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Carotid Endarterectomy Under Local Anaesthesia does not Increase Plasma Homocysteine Concentration

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Introduction. *The purpose of this cohort study was to evaluate the effect of carotid endarterectomy under local anaesthesia on homocysteine (Hcy) concentrations.*
Patients and methods. *Of 100 patients with internal carotid artery (ICA) stenosis >70%, the complete data set was available for 91 patients (39 asymptomatic and 52 symptomatic). All patients underwent eversion endarterectomy of the ICA under regional anaesthesia.*
Results. *Thirty-two percent of the examined patients had a total Hcy above 15 μmol/l. The mean Hcy levels preoperatively were 13.9±4.8 μmol/l. The Hcy levels on day 5 were 13.1±5.0 μmol/l and after 6 months 14.0±5.8 μmol/l. There was no significant change during follow-up. No intraoperative strokes and deaths were observed and during the 6 months follow-up no recurrent strokes, TIAs or deaths occurred.*
Conclusion. *Patients undergoing carotid endarterectomy under regional anaesthesia do not have an increase in total Hcy postoperatively. This finding is in contrast to results from cardiac surgery and carotid endarterectomy in a recently published animal study, both performing surgery under general anaesthesia.*

Keywords: Homocysteine; Carotid endarterectomy; Regional anaesthesia; Risk factor.

Introduction

Homocysteine (Hcy) is a sulphur-containing amino acid formed during methionine metabolism.[1] Elevated plasma homocysteine levels are a recognised independent risk factor for coronary, cerebral, and peripheral arterial occlusive disease.[2–4] Literature regarding the effect of operative procedures on plasma homocysteine levels are sparse. There is growing evidence that homocysteine may play a role in postprocedural intimal hyperplasia and arterial thrombosis and, therefore, the effect of surgery on homocysteine levels is important.

In humans, it was reported that coronary bypass grafting leads to sustained elevation of homocysteine levels.[5] In a recently published animal study, it was shown that carotid endarterectomy under general anaesthesia leads to sustained and marked elevation of homocysteine.[6] This study suggested that anaesthetics alone, surgical dissection, and manipulation of the intima within the artery were not the prime aetiological factors in producing elevated homocysteine levels. The authors concluded that the carotid artery itself needs to be clamped and an endarterectomy performed to produce a significant six-fold increase in plasma homocysteine.[6]

The purpose of this cohort study was to evaluate the effect of carotid endarterectomy under local anaesthesia on plasma homocysteine levels in patients undergoing elective carotid endarterectomy.

Patients and Methods

Patient recruitment

One hundred consecutive patients with symptomatic and asymptomatic internal carotid artery (ICA) stenosis >70% who were admitted to our department for surgery were enrolled in the study. All patients were planned for routine eversion endarterectomy

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(EEA) of the ICA under regional anaesthesia. As an anaesthetic agent, 1% lidocaine was routinely used for a combined superficial and deep cervical block. All patients underwent preoperatively duplex scanning and magnetic resonance angiography to assess the maximum lumen reduction of the ICA. Symptomatic patients with stroke had an interval of less than 6 weeks prior to surgery; symptomatic patients with transient hemispheric and non-hemispheric symptoms were all operated on within 3 weeks of presentation. Patients were counselled and encouraged to reduce cardiovascular risk-factors and to eat a healthy diet. Patients on vitamin supplementation were excluded from the study. All patients had a preoperative measurement of fasting total serum homocysteine 1 day prior to surgery. The second blood sample for fasting homocysteine testing was drawn on day 5 after surgery; the final sample was obtained 6 months after surgery. All patients gave written informed consent. The study was approved by the local ethics committee.

Patients

The demographic data of patients are listed in Table 1. All three-blood samples were available for 91 patients. Of the 91 patients included in the evaluation, 53 were male and 38 were female. The age ranged from 41 to 86 years (median = 69; Q25th = 62; Q75th = 77). Thirty-nine patients (43%) had asymptomatic ICA stenosis, 32 patients (35%) had transient ischaemic episodes and 20 patients (22%) were operated on for non-disabling stroke.

Sample handling and homocysteine measurement

Blood was drawn by venipuncture of the antecubital vein and collected in standard K3-EDTA tubes (Vacuette EDTA Tubes, Greiner Bio-One, Kremsmünster, Austria). Blood samples were centrifuged (10 min at 2000 *g*) and plasma was decanted within 15 min after sampling. Plasma samples were stored at −80 °C until analysis. Homocysteine measurements were performed on an Abbott AxSYM Plus Immunology analyzer (Abbott Diagnostics, Abbott Park, IL, USA) using Abbott AxSYM Hcy reagents according to manufacturer's instructions. The sensitivity of the AxSYM homocysteine assay was assessed to be 0.8 μmol/l by replicate analyses of the zero-level calibrator. Sensitivity was defined as the concentration at two standard deviations from the mean and represents the lowest measurable concentration of homocysteine that can be distinguished from zero. The coefficient of variation (CV) resulting from intra-assay precision experiments was between 4.5 and 1.4% at concentrations ranging from 7.6 to 28.2 μmol/l. Evaluating inter-assay imprecision in the same concentration range the CV was between 2.0 and 5.1%.

Statistics

The results were analysed using a software package (Epi-Info 2002 software package, Centers for Disease Control and Prevention, GA, Atlanta). Continuous variables were analysed to evaluate normality of distribution. When data were normally distributed they were presented as mean ± SD; means were compared with a Student's *t*-test for unpaired variables. When distribution was non-normal, variables were expressed as median and percentile range (Q25th; Q75th) and compared with the Wilcoxon rank sum test. Discrete variables were expressed as numbers (percentages) and compared by chi-square test or by Fisher's exact test. To calculate linear correlation for comparing three unmatched groups, the Kruskal-Wallis test was performed. All tests of

Table 1. Patients' basic demographic data

	Male ($n=53$)	Female ($n=38$)	Total ($n=91$)	*P*-value
Age	41–83; 70 (62; 76)	47–86; 68 (59; 79)	41–86; 69 (62; 77)	0.912
BMI	27.1 ± 4.1	26.1 ± 3.7	26.7 ± 3.9	0.252
Alcohol g/week	0–805; 25 (0;120)	0–405; 0 (0;0)	0–805; 0 (0;50)	0.006*
Nicotine (cigarettes/day)	0–80; 0 (0;10)	0–25; 0 (0;10)	0–80; 0 (0;10)	0.369
Hypertension	41 (77%)	32 (84%)	73 (80%)	0.418
Diabetes	21 (39%)	4 (10%)	25 (27%)	0.002*
ICA stenosis, asymptomatic	20 (38%)	19 (50%)	39 (43%)	0.244
ICA stenosis, TIA	19 (36%)	13 (34%)	32 (35%)	0.872
ICA stenosis, stroke	14 (27%)	6 (16%)	20 (22%)	0.227

Basic demographic data. BMI, body mass index; TIA, transient ischaemic attack; *, statistically significant results; *p*-values of < 0.05 were considered significant.

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