



Title	Reduced susceptibility to ischaemic brain damage following photochemical stroke in transgenic mice overexpressing the amyloid precursor protein
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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. 81
Issued Date	2003
URL	http://hdl.handle.net/10722/46691
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NUS-19 A study of hemiplegic shoulder pain at Tung Wah Hospital

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Introduction: Hemiplegic shoulder pain was reported in 30-40% of stroke patients in Western literature and found to be more common during the spastic phase of motor recovery. It is our impression that the incidence of hemiplegic shoulder pain in Hong Kong is not as high. This study aims to investigate the incidence, diagnosis and association factors of shoulder pain in stroke survivors at Tung Wah Hospital, one of the regional rehabilitation centres of the Hong Kong West cluster.

Method: Retrospective case note review of all 114 stroke patients who were admitted into Tung Wah Hospital Stroke Rehabilitation Unit from March to June 2002. Hospital notes were meticulously scrutinised including prescription details for any consumption of analgesics and all records of the doctors, nurses, physiotherapists and occupational therapists.

Results: As on September 30, 2002, all patients were followed for at least 3 months. Nine of 114 of patients were found to have hemiplegic shoulder pain giving an incidence of 7.8%. The occurrence of shoulder pain was not significantly related to the motor power or muscle tone of the hemiplegic arm. There were also no relations with age/sex of patients; the type/side of strokes and other medical comorbidities. Causes of shoulder pain were mainly adhesive capsulitis, impingement syndrome, biceps tendinitis and glenohumeral subluxation. Most shoulder problems responded to simple analgesics and physical modalities.

Conclusion: The incidence of hemiplegic shoulder pain is rather low as compared with that reported in other Western countries but the pattern of shoulder problems is quite similar. We postulated that the low incidence of shoulder pain could be related to our active prevention programme during rehabilitation through staff, patient and caregiver training and education on proper positioning and handling of the hemiplegic arm. Furthermore, the Chinese culture, which usually values a higher pain tolerance, may be another contributing factor.

NUS-20 Reduced susceptibility to ischaemic brain damage following photochemical stroke in transgenic mice overexpressing the amyloid precursor protein

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Introduction: Amyloid precursor protein (APP) is the source of beta-amyloid, the principal component of amyloid plaques in the brain of Alzheimer's disease. Mice overexpressing APP have an increased vulnerability to brain ischaemia induced by endovascular middle cerebral artery occlusion. In this study, we investigate the role of APP in ischaemic brain damage due to photochemically induced thrombosis of cortical microvessels in transgenic mice overexpressing APP. Non-transgenic mice were used as a control group.

Method: The brains of transgenic mice overexpressing APP and non-transgenic mice were illuminated with a cold light source through the intact skull for 15 min or 3 min at 1 min following an injection of 0.1 mL or 0.04 mL of Rose Bengal respectively. Infarct volume was assessed 48 hours later from the triphenyltetrazolium chloride-stained brain slices.

Results: The relative infarct volume in the transgenic mice and non-transgenic mice following 15 min of photochemically induced thrombosis was $7.87 \pm 1.25\%$ (mean \pm S.E.M.; n=3) and $14.47 \pm 4.16\%$ (n=4), respectively. Thus, the infarct volume in the transgenic mice was reduced by 45.6% (P<0.05). The relative infarct volume in the transgenic mice and non-transgenic mice following 3 min of photochemically induced thrombosis was $1.53 \pm 1.36\%$ (n=3) and $3.48 \pm 0.64\%$ (n=4), respectively. Thus, the infarct volume in the transgenic mice was reduced by 56.0% (P=0.05).

Conclusion: We conclude that the mice brain overexpressing APP is less susceptible to ischaemic damage due to photothrombosis of cortical microvessels. Our results are different from the reported findings when focal ischaemia was induced by endovascular middle cerebral artery occlusion.