8th International Conference on

Dementia and Dementia Care

September 18-19, 2017 Dublin, Ireland

Keynote Forum

Day 1

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Ahmet Turan ISIK

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DPD4 inhibitors for Alzheimer's disease: The effects of Sitagliptin, a DPD-4 inhibitor on cognitive functions in eldery diabetic patients with or without AD

Aims: The present study aimed to evaluate effect of sitagliptin, a dipeptidyl peptidase-4 inhibitor (DPP-4I), on cognitive functions in elderly diabetic patients with and without cognitive impairment.

Methods: 253 elderly patients with type 2 DM, were enrolled in this prospective and observational study. After comprehensive geriatric assessment, the patients were divided into either sitagliptin or non-sitagliptin group.

Results: A total of 205 patients who completed the study (52 with Alzheimer's Disease (AD)) were re-evaluated 6 months later. Sixth-month evaluation revealed no difference between sitagliptin and non-sitagliptin groups in terms of weight, body mass index, and HbA1c ($p > 0.05$). However, the number of patients that required reduced insulin dose was significantly higher in the sitagliptin group ($p = 0.01$). Sitagliptin therapy was associated with an increase in the Mini-Mental State Examination (MMSE) scores ($p = 0.034$); patients without AD receiving only sitagliptin or insulin showed higher MMSE scores as compared to the patients receiving metformin alone ($p = 0.024$). Likewise, the change in MMSE scores in AD patients receiving sitagliptin was significant and indicated improvement as compared to the patients receiving metformin ($p = 0.047$).

Conclusion: Besides its effects similar to those of insulin and metformin in glycemic control and in reducing need for insulin, 6-month sitagliptin therapy may also associated with improvement of cognitive function in elderly diabetic patients with and without AD. Further randomized controlled trials are needed to support these results.

Biography

Ahmet Turan ISIK finished his graduation from Gulhane Military Medical Academy in 1995. He received specialist training in internal medicine in 2002 and became a specialist in internal medicine. In 2007, He completed geriatric side branch education and became a geriatrician. He had completed postgraduate studies both in the country and abroad on cognitive disorders and dementia (dementia) syndromes, especially Alzheimer's disease in the ages. He received associate professor position on December 2006 and Professor Title on March 2012. He is currently serving as the Head of the Department of Geriatrics at Dokuz Eylul University Medical Faculty. He is also a part-time faculty member at the Faculty of Health Sciences of Eastern Mediterranean University. His research interests reflect in his wide range of publications in various national and international journals. He is also author of many international books.

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Panteleimon Giannakopoulos

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Alzheimer disease biomarkers: Facing the complexity

Early clinic-pathological studies demonstrated that the two cardinal lesions associated with Alzheimer disease (AD), neurofibrillary tangles (NFT) and amyloid deposits, have a differential impact on cognition both at early and late stages of the neurodegenerative process. In contrast to ß-amyloid (Aß) deposition that occurs diffusely in the human brain over 60 years of age, NFT formation follows hierarchical schemes of regional and cellular vulnerability affecting first the entorhinal cortex and parahippocampal formation before moving in adjacent neocortical association areas. Long before the emergence of novel imaging techniques, it was clear that Aß deposits correlate very weekly with cognition and downstream neurodegenerative biomarkers. In contrast, NFT and associated synaptic loss is strictly related to the loss of cognitive functions not only at late but also at early stages of AD. The last decade was characterized by the exponential increase of knowledge in the field of AD predictive biomarkers and, most importantly, characterization of tracers for ß-amyloid (Aß). It is now widely acknowledged that amyloid deposits in positron emission tomography (PET) with Pittsburg compound B (PiB; a marker of Aß fibrillar deposits) precede dementia by 5-10 years, and PiB burden inversely correlates with concentration of Aß42 in the cerebro-spinal fluid. However, increased PiB burden was reported in nearly 20% to 30% of controls in the general population pointing to the fact that Aß deposition is not sufficient to cause cognitive decline in AD. Moreover, the rate of Aß accumulation is not related to neurodegeneration at baseline and only 8% of controls display both decreased hippocampal volume and increased PiB signal. According to Jack's model, all of the aforementioned markers become positive well before dementia onset, and the ones related to amyloid pathology already reach their plateau at the time of first cognitive deficits. More recently, selective tau tracers became available for clinical research. Although a PiB equivalent is not yet ready for tau imaging, the recent development of tau tracers with higher selectivity, reduced non-specific binding and improved tracer kinetics compared to the first molecules raise increasing expectations among the scientific community. Given the tight association between tau deposition, cognition and neurodegeneration, and unlike Aß imaging, tau imaging will be essential for assessing disease progression. Furthermore, they may help to resolve the controversy about the temporal sequence of tau pathology in AD. The new diagnostic criteria by Dubois and collaborators consider that the development of tau pathology, at least under its fibrillar forms, is a late phenomenon in AD dependent, at least partly, on the Aß deposition in prodromal states. Recent contributions showed that tau-related markers (but also structural MRI changes) might become positive in the absence of PiB deposits mainly in preclinical cases. Ultimately, tau imaging will provide the tool to change the landscape and explore whether or not presymptomatic administration of anti-Aß therapy impacts on the progression of tau pathology that determines the clinical expression of AD.

