired quality of life and reduced life expectancy.

Cognitive disorders are highly prevalent in PD and deficit varies from mild to severe dementia. Normally, dementia occurs in advanced stages of the diseases. Patients with this disease that has lasted 20 years there were 80% with dementia. In contrast, mild cognitive impairment occurs in diseases of early PD and one study reported that more than a third of newly diagnosed PD patients had mild cognitive deficits. PD patients with non-dementia had an average of 26.7% (range: 18.9%-38.2%) mild cognitive impairment.

The important note, cognitive impairment will have an impact on quality of life, increase the burden on the caregiver stress, and increase the risk for specific nursing placement. This disorder tends to develop into dementia so it needs early recognition of the existence of mild cognitive impairment to a new therapeutic intervention, which aims to change the course of the disease.

II. **Clinical Spectrum of Dystonia That Are Responsive to Dopamine Related Drugs**
(Prof. Beom S. Jeon, MD, PhD)

Dopa-responsive dystonia (DRD) is a disease entity with a variety of spectrum. The most classic DRD has the following characteristics - childhood or adolescence-onset, mild parkinsonism, marked diurnal fluctuation, improvement by sleep or rest, and a dramatic and sustained response to low doses of L-dopa without motor fluctuations or dyskinesias, whereas some reports show DRD cases with atypical phenotypes. In addition, we cannot exclude DRD even if the result of genetic test is negative, because the yield of genetic diagnosis in DRD is not high. The genetic confirmation could not always guarantee therapeutic effects and prognosis.

The definitions of DRD and DRD-plus are suggested to compensate the defects of clinical and genetic diagnosis. DRD is defined as a syndrome of selective nigrostriatal dopamine deficiency caused by genetic defects in the dopamine synthetic pathway without nigral cell loss. DRD-plus means a inherited metabolic disorder that has features of DRD and additional features that are not seen in DRD. The
concepts of DRD and DRD-plus are more practical in diagnosis and treatment, because they contain pathogenesis in biochemical level. In this presentation, biochemical defects, genetics, clinical phenotypes of DRD and DRD-plus will be discussed. In addition, the concept of DRD look-alikes will be discussed.

III. **Myoclonus and Other Jerks** (Prof. Beom S. Jeon, MD, PhD)

Myoclonus is a brief, shock-like contraction of a muscle or group of muscles. These myoclonic twitches, jerks, or seizures are usually caused by sudden muscle contractions (positive myoclonus) or brief lapses of contraction (negative myoclonus). Myoclonic jerks may occur alone or in sequence, in a pattern or without pattern. They may occur infrequently or many times each minute. Myoclonus sometimes occurs in response to an external event or when a person attempts to make a movement. The twitching cannot be controlled by the person experiencing it. In its simplest form, myoclonus consists of a muscle twitch followed by relaxation. A hiccup is an example of this type of myoclonus. Other familiar examples of myoclonus are the jerks or "sleep starts" that some people experience while drifting off to sleep. These simple forms of myoclonus occur in normal, healthy persons and cause no difficulties. When more widespread, myoclonus may involve persistent, shock-like contractions in a group of muscles. In some cases, myoclonus begins in one region of the body and spreads to muscles in other areas. More severe cases of myoclonus can distort movement and severely limit a person's ability to eat, talk, or walk.

Diagnosis is clinical and sometimes confirmed by electromyographic testing. Classifying the many different forms of myoclonus is difficult because the causes, effects, and responses to therapy vary widely. Treatment includes correction of reversible causes and, when necessary, oral drugs to relieve symptoms. In this presentation, clinical phenotypes, classification, pathophysiology, and treatment will be discussed.

IV. **Chorea and Paroxysmal Dyskinesia** (dr. Banon Sukoandri, Sp.S)

Chorea consists of involuntary, continual, abrupt, rapid, brief, un-sustained, irregular movement that flow randomly from one body part to another. The pathophysiology of chorea is poorly understood, but intracortical inhibition of the motor cortex is still normal. Chorea can be manifestation of primary neurologic disorders, or is occur as neurologic complication of systemic, toxic and another disorders; also can be induced by any drugs. The first step in management of chorea is identification a specific etiology. Anti choreic agent that have ability to blocking D2 receptors can control choreic symptom. Any medications can be useful for chorea such as: dopamine receptor blocking drug (neuroleptic), reserpine and tetrabenazine; sodium valproate, levetiracetam and clonazepam may be effective in the treatment of chorea.

