Title	Clinical features and risk factors of cognitive impairment after stroke in Hong Kong Chinese
Author(s)	Zou, LY; Li, LSW; Cheung, RTF
Citation	The 8th Medical Research Conference, Hong Kong, China, 25-26 January 2003. In Hong Kong Medical Journal, 2003, v. 9 n. 1 Supp 1, p. 82
Issued Date	2003
URL	http://hdl.handle.net/10722/46690
Rights	Creative Commons: Attribution 3.0 Hong Kong License

NUS-21 Clinical features and risk factors of cognitive impairment after stroke in Hong Kong Chinese

LY Zou[1], LSW Li[2] & RTF Cheung[1]. [1]Division of Neurology, University Department of Medicine, The University of Hong Kong and [2]Department of Medicine, Tung Wah Hospital, Hong Kong

Introduction: Stroke is recognized as an important cause of dementia. The goal of the present study is to examine a series of clinical features and risk factors of cognitive impairment after stroke.
Method: A standard protocol was applied to 185 consecutive unselected stroke patients within two weeks after onset of their strokes. Demographic and clinical data were collected. Barthel Index was used to assess the activities of daily living and Mini Mental Status Examination (MMSE) was used to assess the cognitive function. The cutoffs on MMSE score for cognitive impairment were selected according to Crum's criteria and the education level.
Results: Seventy-seven stroke patients (41.6%) had cognitive impairment or worse. Cognitive impairment was unrelated to age, gender, marital status, handedness, type of stroke (ischaemic/haemorrhagic), side of the lesion, location of stroke, smoking habit, drinking habit, diabetes mellitus, and hyperlipidaemia. Low education level, low Barthel Index and hypertension were the independent predictors of cognitive impairment in logistic regression analysis ($P<0.05$).
Conclusion: The local incidence of cognitive impairment after stroke is high. Education level, functional impairment and hypertension may increase the risk of cognitive impairment after stroke.

NUS-22 Research strategies in molecular signaling of neuronal apoptosis in Alzheimer's disease

Raymond Chuen-Chung CHANG, Ka-Chun Suen, Man-Shan Yu, Jacques Hugon
Department of Anatomy, Faculty of Medicine, The University of Hong Kong

Introduction: Neuronal loss is a key issue in the pathogenesis of Alzheimer's disease (AD). It is evident that neuronal apoptosis is one mode of neurodegeneration. Among all the factors leading to neuronal apoptosis, _-amyloid peptides (A_) are the most important toxin in AD pathogenesis. Therefore, the molecular signaling events of neuronal apoptosis receive much attention in AD research.
Methods: Different cell culture models were employed to prove the involvement of a particular protein kinase in A_ neurotoxicity. To verify the involvement of a protein kinase, both the phosphorylation of the kinase and its substrate had to be examined. Having done these experiments, the next step was to elucidate how significance of a kinase in A_ neurotoxicity by using molecular biology technique to transfect neurons over-expressing wild-type or negatively mutated kinase. Furthermore, neurons from knockout and the wild-type mice were used to prove the findings from genetically manipulated neurons. Apart from these experiments, investigation of whether the kinase involved in real clinical case was another important strategy to show the significance of the kinase in the pathogenesis of AD. Afterwards, it is also essential to show how a particular kinase incorporates into other well-known apoptotic pathways.
Results and Conclusion: By using these strategies, we have demonstrated that a novel double-stranded RNA-dependent protein kinase (PKR) is significantly involved in A_-induced neuronal apoptosis. It also plays significant roles in the pathogenesis of AD.
Acknowledgement: The study was supported by Seed Funding Grant 2001-2002 to RCCC, and RGC grant (HKU 7305/00M) to JH.