Call for Abstract for 2022 Annual Meeting of the Taiwan Movement Disorder Society, 12th-13th March 2022, Taichung, Taiwan

1. Date: 2022/03/12-13
2. Venue: Room 401 and 402, GIS Taichung Xinwuri Convention Center, No. 26, Gaotie E. 1st Rd., Wuri Dist., Taichung City 414, Taiwan.
3. The abstract submission is now open and will be closed on 13th of February 2022. We invited all participants to submit abstracts relevant to the fields of movement disorders. Abstract submission is limited to ONE abstract per presenter, first author by default. Please prepare the manuscript with Microsoft Word document. The Committee has the right to reject the abstract which is not fit the required standard. Please follow the submission regulation (see below) for Original study or Case report. Please do specify the abstract category, affiliation of corresponding author and full contact details. Please send the file to mds.taiwan@gmail.com (For Secretary Miss Chen's attention). Email confirming submission will be sent on the next day; please contact TMDS if no confirmation email is received.
4. The abstract submission will be directly synchronously submitted to 2022 TIC-PDMD.
5. The Scientific Committee reserves the right to review all submission. Abstracts required further revision will be sent back to corresponding authors by 25th of February. Please do re-submit the revised abstract by 2nd of March 2022.
6. TMDS provides young scholar award for the first author under 40 y/o (inclusive):
   1) Original Study: 1st Prize NTD 10,000; 2nd Prize NTD 8,000; 3rd Prize NTD: 6,000; 5 special Prize NTD 2,000 each;
   2) Case Report: 1st Prize NTD 8,000; 2nd Prize NTD 7,000; 3rd Prize NTD 5,000.

Guideline for Abstract Submission,

1. Manuscripts should be written in English and submitted in the form of Microsoft Word. The formation should be double-spaced, using a 12-point font size and a default typeface (recommended fonts are Times, Times New Roman, Courier, Helvetica, and Arial), at A4 paper dimensions, and **maximum** one A4 page.
2. Article title: Please include abstract tile, all authors' names, degrees and affiliations, and corresponding author's contact information. The first author is the presenter by default. Please restrain from listing more than 6 co-authors.
3. Abstracts of Original articles must be submitted in the following structure: **Background**, **Objectives**, **Methods**, **Results**, and **Conclusion**. Abstracts of Case report must be submitted in the following structure: **Background**, **Case report**, and **Conclusion**. Please be concise and do not include fingers, tables, keywords or references.
4. Examples for Original articles and Case report are listed below.

Example 1 (Original study)

Impaired cerebellum to motor cortex associative plasticity in Parkinson's disease

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Background

Patients with PD have been reported to bear dysfunction in the cerebello-dentato-thalamo-cortical (CDTC) circuit. Previous evidence showed that bidirectional STDP-like plasticity in primary motor cortex (M1) can be mediated by associative stimulation of the CDTC pathway and M1 in young adults. The feature of such plasticity has never been addressed in elder people and in pathological conditions.

Objective

To compare the corticocortical paired associative stimulation (PAS)-induced spike-timing dependent plasticity (STDP)-like plasticity between Parkinson's disease (PD) and healthy elder subjects.

Methods

Nine patients with PD (Hoehn and Yahr stage 2-3) and nine age-matched healthy subjects were studied. One hundred and twenty pairs of transcranial magnetic stimulation (TMS) of the left M1 preceded by right lateral cerebellum (CB) TMS at an interstimulus interval of 2 ms (CB→M1 PAS2ms) or 6 ms (CB→M1 PAS6ms) were applied. M1 excitability was assessed by motor-evoked potential (MEP) amplitude, short-interval intracortical inhibition (SICI), intracortical facilitation (ICF) and cerebellar inhibition (CBI) in the first dorsal interosseous muscle of the right hand before and after CB→M1 PAS.

Results

Temporary MEP potentiation was found by CB→M1 PAS2ms protocol. In contrast, CB→M1 PAS6ms resulted in a long MEP depression. These effects were only observed in the healthy elder subjects but not in the patients with PD. SICI, ICF and CBI did not show any significant change.

Conclusion

The current findings suggest that CB→M1 PAS-induced bidirectional STDP-like plasticity in M1 may be preserved in elders and significantly impaired in patients with PD. Findings also support the notion of functional perturbation within the CDTC circuit in the patients with PD.

Abstract category：Original study

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Example 2 (Case report)

Deferiprone in the Treatment of Superficial Siderosis

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Background

Superficial siderosis of the central nervous system results from hemosiderin deposition in the subpial layers in the brain and spinal cord, with the presentation of cerebellar ataxia, sensorineural hearing impairment and myelopathy. No definite treatment has been established for relieving symptoms or modifying disease course. Oral deferiprone, a lipid-soluble iron chelator, has been tried in a pilot safety study without significant adverse effects, and the brain image revealed reduction in hemosiderin deposition. Herein we reported a case of superficial siderosis treated with oral deferiprone for 6 months.

Case Report

Ms. Lin, a 59 year-old retired cook, presented with progressive gait unsteadiness since 2009, together with tinnitus, hearing impairment, and scanning speech. There was no history of subarachnoid hemorrhage, head trauma, or surgery of brain and spine. The neurological examination showed generalized hyperreflexia, wide-based gait, bilateral limbs dysmetria and dysdiadochokinesia. The Pure tone audiometry revealed sensorineural hearing impairment, and the auditory brainstem response was normal. The magnetic resonance imaging (MRI) showed hypointensity on T2-wieghted and T2*GRE sequences, covering the surface of brainstem, cerebellum, sylvian fissure, and spinal cord. No microbleeds were detected in the brain parenchyma. The series of MRI over 6 years revealed gradual increase of hemosiderin deposition.

She began oral deferiprone 500mg twice a day from March, 2015. We used SARA (Scale for the Assessment and Rating of Ataxia) score as the tool of assessment, and its total scores range from 0 (no ataxia) to 40 (most severe ataxia). Her SARA score improved from 13 in March to 10.5 in July after four months of treatment, especially in the domains of limb coordination and speech. The patient felt improvement in scanning speech and trunk balance subjectively. No adverse effect was reported. The following MRI six months after treatment revealed slight reduction in hemosiderin deposition on the surface of brainstem, cerebellum and cortex compared with the MRI before treatment.

Conclusion

Our patient with superficial siderosis tolerated oral deferiprone well and showed slight improvement both in SARA score and hypointensity in T2W MRI. Future trials with more subjects and close follow ups with both SARA scale and MRI are warranted to confirm the therapeutic effects.

Abstract category：Case report

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