European Journal of Radiology 83 (2014) 1442–1447

Contents lists available at ScienceDirect

European Journal of Radiology

journal homepage: www.elsevier.com/locate/ejrad

MRI evidence for preserved regulation of intracranial pressure in patients with cerebral arteriovenous malformations

Felix G. Meinel[a], Judith Fischer[a], Andreas Pomschar[a], Natalie Wöhrle[a], Inga K. Koerte[a], Denise Steffinger[a], Rüdiger P. Laubender[b], Alexander Muacevic[c], Maximilian F. Reiser[a], Noam Alperin[d], Birgit Ertl-Wagner[a,*]

[a] Institute for Clinical Radiology, Ludwig-Maximilians-University Hospital, Marchioninistr. 15, 81377 Munich, Germany
[b] Institute of Medical Informatics, Biometry and Epidemiology, Ludwig-Maximilians-University, Marchioninistr. 15, 81377 Munich, Germany
[c] European Cyberknife Center Munich, 81377 Munich, Germany
[d] Department of Radiology, Miller School of Medicine, University of Miami, Miami, FL 33136, USA

ARTICLE INFO

Article history:
Received 21 March 2014
Accepted 6 May 2014

Keywords:
Arteriovenous malformations
MRI
Phase-contrast MRI
Blood flow
Cerebrospinal fluid

ABSTRACT

Purpose: The purpose of this study was to investigate intracranial pressure and associated hemo- and hydrodynamic parameters in patients with cerebral arteriovenous malformations AVMs.
Methods: Thirty consecutive patients with arteriovenous malformations (median age 38.7 years, 27/30 previously treated with radiosurgery) and 30 age- and gender-matched healthy controls were investigated on a 3.0 T MR scanner. Nidus volume was quantified on dynamic MR angiography. Total arterial cerebral blood flow (tCBF), venous outflow as well as aqueductal and craniospinal stroke volumes were obtained using velocity-encoded cine-phase contrast MRI. Intracranial volume change during the cardiac cycle was calculated and intracranial pressure (ICP) was derived from systolic intracranial volume change (ICVC) and pulse pressure gradient.
Results: TCBF was significantly higher in AVM patients as compared to healthy controls (median 799 vs. 692 mL/min, $p = 0.007$). There was a trend for venous flow to be increased in both the ipsilateral internal jugular vein (IJV, 282 vs. 225 mL/min, $p = 0.16$), and in the contralateral IJV (322 vs. 285 mL/min, $p = 0.09$), but not in secondary veins. There was no significant difference in median ICP between AVM patients and control subjects (6.9 vs. 8.6 mmHg, $p = 0.30$) and ICP did not correlate with nidus volume in AVM patients ($\rho = -0.06$, $p = 0.74$). There was a significant positive correlation between tCBF and craniospinal CSF stroke volume ($\rho = 0.69$, $p = 0.02$).
Conclusions: The elevated cerebral blood flow in patients with AVMs is drained through an increased flow in IJVs but not secondary veins. ICP is maintained within ranges of normal and does not correlate with nidus volume.

1. Introduction

It has been shown that cerebral AVMs are associated with a local dysfunction in autoregulation of cerebral blood flow [1,2]. Vascular injury, abnormal endothelial signaling, microshunt formation, and venous hypertension have been identified as potential mechanisms of impaired autoregulation in cerebral AVMs [2,3]. The dysbalance of autoregulation may drive growth and remodeling of AVMs [2]. Other authors have hypothesized that cerebral AVMs may originate as a compensatory response to a local imbalance in autoregulation of blood flow [4]. A recent study suggests that impaired cerebrovascular reserve in peri-nidal brain areas associated with venous congestion may account for seizures [5]. Other authors, however, have described preserved autoregulatory responsiveness in brain areas adjacent to AVMs [6,7].

