Neuroradiology (2012) 54:673–680
DOI 10.1007/s00234-011-0953-0

DIAGNOSTIC NEURORADIOLOGY

The value of magnetic resonance imaging for the detection of the bleeding source in non-traumatic intracerebral haemorrhages: a comparison with conventional digital subtraction angiography

Nina Lummel · Jürgen Lutz · Hartmut Brückmann · Jennifer Linn

Received: 17 May 2011 / Accepted: 29 August 2011 / Published online: 15 September 2011

Abstract
Introduction Conventional digital subtraction angiography (DSA) is currently regarded as the gold standard in detecting underlying vascular pathologies in patients with intracerebral haemorrhages (ICH). However, the use of magnetic resonance imaging (MRI) in the diagnostic workup of ICHs has considerably increased in recent years. Our aim was to evaluate the diagnostic accuracy and yield of MRI for the detection of the underlying aetiology in ICH patients.
Methods Sixty-seven consecutive patients with an acute ICH who underwent MRI (including magnetic resonance angiography (MRA) and DSA during their diagnostic workup) were included in the study. Magnetic resonance images were retrospectively analysed by two independent neuroradiologists to determine the localisation and cause of the ICH. DSA was used as a reference standard.
Results In seven patients (10.4%), a DSA-positive vascular aetiology was present (one aneurysm, four arteriovenous malformations, one dural arteriovenous fistula and one vasculitis). All of these cases were correctly diagnosed by both readers on MRI. In addition, MRI revealed the following probable bleeding causes in 39 of the 60 DSA-negative patients: cerebral amyloid angiopathy (17), cavernoma (9), arterial hypertension (8), haemorrhagic transformation of an ischaemic infarction (3) and malignant brain tumour with secondary ICH (2).
Conclusion Performing MRI with MRA proved to be an accurate diagnostic tool in detecting vascular malformations in patients with ICH. In addition, MRI provided valuable information regarding DSA-negative ICH causes, and thus had a high diagnostic yield in ICH patients.

Keywords Intracerebral haemorrhage · ICH · MR angiography · MRI · DSA

Abbreviations

AVM	Arteriovenous malformations
dAVF	Dural arterio-venous fistula
DSA	Digital subtraction angiography
DWI	Diffusion weighted imaging
CAA	Cerebral amyloid angiopathy
CI	Confidence interval
CT	Computed tomography
CTA	Computed tomography angiography
FLAIR	Fluid-attenuated inversion recovery
ICH	Intracerebral haemorrhages
IVH	Intraventricular haemorrhage
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
SAH	Subarachnoid haemorrhages
SWI	Susceptibility weighted imaging
T	Tesla
TOF	Time-of-flight

Electronic supplementary material The online version of this article (doi:10.1007/s00234-011-0953-0) contains supplementary material, which is available to authorized users.

N. Lummel (✉) · J. Lutz · H. Brückmann · J. Linn
Department of Neuroradiology, University of Munich,
Marchioninistrasse 15,
81377 Munich, Germany
e-mail: nina.lummel@med.uni-muenchen.de

Introduction

Non-traumatic intracerebral haemorrhages (ICH) account for 10–20% of acute stroke cases [1]. Primary causes of ICHs are long-standing hypertension and cerebral amyloid angiopathy (CAA) [1, 2]. Secondary ICHs can be caused by a wide variety of aetiologies, including arteriovenous malformations (AVMs), ruptured intracranial aneurysms, cavernomas, haemorrhagic tumours, coagulopathies, sinus and venous thromboses, haemorrhagic transformation of an ischaemic infarction and vasculitis [3]. Timely and accurate identification of patients with vascular lesions as the underlying ICH cause is important because lesions such as AVMs and aneurysms require prompt treatment to prevent re-bleeding [4].

Within the last few years, the value of magnetic resonance imaging (MRI) as the initial imaging modality in both ischaemic and haemorrhagic stroke patients has considerably increased. It has been shown that MRI is just as sensitive as computed tomography (CT) in the detection of acute ICHs [5–7]. However, digital subtraction angiography (DSA) is still regarded as the gold standard for identifying vascular malformations as sources of bleeding in ICHs [8]. However, DSA is an invasive procedure with small but residual risks, including bleeding, allergy, nephrotoxicity and thromboembolism, all of which add up to a risk of 0.1–1% for permanent neurologic deficits [9–11].

Thus, our aim was to evaluate the diagnostic accuracy and yield of MRI for the detection of vascular malformations and other bleeding sources in ICH patients compared to DSA.

Methods

This study was approved by our Institutional Review Board.

