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Video Presentation

35 YEARS OF EXCELLENCE IN MEDICAL PUBLISHING

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Facilitators and barriers to the delivery of palliative care to patients with Parkinson's disease: a qualitative study of the perceptions and experiences of stakeholders using the socio-ecological model

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Statement of the Problem: Palliative care (PC) can improve the quality of life of Parkinson's disease (PD) patients and their care givers. However, current research on the impact of the provision of PC services for patients with PD is still unclear. We sought to identify the barriers and facilitators affecting the provision of PC services for patients with PD based on Social Ecological Model (SEM) framework.

Methods: We conducted semi-structured interviews and the SEM was used to organize themes and identify potential solutions across multiple levels.

Results: Twenty-nine patients were interviewed. Facilitators and barriers were identified according to the levels of the SEM. There are 2 facilitators and The facilitators were as follows: (1) individual level: strong needs from PD patients and relatives, health professionals' desire for PC knowledge; (2) interpersonal level: social support; (3) organizational level: firmly attitudes towards the systematization of PC, a large team of nurses; (4) community level: convenience of community services, a Hospital-Community-Family-Based service; (5) culture and policy level: existing policy. While the barriers were: (1) individual level: false beliefs about palliative care, economic burden; (2) interpersonal level: lack of communication time, low quality of multidisciplinary teamwork; (3) organizational level: lack of PC specialist nurses, lack of referral criteria for PC, absence of economic benefits indicators; (4) community level: lack of PC resource access, discontinuity of care; (5) culture and policy level: culture of death, ethical dilemmas, lack of health insurance for PC.

Conclusion: The application of a social ecological model in this study helped to illuminate the complex and multilevel factors that may influence the delivery of palliative care among PD patients.

Recent Publications:

1. Armstrong, M. J., & Okun, M. S. (2020). Diagnosis and Treatment of Parkinson Disease: A Review. Jama, 323(6), 548-560.

2. Boersma, I., Jones, J., Coughlan, C., Carter, J., Bekelman, D., Miyasaki, J., . . . Kluger, B. (2017). Palliative Care and Parkinson's Disease: Caregiver Perspectives. J Palliat Med, 20(9), 930-938.

3. Bouça-Machado, R., Titova, N., Chaudhuri, K. R., Bloem, B. R., & Ferreira, J. J. (2017). Palliative Care for Patients and Families With Parkinson's Disease. Int Rev Neurobiol, 132, 475-509.

4. Room R, BaborT,Rehm J (2005) Alcohol and public health. Lancet 365: 519-530.

5. Sullivan EV, Zahr NM (2008) Neuroinflammation as a neurotoxic mechanism in alcoholism: Commentary on "Increased MCP- 1 and microglia in various regions of human alcoholic brain". Experimental neurology 213:10-17.

Biography

Yiping Chen has her expertise in improving and promoting the palliative care in Parkinson's disease. She focuses not only on people with Parkinson's disease, but also on the caregiving burden of their care givers, developing a corresponding theoretical model of caregiving burden. She is committed to promoting clinical practice through the integration and mining of evidence, and specialises in qualitative research methods to uncover the barriers and facilitators behind palliative care from a constructivist perspective.

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Accepted Abstracts

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Live your best: discovering the win in chronic illness

Tim Hague Sr
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At 46 years old, you find your life turned upside down by three little words, 'You have Parkinson's'. How does one move beyond the debilitating diagnosis of a chronic disease to live life to the full? I have discovered three other words that, when taken together, have changed everything, 'Live Your Best'! At the insistence of my wife, and two years after being diagnosed with Parkinson's Disease, my son and I applied for, were accepted to and won season one of The Amazing Race Canada. How does one go from a diagnosis of Parkinson's disease to winning a gruelling, travel adventure, reality television show? You learn to Live Your Best.

When we come to understand that having *the strength to simply do our best will always be enough, when we uncover the courage to be content with what our best produces and when we discover that perseverance* is a skill that can be learned, we will experience more joy and success in life than we ever imagined. In this session, you will learn from a veteran speaker and Tedx presenter how to meet a challenging life event like a diagnosis of Parkinson's disease and still succeed. Drawing on my 20+ years as a Registered Nurse and now 7+ years as a person with Parkinson's I will teach you why the statement 'don't give up' is so empty and in its place, find the power of perseverance.