Paroxysmal Dyskinesia (PxD) are a group of rare movement disorders characterized by their recurrent and episodic nature, arising from a background of normal motor activity and behavior. These can be manifest in the form of ballism, dystonia, chorea and athetosis or a combination of these (Sethi 2000). From historical perspective, these disorders initially reported as a type of epilepsy. PxD classified into four types: (i) Paroxysmal kinesigenic dyskinesia, (ii) Paroxysmal non-kinesigenic dyskinesia, (iii) Paroxysmal exertion-induced dyskinesia, (iv) Paroxysmal hipogenic dyskinesia. Treatment of PxD
depend on type of symptom. PKD respond well to anti-convulsants, such as Carbamazepin, Phenytoin, Lamotrigine. But PNKD less respond to anti-convulsant; clonazepam, haloperidol, anti cholinergic should be tried.

V. **Pitfalls in The Management of Parkinson Disease** (dr. Banon Sukoandri, Sp.S)

Management of Parkinson's disease is unique and individual for every people with Parkinson Disease. Some of them are often forgotten and becomes a trap in daily clinical practice, such as the diagnosing process through clinical examination can’t be done only in one visit, because the diagnosis may change over time when a few new symptoms that should not exist in this disease are found. Determination of treatment and anti-Parkinson's drug selection is also important because it can affect the course of disease and the emergence of side effects of medications, especially Levodopa. The symptoms monitoring only focus on the motor symptoms, while non-motor symptom often receive less attention. Management of the disease can not only use any medical treatment, but also the modalities other than drugs, such as exercise and sports, that also plays an important role in the successful management of this disease. Consideration of surgery in this disease should also be appropriate to obtain maximum results.

VI. **Tics and Tourette Syndrome** (dr. George Dewanto, Sp.S (K))

Tourette Syndrome (TS), which should be more appropriate called Gilles de la Tourette Syndrome, is a neurologic disorder manifested by motor and vocal or phonic tics. Usually starting during childhood and often accompanied by obsessive compulsive disorder (OCD), attention deficit hyperactivity disorder (ADHD), poor impulse control, and other comorbid behavioral problems. Tics are its core feature. TS is now recognized as a relatively common and complex neurobehavioral disorder. The prevalence of TS remains unclear partly because of the lack of bilological markers and the wide symptom fluctuations and partly because of the lack of a consensus regarding the definition of this disorder. It is estimated that between 1 and 3 % of the school age population is affected by TS. The cause of TS is yet unknown, but appears to be inherited in the majority of patients. Although the pathogenic mechanism of TS are still unknown, the weight of evidence supports an organic rather than psychogenic origin, probably involving the basal ganglia circuitry. The first therapeutic approach in TS is education and demystification of the symptoms. People in frequent contact with the child should be informed about tics, fluctuations, and possible comorbidities.

VII. **How to Choose Antiparkinsonian Drug to Initiate Treatment of Parkinson Disease**
(dr. Andradi Suryamiharja, Sp.S (K))

The current treatment of Parkinson's Disease is only symptomatic, while treatment to slow or inhibit the progression of the disease, although it has been widely researched, no one has given the convincing results. Symptomatic treatment modalities include pharmacologic therapy, non-pharmacological, and surgical therapy. The aim of symptomatic therapy is to improve Quality of Life (QOL) of patients, by making them as independent as possible. Therefore, from the beginning we have to choose the right medicine as initial therapy, which is effective in treating the symptoms of PD, and give fewest side effects and complications of long-term treatment. Antiparkinson drugs include class of dopaminergic ---- Levodopa, Dopamine Agonist (pramipexole, Ropinirole, Rotigotine transdermal patch, Apomorphine Injection)), Akmantadine, MAOB-inhibitor (Selegiline, Rasagilin), COMT-inhibitor -- --, kholinergik ( trihexyphenidyl). In choosing which drug to be used as initial therapy, several aspects must be considered: the patient’s age, severity of symptoms / disease stage, the predominant
symptoms, and medication profiles. For younger patients (<40 years), initial treatment with agonists, MAOB-I, or antikolinergik, and delay the use of levodopa; in older age (> 60 years) therapy begins with agonist, agonist, or MAOB-I when the symptoms are mild, or if their symptoms are moderate - weight can be directly start with Levodopa. Avoid antikolinergik in this age group; for ages 40-60 years old, at an age closer to 40 years initially as therapy at a young age <40 years. While approaching the age of 60 years, start the therapy as in older age> 60 years. Consideration of severity, in which light can be prefixed by Agonist, MAOB-I, or Antikolinergik, wa; aupport after months Are Some years need to be added to levodopa. If symptoms are severe, it can be initiated directly with Levodopa.
I. ACTIVITY CONTENT AND OBJECTIVES
   A. Learning Objective 1
      (Better understanding about an approach to patients with movement disorder (diagnosis, clinical features, progression))