Patient selection

At our institution, all patients presenting with ICH are initially investigated using CT including computed tomography angiography (CTA). If CTA reveals a vascular malformation that requires immediate treatment (e.g. cerebral aneurysm) but also in cases of CTA-negative subarachnoid haemorrhage (SAH), DSA is performed promptly after CTA. All other patients with spontaneous ICH undergo MRI with magnetic resonance angiography (MRA) during their further diagnostic workup.

We used our electronic in-hospital database of clinical records to search for the key words ‘intracerebral haemorrhage’, ‘ICH’ and ‘DSA’ to identify all consecutive patients with an ICH who were admitted to our institution between 2003 and 2009 and had a DSA performed.

Patients had to meet the following additional inclusion criteria:

1. MRI with MRA performed within the first 20 days after symptom onset and before surgical haematoma evacuation or surgical or neurointerventional treatment of the bleeding source
2. An image quality of both DSA and MRI that was sufficient for evaluation
3. Cases with traumatic ICHs were excluded.

Image acquisition

MRI was either performed on a 1.5-Tesla (T) Scanner (Magnetom Symphony, Siemens Medical Solutions, Erlangen, Germany) or on a 3-T Scanner (Signa HDxt, GE Healthcare, Milwaukee, WI, USA). Due to the retrospective character of this study, the MRIs were not performed according to a standardised protocol. Available sequences and parameters are indicated in Table 1.

DSA was performed on a biplanar DSA unit with an image matrix of 1,024×1,024 (Neurostar; Siemens Medical Solutions) using a saline-flushed guide catheter under systemic heparinisation. Diagnostic series included selective catheterisations of both internal and external carotid arteries and the dominant vertebral artery, as well as late venous phases.

Image analysis

First, the MRI data sets were reviewed independently by two neuroradiologists who were blinded to all patient information, clinical data and other imaging modalities (including CT/CTA and DSA). Readers assessed the following details: (1) ICH localisation (left or right side; basal ganglia, thalamus, frontal, temporal, parietal, or occipital lobe or infratentorial), (2) presence of associated intraventricular or SAH, (3) evidence of an underlying

Table 1 MRI sequences available in ICH patients

Sequences available	MRA 1.5/3T	T1 1.5/3T	T1ce 1.5/3T	T2 1.5/3T	PD 1.5/3T	FLAIR 1.5/3T	T2* 1.5/3T	DWI 1.5/3T
Number of patients	53/14	43/11	34/6	49/11	44/6	24/11	36/13	39/13

MRI magnetic resonance imaging, *MRA* time-of-flight magnetic resonance angiography, *T1* non-enhanced T1-weighted images, *T1ce* contrast-enhanced T1-weighted images, *T2* T2-weighted images, *PD* proton-density-weighted images, *FLAIR* fluid-attenuated inversion recovery-weighted imaging, *T2** T2*-weighted gradient echo images, *DWI* diffusion-weighted images, *T* Tesla

vascular malformation (aneurysm, AVM or dural arteriovenous fistula (dAVF)), (4) signs of other angiographically positive ICH aetiologies (e.g. vasculitis or sinus or venous thrombosis) and (5) signs indicative of an angiographically negative ICH aetiology (e.g. CAA or cavernomas). If an underlying vascular pathology was suspected, readers also noted the exact localisation and the affected vessels. In cases of a disagreement concerning a non-vascular cause, MRI and MRA were re-evaluated by both readers to reach a consensus on a final MRI diagnosis. If no consensus could be reached for a given case, it was classified as 'unknown cause'.

After the evaluation of all MRI data sets, the DSA series was analysed together with all patient information and clinical data by the same two neuroradiologists to determine the reference standard concerning the presence of a DSA-positive vascular aetiology.

Statistical analysis

For DSA-positive pathologies, sensitivity, specificity, positive predictive value and negative predictive value of MRI were calculated. Furthermore, an inter-rater reliability analysis by Cohen's kappa statistics was performed to determine consistency among raters for the detection of an underlying vascular aetiology of the ICH. Statistical analyses were performed using the Statistical Package for the Social Sciences version 17.0 (SPSS, Chicago, IL, USA).

Results

A total of 120 consecutive patients with spontaneous SAH underwent DSA during the study period. Sixty-seven of these patients met the inclusion and exclusion criteria (mean age, 53.4 years; range, 14–82 years; 44 males; 23 females).

In 36 patients, a lobar ICH in one or more lobes was found (12 frontal, 15 temporal, 13 parietal and 7 occipital). The ICH was located in the basal ganglia in 13 patients, and infratentorial localisation was present in 18 patients. Associated haemorrhages included intraventricular haemorrhages (IVHs) in eight cases and SAHs in five patients.

