ABSTRACTS

GENERAL INTEREST

001

Prevalence of Extracranial Cerebrovascular Disease in Chinese Patients with Coronary Artery Disease

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Racial differences have been reported to account for the extremely low incidence of carotid and vertebral artery disease in Chinese and Japanese. Extracranial cerebral vascular disease (ECCVD) is associated with coronary artery disease (CAD) in Caucasians. There is no data on the prevalence of ECCVD among Chinese patients with CAD. We studied 135 consecutive patients with angiographically documented CAD. Patient demographics, vascular risk factors, history of myocardial infarction, prior neurologic events, concomitant peripheral vascular disease (PVD), carotid bruits, severity of CAD and degree of left ventricular dysfunction were evaluated. Coronary angiograms, carotid and vertebral duplex ultrasound studies were performed in all patients. Significant ($\geq$ 50%) stenosis of $\geq$ 1 of the extracranial cerebral vessels was found in 29 patients (21%) (common carotid in 3/29 patients (10%), internal carotid in 15/29 (52%; bilateral in 4), external carotid in 17/29 (59%; bilateral in 8) and vertebral in 6/29 (21%). There was no difference in demographics or vascular risk factors between CAD patients with (group I, n=29) and without (group II, n=106) ECCVD. Group I patients were more likely to have PVD (24% vs 2%, $p<0.05$). The sensitivity and specificity of a carotid bruit for carotid stenosis was 95% and 69% respectively (positive predictive value 26%, negative predictive value 99%). Conclusion: 1) Among Chinese patients with CAD, significant stenosis in the extracranial cerebral vessels is not uncommon. 2) Apart from PVD, other patient demographics and risk factors are equally prevalent in those CAD patients with and without ECCVD. 3) Presence of carotid bruit is a sensitive test in identifying those with significant ECCVD.

002

DOES LOW MOLECULAR WEIGHT HEPARIN REDUCE RESTENOSIS AFTER CORONARY STENTING?

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In 1995, 188 Chinese (142 males, mean age 61.4± 9.9) underwent 196 percutaneous transluminal coronary stenting electively in 148 and ad-hoc in 48. Indications for stenting were restenosis in 24, bail-out in 5 and de novo stenting for the rest. 235 stents were implanted in 206 vessels and mean vessel diameter was 3.24±0.36mm. High pressure intrastent inflation (>10 atmospheres) was employed. In phase 1 all patients received subcutaneous low molecular weight heparin (LMWH), nadroparin 5 000 U twicely daily for 7 to 14 days. In phase 2, LMWH was indicated only for suboptimal result/recent myocardial infarction. The aspirin/ticlopidine regime was used. The gender, age, baseline clinical characteristics and risk factors were equally distributed and the degree of coronary arterial stenosis and the left ventricular function were similar between the LMWH group (101 patients) and the no LMWH group (87 patients). Procedural success was 98.4%. Vascular complications occurred in 12 (6.1%) with five groin haematoma in LMWH group and 3 in no LMWH group, 1 pseudoaneurysm and 1 arteriovenous fistula in each group. There was 1 death and 1 subacute thrombosis (SAT). Follow up from 9 to 21 months 140 patients remained asymptomatic with one unrelated death. 46 patients with symptoms were restudied and 41 had angiography among whom 22 had patent stents, 19 patients developed restenosis (RS)(including 1 SAT), 17 had successful rePTCA and 2 were referred for bypass. 10 additional balloon angioplasties were needed for out-of-stent stenoses. Clinical RS was 24.7% and angiographic RS was 10.2%. In the LMWH group there were 5 RS whereas in the no LMWH group there were 14 (p value < 0.05). LMWH does have an antiporliferative effect on balloon injured arteries of hypercholesterolaemic animals whereas there is no clinical trial to suggest that the use of LMWH after balloon angioplasty would reduce RS in human. Coronary stenting activates more the platelet aggregation system and the use of ticlopidine/aspirin has circumvented the problem of SAT. The mechanism of RS as confirmed by intravascular ultrasound has shown that late luminal loss after angioplasty is due to a predominant effect of remodelling whereas after stenting this is mainly due to intimal hyperplasia. It is possible that the use of LMWH immediately after stenting might have ameliorated endothelial and intimal proliferation which is reflected in the longer term as reduced RS. The role of LMWH in coronary stenting has yet to be defined and the question remains whether it is cost-effective to use LMWH after stenting so as to reduce the number of RS and the associated morbidity and need for repeat revascularisation.