Biography

Panteleimon Giannakopoulos was born in 1965 in Greece. He obtained his MD degree in the University of Athens in 1989 before completing a full training on psychiatry and psychotherapy in London (Maudsley Hospital and Geneva) as well as postdoc training in Paris (La Pitié-Sâlpetrière Hospital, Federation of Neurology). In 1998, aged 33 years, he has been appointed as associate professor and medical head of the Division of Geriatric Psychiatry of the University Hospitals of Geneva. Later on (2004) he obtained the position of full tenured professor of Psychiatry in the University of Geneva. From 2003 to 2011, he also assumed a parallel position of full professor of Old Age Psychiatry in the University of Lausanne in order to promote the academic careers of junior staff locally. He has been Chairman of the Department of Mental Health and Psychiatry in Geneva for ten years (2005-2015) and vice dean of the Faculty of Medicine in the University of Geneva in charge of postgraduate and continuous education (2003-2011). From December 1st 2015, he is the medical head of the forensic psychiatry development in Geneva country. Specialist of Alzheimer disease research, he published more than 240 peer-reviewed articles in the field of neurobiology of aging with particular focus on predictive biomarkers of cognitive decline.

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Day 2

Harry S Goldsmith, J Alzheimers Dis Parkinsonism 2017, 7:4(Suppl)
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Harry S Goldsmith

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Omental transposition to the brain of Alzheimer patients

Introduction: The surgical placement of an intact vascularized omental pedicle directly on the human brain can result in a significant increase in cerebral blood flow (CBF). Placing an omental pedicle on the brain of Alzheimer (AD) patients, who are known to have a decreased CBF, may explain the cognitive improvement that has followed this surgical procedure.

Methods: The omentum is surgically lengthened with its blood supply remaining intact. Following this lengthening process the omentum is brought up through a subcutaneous tunnel placed along the chest and neck up to the head. A craniotomy is performed and the Dura mater is opened. The omentum is then simply laid on the brain without the need for any anastomoses.

Results: Omental transposition (OT) to the brain allows omental arteries to penetrate directly and deeply into the brain resulting in a marked increase in CBF. Of twenty-five advanced Alzheimer patients who underwent OT to the brain six patients showed no post-operative improvement, ten demonstrated slight changes with nine patients demonstrating marked cognitive improvement.

Conclusion: There is increasing interest that AD is the result of decreased CBF which negatively effects the intra-neuronal mitochondria which directly influences the production of neuronal adenosine triphosphate (ATP) which is the energy source of neurons. The increased CBF originating from the omentum may explain the improved cognition that has followed OT to the brain of AD patients.

Biography

Harry S Goldsmith has been a professor of surgery and neurosurgery for more than 45 years and a student of medical history throughout his life. He has invented several surgical procedures including an operation to treat Alzheimer's disease and a procedure to treat acute and chronic spinal cord injuries. He is an author of 261 papers or book chapters, has edited three surgical texts, and has received honorary degrees from two Chinese universities. He is a surgeon, worldwide lecturer, and advisor on the application of his surgical procedures..

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Ken Nagata

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Common risk factors for Alzheimer's disease and Vascular dementia

According to the recent epidemiological reports, there are common risk factors in Alzheimer's disease (AD) and vascular dementia (VaD). They can be classified into 4 major categories: demographic, genetic, vascular and comorbidity risk factors. The demographic risk factor includes gender, aging, past history of severe head injury, educational carrier and occupational attainment. Male gender is a risk for VaD and stroke, whereas female gender is known as a risk factor for AD. ApoE ε4 is known to be a possible common genetic factor for both AD and cerebrovascular disease including VaD. Obesity in midlife, lack of physical activity, cigarette smoking, and excessive alcohol intake are also regarded as risk for dementia in late life. The vascular risk factors encompass hypertension in midlife, diabetes mellitus, dyslipidemia, congestive heart failure, myocardial infarction, atrial fibrillation, and chronic kidney disease. It is suggested that effective management of these vascular risk factors in midlife prevents dementia and cognitive decline in late life. Placebo-controlled randomized clinical trials of antihypertensive drugs demonstrated that intensive antihypertensive therapy reduced the risk of VaD as well as AD in late life. Low cardiac output due to hypotension and/or congestive heart failure has been regarded as a risk factor for cognitive impairment and dementia especially in elderly patients whose autoregulation of cerebral blood flow is impaired. Although further research is needed, those evidences may support a rationale for the efficacious management of vascular risk factors in the prevention of VaD as well as AD.

Biography

Ken Nagata is currently a director of Clinical Research Institute, Yokohama General Hospital, Yokohama, Japan. He was graduated from Hirosaki University School of Medicine in 1978, and had neurology training at Mihara Memorial Hospital. He received a Ph.D. degree from Hirosaki University in 1988. He was a visiting assistant professor at the Department of Neurology, University of Colorado Health Sciences Center in Denver, USA in 1983-1984. He is a founding chairman of the Japanese Vas-Cog Society, and hosted the Vas-Cog World Congress 2015 in Tokyo. His main interests include cerebrovascular disease, dementia and neuroimaging.

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