While there is extensive research about local autoregulation in perinidal areas, little is known about how cerebral AVMs affect global cerebral autoregulation and ICP. Papilledema caused by intracranial hypertension is a rare but recognized presentation

Abbreviations: AVMs, arteriovenous malformations; CSF, craniospinal fluid; ICP, intracranial pressure; ICVC, intracranial volume change; tCBF, total (arterial) cerebral blood flow; ICAs, internal carotid arteries; VAs, vertebral arteries; IJVs, internal jugular veins; VVs, vertebral veins; EVs, epidural veins; DCVs, deep cervical veins.

* Corresponding author at: Institute of Clinical Radiology, Ludwig-Maximilians-University, Großhadern Campus, Marchioninistr. 15, 81377 Munich, Germany.
E-mail address: birgit.ertl-wagner@med.uni-muenchen.de (B. Ertl-Wagner).

http://dx.doi.org/10.1016/j.ejrad.2014.05.011
0720-048X/

of unruptured cerebral AVMs [8,9–12]. High-volume shunts may potentially exhaust the venous drainage capacity and thus lead to congestion of the venous system, associated with an increase in cerebral blood volume, impairment of CSF absorption, and increase in CSF production [10,13]. Direct mass effect may be another mechanism of AVM-associated intracranial hypertension [9,10]. In one recent case series, 3 out of 4 cases of intracranial hypertension (identified as papilledema) associated with cerebral AVMs were considered to be due to overload of draining veins, while 1 case was caused by thrombotic occlusion of the straight sinus [8]. All four cases occurred in patients with large cerebral AVMs. The diagnosis of intracranial hypertension had immediate therapeutic consequences in these patients, as partial endovascular embolization was performed and immediately reduced the massive overload of the venous system and thus relieved neurological symptoms [8]. Identifying patients with cerebral AVMs at risk for intracranial hypertension and monitoring their ICP may therefore be of significant clinical relevance in order to prevent serious complications.

We aimed to assess how the presence of a cerebral AVM affects ICP depending on nidus volume. Moreover, we aimed to analyze relevant hydro- and hemodynamic parameters that may compensate the increased flow in the AVM.

2. Methods

2.1. Informed consent and ethical approval

Institutional Review Board approval was obtained prior to the commencement of the study. Written consent was obtained from all patients and healthy individuals (parents or legal guardians for minors) prior to enrollment.

2.2. Patient population and control subjects

30 consecutive patients with cerebral AVMs referred to MR imaging were enrolled in the study. All examinations were performed for clinical indications. Both treatment-naïve patients and patients previously treated with radiosurgery were included. For each patient, a healthy individual of the same gender and with no more than 12 months of difference in age served as a paired reference. To minimize confounding of perfusion parameters, exclusion criteria for both patients and control participants included a history of diabetes, vascular disease, stroke or intracranial hemorrhage. General exclusion criteria for MR imaging included claustrophobia, ferromagnetic implants, cochlear implants and cardiac pacemakers.

2.3. MRI examination protocol

All subjects underwent MR imaging on a 3-T MR scanner (Magnetom Verio, Siemens Healthcare, Erlangen, Germany), using a 12-element phased-array head coil. 3D magnetization-prepared rapid-acquisition gradient echo imaging (MP-RAGE) was obtained for structural information using the following parameters: repetition time (TR) = 11 ms; echo time (TE) = 4.76 ms; field of view (FOV) = 230 mm; voxel size = 0.9 mm × 0.9 mm × 0.9 mm. 160 sagittal slices parallel to the falx cerebri were acquired, covering the entire brain. The data were acquired with a parallel acquisition technique (iPAT, acceleration factor 2). A dynamic contrast-enhanced angiography sequence (TWIST) was obtained using the following acquisition parameters: field of view 280 mm, slice thickness 2.0 mm, TR 2.33 ms, TE 0.94 ms, flip angle 21°, base resolution 256 × 256, phase resolution 80%.