   1. Before the Activity (n=19)

   2. After the Activity (n=19)
B. Learning Objective 2
(Better understanding about the treatment of PD (how to choose antiparkinsonian drug))

1. Before the Activity (n=19)

2. After the Activity (n=19)
C. Learning Objective 3
(Increased knowledge and skill of Botulinum Toxin Injection Technique)

1. Before the Activity (n=19)

2. After the Activity (n=19)
D. Learning Objective 4
(Better understanding about Deep Brain Stimulation for Parkinson's movement disorders)

1. Before the Activity (n=19)

2. After the Activity (n=19)
E. Learning Objective 5
(Being recognized to Deep Brain Stimulation for Non-Parkinsonis Movement Disorder)

1. Before the Activity (n=19)

2. After the Activity (n=19)
II. LEVEL OF AGREEMENT OF THE STATEMENT
   A. The content of this program is relevant to my practice (n=19)

   ![Bar chart](chart1)

   - Strongly Agree: 5
   - Agree: 14
   - Disagree: 0
   - Strongly Disagree: 0

   B. Participation in this activity enhanced my professional effectiveness (n=19)

   ![Bar chart](chart2)

   - Strongly Agree: 6
   - Agree: 13
   - Disagree: 0
   - Strongly Disagree: 0

   C. The science and medical knowledge advanced by this activity will ultimately enhance care of patients with Movement Disorder (n=19)

   ![Bar chart](chart3)

   - Strongly Agree: 5
   - Agree: 14
   - Disagree: 0
   - Strongly Disagree: 0
D. The audiovisuals were effective (n=19)

E. The overall format of this activity was effective (n=19)

F. I would like MDS to continue to offer educational activities on this topic (n=19)
III. COURSE DIRECTOR
(Dr. Yohanna Kusuma, Sp.S, ASN, WFN-NSRG)

A. The course director ensured the activity and its component presentations began and ended on time (n=19)

B. The course director ensured the faculty adequately addressed the learning objectives of this activity (n=19)

C. The course director objectively moderated question/answer discussions associated with the activity (n=19)
D. The presentation was free of commercial bias. If No, please comment. (n=19)

IV. FACULTY
(Beom Seok Jeon, MD, Ph.D and Lim Shen Yang, MBBS, MD, FRACP)

A. The speaker is knowledgeable and demonstrated appropriate expertise in the subject area (n=19)
B. The speaker was clear, concise, and able to keep my attention (n=19)

C. The presentation materials were appropriate and effective (n=19)
D. The presentation was free of commercial bias. If No, please comment. (n=19)

V. OTHER COMMENTS
A. What would you like to change in your practice because of this course?
   1. to give more attention and be detailed about diagnosing movement disorders, in giving therapies, not just pharmacologic but also non-pharmacologic, care of patients and quality of life
   2. a better approach for patient with PD

B. The major strengths of this activity were:
   1. treatments of PD: pharmacology, surgery (DBS)
   2. movement disorders: an approach to patients
   3. more time for discussion session
   4. new knowledge
   5. Botulinum Injection course

C. How would you improve this activity?
   1. more interactive activities
   2. more discussion and hands on course during the program
   3. will join the workshop again
D. How did you learn about this activity?

VI. FEEDBACK TO IMPROVE OVERALL MDS EDUCATION PROGRAM
A. Live, lecture-style educational activities (n=19)

B. Live, interactive educational activities (n=19)
C. Printed continuing medical education (CME) materials (n=19)

D. Online, web-based CME (n=19)

E. CD-ROM based CME (n=19)
VII. I am interested in attending future educational activities on the following topics:

Other:

Botox Injection
CHAPTER III
WORKSHOP DOCUMENTATIONS

Picture 1. Participant’s registration for the workshop

Picture 2. Opening Ceremony of The 3rd Scientific Meeting Updates in Neurology 2016
Prof. Lim Shen Yang, MD, MBBS, FRACP is delivering his lecture about an approach to patients with Movement Disorder.

Prof. Beom S. Jeon, MD, PhD is delivering his lecture about Dystonia and Myoclonus.
Picture 5. Prof. Lim Shen Yang, MD, MBBS, FRACP during his lecture about PD management strategies

Picture 6. Prof. Jusuf Misbach, Sp.S (K) is performing Live Demo on Botulinum Injection technique
Participants during the symposium

Participants during the workshop
Picture 9. dr. Thamrin Syamsudin is delivering his lecture