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Molecular Pathogenesis and Neuroinflammation in Parkinson Disease beyond Alfa-synuclein: A Current Overview

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Various mechanisms play an essential role in the pathogenesis of Parkinson's disease, including a disruption of the cellular energy balance (mitochondrial dysfunction) and oxidative stress and disruption of protein breakdown (lysosomal and proteasomal dysfunction). The protein α-synuclein plays a central role in the pathogenesis of Parkinson's disease and is involved in many intracellular functions or their disruption in Parkinson's disease. A number of important cellular processes are inhibited by aggregated α-synuclein. The disruption of these processes, in turn, leads to increased aggregation of α-synuclein. Therefore, protein misfolding, aggregation, and accumulation of aggregated α-synuclein is a key feature of Parkinson's disease. In the course of the disease, there is an inflammatory reaction in the brain (neuroinflammation), in which mainly microglial cells are involved. The exact triggers of neuroinflammation are not known. However, it is known that aggregated α-synuclein and neuromelanin are able to activate microglia. In the course of neuroinflammation, there is a change in the expression of toll-like receptors (TLR) on the microglial cells. Hence, in Parkinson's disease, both the aggregation of proteins, primarily α-synuclein and one that occurs during the course of the disease play a role in neuroinflammation with microglial activation in the brain of Parkinson's patients playing a role in the pathogenesis of the disease.

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Prevalence and clinical characteristics of probable REM behavior disorder in Thai Parkinson's disease patients

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Background: Previous studies have shown that Parkinson's disease (PD) patients who have REM behavior disorder (PD with RBD) might be a PD subtype since they have different symptom clusters and disease trajectories from PD without RBD.

Objective: To study the prevalence of PD with pRBD and to compare the clinical characteristics with PD without pRBD. The feasibility of clinical interview of items adopted from the Mayo Sleep Questionnaire was also to be determined.

Methods: A total of 140 Parkinson's patients visiting neurological clinics during January to December 2016 were enrolled in this study. "Probable RBD (pRBD)" was defined as present when the patient answered "yes" to a question adapted from the first Mayo Sleep Questionnaire (MSQ). The demographic data, motor symptoms, and nonmotor symptoms were obtained.

Results: The prevalence of pRBD among this study's PD patients was 48.5% (68 out of the total of 140). The median onset of RBD before PD diagnosis was 5 years (range: 0–11 years). By comparison of PD with pRBD and PD without pRBD, this study showed significant difference in the levodopa equivalent dose (742 mg/day versus 566 mg/day; $p < 0.01$), prevalence of symptomatic orthostatic hypotension (35.3% versus 8.3%; $p < 0.01$). The multivariable analysis found that pRBD is independently associated with orthostatic hypotension (OR = 5.02, $p < 0.01$). Conclusion: The findings regarding prevalence and main clinical features of PD with pRBD in this study were similar to those of a previous study of PD with polysomnogram-(PSG-) proven RBD. This study hypothesized that interviewing by adopted MSQ may be a cost-effective tool for screening RBD. Further studies with direct comparison are needed.

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Hallucinations in Parkinson's disease: A window into the phenomenology and physiopathological bases of delusions in neurodegenerative diseases

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Hallucinations are a frequent neuropsychiatric complication in Parkinson's disease (PD). They may be present from the earliest stages of the disease and manifest from mild delusional phenomena, including passage and presence hallucinations, to well-structured visual hallucinations with loss of insight.

Tracking the progressive phenomenological changes of hallucinations and delusions in PD helps to understand the different neuronal networks that become impaired through the neurodegenerative process. Further, the different contribution of visuoperceptive areas and their functional connections with the dorsal and ventral attentional networks and the default mode network delineate also a framework that explains how environmental stimuli are perceived, built-up into visual constructs, and finally transferred into consciousness.

The 'big jump' between the existence of external objects and their perception by human brain explains how mental imagery is able to intrude into consciousness and create such false but vivid hallucinatory experiences.

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Targeting fyn by localized RNA therapy to reduce levodopa induced dyskinesias in Parkinson's disease

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PLevodopa is the gold standard treatment for Parkinson's disease (PD) despite after its necessary and prolonged use most patients often develop side effects, known as levodopa-induced dyskinesia (LID). The management of LID usually requires therapeutic intervention, and the only available alternative is the use of the NMDA receptor antagonist amantadine, which provides limited efficacy. LID is currently one of the major challenges in PD research and the NMDA receptor is indeed the most plausible target to its management. In this context, we postulated the kinase Fyn, a key NMDA receptor regulator, as a new putative molecular target against LID. Our group developed an experimental therapeutic strategy to knock down Fyn expression to alleviate LID in 6-OHDA-lesioned mice treated with levodopa. To such a goal we performed intra-striatal delivery of a designed micro-RNA against Fyn (miRNA-Fyn), which was delivered either before or after levodopa exposure to assess its ability to prevent or revert dyskinesia. Pre-administration of miRNA-Fyn reduced LID with a concomitant reduction of FosB-ΔFosB protein levels –a marker of LID– as well as decreased phosphorylation of the NR2B-NMDA subunit, which is a main target of Fyn. On the other hand, post L-DOPA delivery of miRNA-Fyn was less effective to revert already established dyskinesia, suggesting that early blocking of Fyn activity might be a more efficient therapeutic approach. Our results provide proof of concept about Fyn as a plausible therapeutic target to manage LID, and validate RNA silencing as a potential approach to locally reduce striatal Fyn, rising new perspectives for RNA therapy interventions in PD.

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