DSA as the reference standard for angiographically positive pathologies revealed an AVM in four cases (6%; Fig. 1), a dAVF in one patient (1.5%; Fig. 2), a dissecting

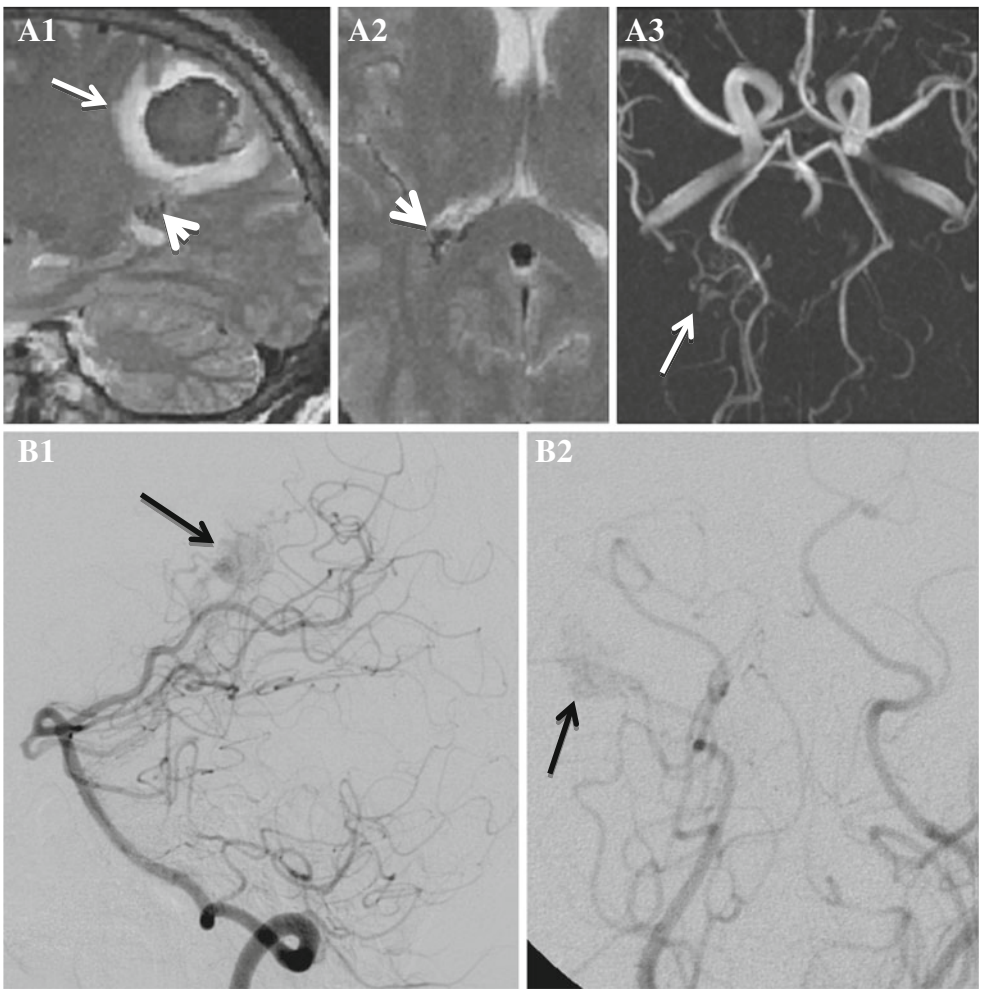

Fig. 1 MRI performed at 1.5 T (*A*) and DSA (*B*) of a 36-year-old male with a parietal ICH due to an AVM originated from the right posterior cerebral artery. The sagittal T2-weighted image (*A1*) shows the lobar haemorrhage with perifocal oedema (*normal white arrow*). Adjacent to the right lateral ventricle, prominent vessels can be detected (*A1*, *A2*; *short white arrows*) on T2-weighted imaging (*A1*, *A2*) as well as on the TOF-MRA (*A3*; *normal white arrow*). DSA with selective catheterization of the left vertebral artery in lateral (*B1*) and anterioposterior view (*B2*) confirms the AVM (*black arrows*)

