Association between the volume of carotid artery plaque and its sub-components and the volume of white matter lesion

Poster No.: C-1179
Congress: ECR 2013
Type: Scientific Exhibit
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Keywords: Neuroradiology brain, CT-Angiography, MR, Diagnostic procedure
DOI: 10.1594/ecr2013/C-1179

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Purpose

In some studies, the amount of cerebral white matter disease and the severity of the carotid artery disease have been shown to be correlated. Our aim is to evaluate the association between the severity of white matter disease and the volume of the different components of the carotid artery plaque.
Methods and Materials

In this study 54 consecutive patients (mean age 72 ± 9 years, males 42) with carotid artery stenosis were prospectively recruited at our Institution before undergoing carotid endarterectomy.

The patients underwent an admission head/neck computed tomography angiography (CTA) and an MRI of the brain which included T2-FLAIR, T2-SE and DWI sequences.

Each patient underwent CTA of the carotid arteries with a 16-multi-detector-row CT system (Philips Brilliance, Eindhoven, Netherlands). Examination was performed by injecting 70-80 mL of contrast medium (Ultravist 370; Bayer, Leverkusen, Germany) at a flow rate of 5 mL/s. A bolus tracking technique was used to calculate the correct timing of the scan. CT technical parameters included: matrix 512x512, field of view (FOV) 14-19 cm; mAs 180-220; kV 120-140; an intermediate reconstruction algorithm (C-filter) was used. Angiographic acquisition included the carotid siphon. None of the patients included in the study had a medical history of cardiac output failure, or any contraindications to iodinated contrast media.

Imaging examinations were performed on a Gyroscan 1.5-T superconducting magnet (Philips, Best, The Netherlands) with a head coil. The following sequences were performed in our brain protocol: (a) Axial DWI with single-shot spin-echo with 2 b values of 0 and 1000 s/mm2; (b) axial spin-echo T1-weighted sequence (500-600/15/2 for TR/TE/excitations) with flip angle, 30°; matrix, 512 x 512; field of view (FOV) 240 mm2, section thickness of 5 mm; (c) axial fast spin-echo T2-weighted images (2200-3200/80-120/1.2 for TR/TE/excitations; turbo factor 4, matrix, 512 x 512; FOV, 240 mm2; section thickness, 5 mm; (d) Axial and sagittal 2D FLAIR (10000/140/2200 for TR/TE/TI) matrix, 512 x 512; FOV, 240 mm2; section thickness, 5 mm; (e) Axial 2D T2*-weighted GRE (TR shortest; TE 23 ms; flip angle 15°; in-plane resolution 0.9x0.9 mm; FOV 230 mm2; matrix 512x512; slice thickness 5 mm. (f) MR angiography of the circle of Willis was performed with a 3D multislab TOF sequence from the petrous portion of the internal carotid artery to the corpus callosum (25-40/4-8/1 for TR/TE/excitation, flip angle of 20°, section thickness of 0.8 mm, FOV of 250 mm2, matrix of 256 × 256.

The CTA-based volume, as well as the percentages of the 3 main plaque components (fatty, mixed, calcified) were calculated according to the attenuation values. FLAIR-leukoaraiosis lesion volume was performed using a semi-automated segmentation technique (Jim, Xinapse System, Leicester, UK). Pearson correlation was conducted between the FLAIR-leukoaraiosis lesion volume and the volumes of the different plaque components.
Results

No patients were excluded. For each hemisphere the mean WML volume was 2210 mm³ (SD 1132 mm³). The mean WML volume in the right hemisphere was 2314 mm³ (SD 1178 mm³) whereas the contralateral hemisphere was 2106 mm³ (SD 1056 mm³). Mann-Whitney test showed that there was no statistically significant difference in the volume of WML between the right and left hemispheres (p value of 0.813).

The mean carotid artery plaque volume was 957 mm³ (SD 580 mm³). The mean plaque volume of the right carotid was 1035 mm³ (SD 634 mm³) whereas the left carotid mean plaque volume was 898 mm³ (SD 536 mm³). Mann-Whitney test showed that there was no statistically significant difference in plaque volume between right and left side (p value of 0.385).

Pearson correlation demonstrated the following values for FLAIR-leukoaraiosis lesion volume versus total carotid plaque volume (rho = 0.2608, p value = 0.0248), fatty plaque volume (rho = 0.394, p value = 0.0005), mixed plaque volume (rho =0.1732, p value = 0.14) and calcified plaque volume (rho = 0.0151, p value 0.8986). The following values were obtained by comparing the FLAIR-leukoaraiosis lesion volume versus fatty plaque percentage (rho = 0.3652, p value = 0.0014), mixed plaque percentage (rho = -0.1852, p value = 0.1142) and calcified plaque percentage (rho = -0.2159, p value 0.0646).
Fig. 1: Figure 1: axial Flair image demonstrating the leukoaraisis

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Fig. 2: Figure 2: CTA axial image with the automated volume analysis example in a 75 yo patient with an atherosclerotic plaque in the right carotid artery.

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**Fig. 3:** Figure 3: scatter plot

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**Fig. 4:** Figure 4: scatterplot

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Conclusion

In the past years it was thought that the WML was determined by the vasculature disorders of the small intra-parenchymal cerebral arteries or arterioles but some recent investigations have demonstrated that the severity of WML is strongly associated with the atherosclerotic disease of the large vessels. In our study we aimed to evaluate the association between the severity of WML and the volume of the carotid artery plaque (and the volume of its different components) in order to assess the potential effect of the plaque\volume composition.

We found that the total carotid artery volume is statistically associated with the WML volume and in particular the volume of the fatty components in markedly associated with the WML whereas no statistically significant association was found between the mixed plaque and calcified plaque volume.

Results of our study indicate that the volume of the cerebral WML and the total volume of the plaque are correlated and that the amount of fat within the plaque is an additional risk factor, while the calcified component seems to be a protecting factor. Larger studies are warranted to confirm this finding and explore the potential clinical impact of the plaque volume\composition and WML.
References


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