In order to obtain blood flow to and from the brain, two retrospectively gated, velocity-encoded cine-phase contrast scans were performed (FOV = 140 mm, matrix = 256 × 179, voxel size = 0.8 mm × 0.5 mm × 6 mm, TR = 40 ms, TE 4.05 ms, FA = 20°, acquisition time = 32 cardiac cycles equaling approximately 3 min as determined by the individual heart rate). First, a high-velocity encoding (70 cm/s) was used to quantify the high-velocity blood flow in the internal carotid arteries (ICAs), vertebral arteries (VAs), and internal jugular veins (IJVs). The sequence was positioned at the upper level of the 2nd cervical vertebra with an orientation perpendicular to the main four arteries (ICAs and VAs) and the IJVs. To assess the low-velocity secondary venous flow, a sequence with low-velocity encoding (7–9 cm/s) was applied at the same level. To visualize primary and secondary venous channels, a 2D time-of-flight MR venography of the infratentorial and upper cervical regions was performed (slice thickness 2.0 mm, FOV 160 mm, TR 23 ms, TE 5.43 ms and FA 40°).

2.4. Post-processing and data analysis

Before performing quantitative analysis, all MRI datasets were reviewed to confirm diagnostic image quality. 32 images of the pulsatile flow per cardiac cycle were derived from the applied cine-phase contrast sequence. Time-dependent volumetric flow rates were calculated by integrating the flow velocities inside the luminal cross-sectional areas over all 32 images, using the pulsatility-based segmentation of lumens conducting non-steady flow algorithm. Mean flow rates were obtained for each of the four main cervical arteries (left and right ICAs and left and right VAs), for the primary venous pathways (left and right IJVs), and the secondary venous pathways including vertebral veins (VVs), epidural veins (EVs), and deep cervical veins (DCVs).

Flow waveforms were obtained for each of the four main cervical arteries: left and right internal carotid artery (LICA, RICA) and left and right vertebral artery (LVA, RVA), for the primary venous pathways, the left and right internal jugular vein (LIJV, RIJV), and for the secondary venous pathways, the vertebral veins (VV), epidural veins (EV), and deep cervical veins (DCV). In addition, cervical CSF stroke volume, i.e., the volume of CSF that flows back and forth between the cranium and the spinal canal, and aqueductal CSF stroke volume were obtained by time integration of the CSF flow waveforms. Total arterial cerebral blood flow (tCBF) was obtained by summation of the flows through the four arteries supplying the brain (left and right ICA, left and right VA). Flow in the basilar artery was calculated as the sum of the flow in both VAs. Secondary venous flow was defined as the sum of the flow through the three main secondary venous channels (VVs, EVs, and DCVs). Total venous outflow was determined as the total flow in all detected veins (IJVs and secondary veins).

Details of the derivation of the intracranial compliance and pressure have been described previously [14,15]. Briefly, based on the physical definition of compliance as a ratio of volume and pressure changes, intracranial compliance is estimated from the ratio of the maximal (systolic) intracranial volume and pressure fluctuations during the cardiac cycle. The change in intracranial volume (ICVC) is obtained from the momentary differences between volumes of blood and CSF entering and leaving the cranium as shown in Eqs. (1) and (2),

$$\Delta\mathrm{ICVC}(i) = [f_A(i) - f_V(i) - f_{\mathrm{CSF}}(i)] \cdot \Delta t \quad (1)$$

$$\sum_{\text{cardiac cycle}} \Delta\mathrm{ICVC}(i) = \sum_{\text{cardiac cycle}} [f_A(i) - f_V(i) - f_{\mathrm{CSF}}(i)] \cdot \Delta t = 0 \quad (2)$$

where f_A is arterial inflow, f_V is venous outflow, and f_{CSF} is the craniospinal CSF flow. Eq. (2) states that in steady state, the intracranial volume is on average constant over an entire cardiac cycle.

The pressure change is derived from the amplitude of the pressure gradient (PG) waveform obtained using the Navier–Stokes relationships between derivatives of CSF velocities and the CSF