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Gadolinium enhancement and white matter lesion in extracranial carotid artery aneurysms

V.E.C. Pourier, C.J.H. van Laarhoven, J. Hendrikse, T. Leiner, MDI Vergouwen, G.J. de Borst

ABSTRACT

Background: The exact etiology of extracranial carotid artery aneurysms (ECAA) and its clinical outcome when treated conservatively are unknown. Gadolinium-enhancement of aneurysm wall has been described as a possible indicator of inflammation which might play a role in aneurysm formation and growth. Also, carotid artery disease related white matter lesion (WML) are considered markers for cerebral tissue damage and increased stroke risk.

Methods: In this study, vessel wall magnetic resonance angiography (MRA) with gadolinium administration was prospectively performed in 15 patients with 17 ECAAs. Primary endpoints were 1) arterial wall gadolinium enhancement (single observer) and 2) the presence of WML (two independent observers), scored at two time points. The intra- and interobserver agreement was calculated using Kappa statistics with 95% confidence interval.

Results: Gadolinium enhancement was observed in 14 of the 17 ECAAs. The intra-observer agreement was excellent for gadolinium enhancement with Kappa of 0.82 (95% I 0.58-1.1). The presence of ipsilateral WML scored by two radiologists, ranged from 7 to 11 of the 15 patients. The intra0observer agreement for WML ranged from substantial (Kappa 0.70, 95% CI 0.14-0.98) to almost perfect (0.91, 95% CI 0.74-1.0). The inter-observer agreement was moderate (0.45, 95% CI 0.13-0.77).

Conclusions: Arterial wall imaging with MRA revealed gadolinium enhancement in the majority of ECAA and most patients had ipsilateral WML.

BACKGROUND

MRI imaging has been suggested as a tool in the early detection of arterial wall changes and may be useful for routine monitoring and evaluation of disease activity. Also, the presence of white matter lesions (WML) is a sign of micro-embolism events and its presence may predict future cerebrovascular risk and cognitive decline over time.

PURPOSE

To determine the presence ECAA disease activity and its effect on the brain to possibly predict future cerebrovascular outcome.

METHODS

MRA with gadolinium administration was prospectively performed in 15 patient with 17 ECAAs. Enhancement was considered present if there was a hyperintensity of the wall on the MRI after gadolinium administration, that was not present on the MRI before gadolinium administration. Two experienced neuro-radiologists (blinded) independently scored two rounds of the MRI images, with a minimum of two weeks between the measurements, scoring the presence of gadolinium enhancement and WML. The agreement on gadolinium enhancement and the presence of WML was calculated using Kappa statistics.

RESULTS

Gadolinium enhancement was present in 14 of the 17 ECAAs.

The intra-observer agreement for gadolinium enhancement was excellent with a Kappa of 0.82 (95% CI: 0.58-1.0).

WML presence ranged from 7 to 11 of the 15 patients.

The intra-observer agreement for WML using the Fazekas scale ranged from substantial to excellent with a Kappa of 0.70 and 0.91, with an average agreement of 0.81.

RESULTS

Figure 1. Unilateral gadolinium wall enhancement (red)

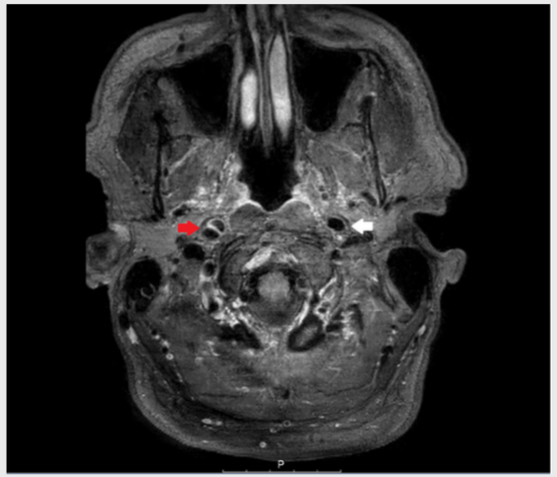
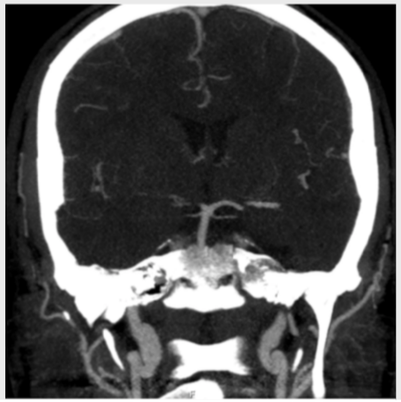


Figure 2. CT scan of same person as figure 1 showing bilateral ECAA



RESULTS

When using a binary scale, the intra-observer agreement ranged from 0.82-0.87, with an average agreement of 0.84. The inter-observer agreement was moderate (Kappa 0.59).

Table 1. inter- and intra-observer agreement on Gadolinium enhancement

Intra- observer agreement
0.82 (0.58-1.0)

Table 2. inter- and intra-observer agreement on WML

Inter- observer agreement	Inter- observer agreement	Intra observer agreement	Average intra-observer agreement
0.46 (0.13-0.77)	0.70 (0.42-0.98)	0.91 (0.74-1.0)	0.81
0.59 (0.21-0.97)	0.87 (0.62-1.15)	0.82 (0.47-1.15)	0.84

CONCLUSION

Gadolinium enhancement and WML are present in asymptomatic ECAAs.

If future studies prove that vessel wall enhancement and an increase in white matter lesions are present in these asymptomatic ECAAs, then vessel wall MRA may cause a change in the clinical approach and management of asymptomatic ECAAs.

DISCLOSURES

None