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Magnetic resonance angiography of intracranial and extracranial arteries in patients with spontaneous migraine without aura: a cross-sectional study

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Summary

Background
Extracranial arterial dilatation has been hypothesised to be the cause of pain in patients who have migraine without aura. To test that hypothesis, we aimed to measure extracranial and intracranial arteries during attacks of migraine without aura.

Methods
In this cross-sectional study, we recruited patients aged 18—60 years from the Danish Headache Centre and via announcements on a Danish website. We did magnetic resonance angiography during spontaneous unilateral migraine attacks. Primary endpoints were difference in circumference of extracranial and intracranial arterial segments comparing attack and attack-free days and the pain and the non-pain side. The extracranial arterial segments measured were the external carotid (ECA), the superficial temporal (STA), the middle meningeal (MMA), and the cervical part of the internal carotid ($ICA_{cervical}$) arteries. The intracranial arterial segments were the cavernous ($ICA_{cavernous}$) and cerebral ($ICA_{cerebral}$) parts of the internal carotid, the middle cerebral (MCA), and the basilar (BA) arteries. This study is registered at Clinicaltrials.gov, number NCT01471314.

Findings
Between Oct 12, 2010, and Feb 8, 2012, we recruited 78 patients, of whom 19 women had a scan during migraine and were included in the final analysis. On migraine compared with non-migraine days, we detected no statistically significant dilatation of the extracranial arteries on the pain side (ECA, mean difference 1·2% [95% CI −5·7 to 8·2] p=0·985, STA 3·6% [−3·7 to 11·0] p=0·532, MMA 1·7% [−1·7 to 5·2] p=0·341, and $ICA_{cervical}$ 2·3% [−0·3 to 4·9] p=0·093); the intracranial arteries were more dilated during attacks (MCA, 13·0% [6·4 to 19·6] p=0·001, $ICA_{cerebral}$ 11·5% [5·6 to 17·3] p=0·0004, and $ICA_{cavernous}$ 11·4% [5·3 to 17·5] p=0·001), except for the BA (1·6% [−2·7 to 5·9] p=0·621). Compared with the non-pain side, during attacks we detected dilatation on the pain side of the intracranial arteries (MCA, mean difference 10·5% [0·7—20·3] p=0·044, $ICA_{cerebral}$ (14·4% [4·6—24·1] p=0·013), and $ICA_{cavernous}$ (9·1% [3·9—14·4] p=0·003) but not of the extracranial arteries (ECA, 2·1% [−3·8 to 9·2] p=0·238, STA, 3·6% [−3·7 to 10·8] p=0·525, MMA, 2·7% [−1·3 to 5·6] p=0·531, and $ICA_{cervical}$, 5·0% [−0·5 to 10·4] p=0·119).

Interpretation
Migraine pain was not accompanied by extracranial arterial dilatation, and by only slight intracranial dilatation. Future migraine research should focus on the peripheral and central pain pathways rather than simple arterial dilatation.

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