QUANTITATIVE ANALYSIS OF CEREBRAL PERFUSION AND RESERVE WITH TECHNETIUM-99M ETHYL CYSTEINATE DIMER SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY AND ACETAZOLAMIDE FOLLOWING CAROTID ARTERY STENTING.

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BACKGROUND AND PURPOSE: Cerebral perfusion through stenosed internal carotid arteries is usually maintained by autoregulation. However, cerebral perfusion reserve may be reduced, suggesting hemodynamically significant stenosis, and such reduction should be improved after treatment. In this study, cerebral perfusion and reserve in patients with internal carotid artery stenosis were quantified before and after carotid artery stenting.

METHOD: Ten patients (six male, four female, mean age 63.4 years, range 26 - 78 years) with unilateral (6/10) or bilateral (4/10) internal carotid artery stenosis were studied. Five patients had history of stroke and three patients had history of transient ischaemic attacks, including one with both. Three patients were entirely asymptomatic. For each patient, cerebral perfusion was evaluated with 4 sets of Tc-99m ethyl cysteinate dimer (ECD) single photon emission computed tomography (SPECT) both at baseline (with patient resting) and after administration of one gram of acetazolamide before and after carotid artery stenting. Four regions-of-interest (ROIs) were placed automatically in the cortex of left and right hemispheres in the mid-thalamic and mid-ventricular transaxial slices for each SPECT scan. The average count density of the two ROIs from the lesion side (LN) was compared with the average count density of the two ROIs from the contralateral side (CL) to give the cerebral perfusion index (CPI), which reflects cerebral perfusion of the territory supplied by the stenosed carotid artery. CPI = (LN - CL)/(LN + CL) x 200%. The side of operation was defined as the lesion side in case of bilateral stenosed arteries. Cerebral perfusion reserve (CPR) was calculated by subtracting CPI of baseline scan (BL) from CPI of the corresponding acetazolamide scan (ACZ). CPR = CPI(ACZ) - CPI(BL). Values of CPI and CPR were obtained from both preoperative scans (Pre) and Postoperative scans (Post) respectively.

RESULTS: The CPI(Post) was -5.61 \pm 8.54% (mean \pm SD) and was not statistically different from the CPI(Pre) (-5.01 \pm 6.49%, n = 10, p = 0.62, paired t test), showing that no significant change in cerebral perfusion occurred following carotid artery stenting. The CPR(Post) was 0.17 \pm 2.23% and was significantly higher than the CPR(Pre) (-3.01 \pm 4.13%, n = 10, p < 0.03, paired t test), showing improved perfusion reserve following the procedure. This improvement in CPR, \triangle CPR = CPR(Post) - CPR (Pre), correlates best negatively with CPR(Pre) (r = -0.85) and less good with CPI(Pre) (r = -0.78). Therefore, a poor preoperative CPR value may predict a good response to carotid artery stenting.

CONCLUSIONS: Reduction in cerebral perfusion reserve existed in patients with hemodynamically significant carotid artery stenosis. SPECT at baseline and with acetazolamide could determine such reduction, identify patients who are likely to benefit from carotid artery stenting and demonstrate improvement in cerebral perfusion reserve following the procedure.

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ACTIVE CARDIAC REHABILITATION PROGRAM IS ABLE TO IMPROVE RISK FACTORS PROFILE

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Objective: This study investigated the role of an active cardiac rehabilitation programme (CRP) in modifying cardiovascular risk factors.

Design and methods:100 patients after acute myocardial infarction or coronary angioplasty were randomised to either an active CRP (consists of education, supervised exercise training and medical therapy) or conventional medical therapy on a 2:1 ratio. The laboratory results, exercise capacity, body weight and smoking status were assessed at the end of each phase.

Result: The mean age and gender in the 2 groups were comparable ($65.6 \pm 11.1 \text{ Vs}$ $65.5 \pm 9.5 \text{ years}$; M:F=7:3, both p=NS). In the active group, the gain in exercise time is significantly more than the conventional group ($2.2 \pm 2.5 \text{ Vs}$ $0.7 \pm 1.4 \text{ METS}$, p=0.001). The body weight is not changed in the active group but increase in the conventional group ($65 \pm 11 \text{ Vs}$ 67 ± 13 , p=0.01). Patients quitted from smoking since phase 2 (X^2 =49, p<0.001), though the rate is not difference in the two groups. There is no difference in the degree of in cholesterol reduction.

Conclusion: Active CRP in the form of supervised exercise and education improved risk factors profile in patients after AMI or PTCA than conventional medical therapy alone.