Protected carotid artery stenting: high frequency of silent cerebral embolic lesions detected by DWI

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Introduction. Increasing evidence indicates that carotid artery stenting (CAS) represents an alternative to surgical carotid endarterectomy (CEA) in the management of symptomatic internal carotid artery stenosis in high risk patients [1]. However, randomised controlled trial studies have demonstrated that stroke and death rates for CAS range from 4.4% to over 12% at 30 days [2], although the use of neuroprotective devices may reduce the risk of cerebral embolisation [1]. The pathophysiology of neurological events among such patients is still rather unclear.

To evaluate possible determinants of cerebral embolism during CAS we have evaluated the incidence, distribution, and size of embolic brain lesions with Diffusion Weighted Imaging (DWI), correlating these findings with the morphology of both the aortic arch and the carotid bifurcation.

Methods. 23 consecutive patients (13 males, aged 76.54 ± 4.62 years) underwent TransEsophageal Echocardiography and epiaortic ultrasound before CAS. Aortic arch lesions were classified into complex and non-complex lesions. Neurological and DWI evaluations were carried out before and within 24 hours of CAS. CAS was carried out in all cases under antiplatelet and anticoagulant protection, distal filter protection, primary stenting and routine postdilation. DWI studies were performed in a 1.5T General Electrics Medical Systems (Milwaukee, Wisconsin) Signa Horizon LX whole-body scanner. Axial DW images were obtained (thickness = 5 mm, inter-slice gap = 0 mm) using a single-shot EPI sequence (matrix size = 192 x 192 mm), as previously reported (3). Diffusion encoding gradients were applied in six directions with gradient strength corresponding to b-values of 900 mm^2/s. In addition, images without diffusion weighting were acquired corresponding to b = 0 mm^2/s and exhibiting a T_2-contrast. The apparent diffusion coefficient (ADC) of each direction was determined pixel-wise assuming a signal attenuation depending mono-exponentially on the b-value. The mean diffusivity map was then generated from the tensor. Pre-CAS images were automatically mapped onto the post-CAS images by rigid body registration (FLIRT; FSL, fMRIB, Oxford [4]). Lesions were identified on the post-CAS images, and the identification confirmed by the absence of hyperintensity on the corresponding pre-CAS images. The volume and mean ADC of lesions were assessed semi-automatically.

Non–parametric statistical analysis was performed first to relate the incidence of DWI lesions to possible patient risk factors and other variables, such as age and sex. The same variables were then used to assess the distribution and quantity of lesions when found.

Results Among the 23 consecutive asymptomatic patients who underwent CAS early neurological complications were observed in two patients (1 hemispherical transient ischemic attack and 1 minor stroke) less than 24 hours after CAS. Clinical follow-up at 6 months is currently in progress. Non-complex lesions of the aortic arc were identified on TransEsophageal Echocardiography prior to CAS in 14 cases and complex lesions in 9 cases.

After CAS, embolic lesions of different sizes appeared on DWI images in 16/23 cases overall. The median number of lesions was 2 (minimum 0-maximum 16), while median volume was 0.4 cc (0.09 to 12 cc). Lesions were not strongly lateralised to CAS side (see table).

Laterality	No. of cases	Ipsilateral	Contralateral
None	7		
Unilateral	6	5	1
Bilateral	10	3 predominantly	2 predominantly

Table showing distribution of laterality of DWI lesions referenced to CAS side.
Cases with more than 75% of lesion volume on one side were considered to be predominantly lateralised.

All cases with complex aortic arc lesions (9/9), but only 50% of cases (7/14) with non-complex lesions presented new DWI lesions.

No correlations were found between the presence of new lesions on DWI and other variables, particularly: age, plaque echogenicity and morphology, and the presence of chronic ischemic lesions on MRI. Likewise, new lesion number, size and mean ADC were not significantly correlated with these variables.

Conclusions

CAS may lead to silent cerebral embolisation in both ipsi- and contralateral hemispheres with roughly equal frequency, and the new embolic lesions correlated with the severity of pre-existing aortic arch disease: patients with aortic arch complex lesions have much greater risk of developing embolisation. Catheterization and carotid cannulation through a diseased aortic arch may be an important determinant of neurological complications during CAS.

References.

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