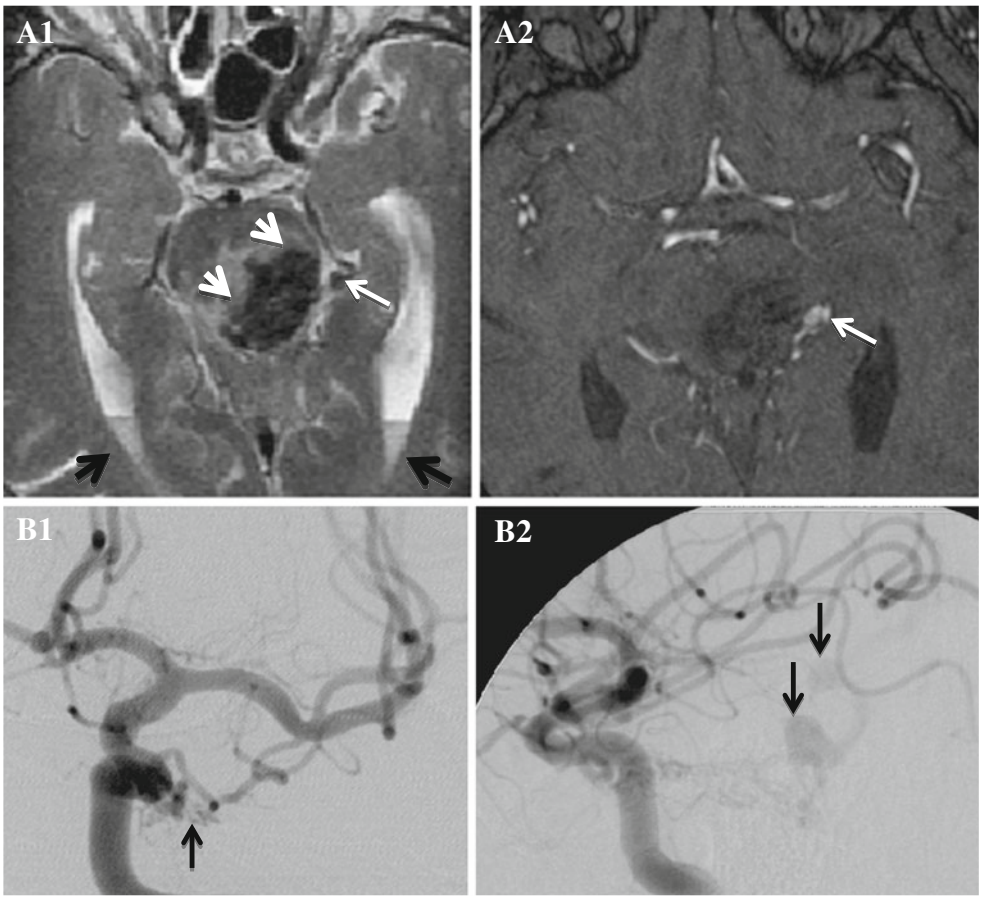

Fig. 2 MRI performed at 1.5 T (*A*) and DSA (*B*) of a 72-year-old male with an infratentorial ICH due to a dural arteriovenous fistula (dAVF). T2-weighted imaging (*A1*) shows the pontine ICH (*white arrowheads*) and the associated IVH (*fat black arrows*). Furthermore, a venous dilatation associated with the dAVF can be depicted on T2w (*A1*) and TOF-MRA (*A2*) (*thin white arrow*). DSA with selective catheterization of the left internal carotid artery in anterioposterior (*B1*) and lateral (*B2*) view confirms the arteriovenous shunt with the venous dilatations (*thin black arrows*)

aneurysm of the vertebral and basilar artery with intraparenchymal and intraventricular ICH in one patient (1.5%) and one case of vasculitis (1.5%). A sinus or deep vein thrombosis was not detected in our population (for details, see Table 2). In the remaining 60 patients, no vessel pathology could be identified on DSA.

All seven DSA-positive pathologies were correctly identified by both readers on MRI. There was one false-positive diagnosis of an AVM. Thus, the sensitivity of MRI for the detection of DSA-positive pathologies was 100% (95% confidence interval (CI)=56.1–100%) with a specificity of 98.3% (95% CI=89.9–99.9%). The positive predictive value was 87.5% (95% CI=46.7–99.3%), and the negative predictive value was 100% (95% CI=92.4–100%; Table 3).

The inter-rater agreement for the detection of a DSA-positive vascular aetiology of the ICH was excellent (kappa (κ)=0.93).

In the independent evaluation of the MRI by the two blinded readers, a disagreement concerning a non-vascular ICH cause was present in eight cases, but a final MRI diagnosis could be achieved in all of these cases during the consensus reading. A non-vascular ICH cause was found in 39 patients (58.2%): CAA was suspected in 17 patients (25.4%; Fig. 3), a cavernoma was present in nine cases (13.4%), arterial hypertension was suspected in eight cases (11.9%), a haemorrhagic transformation of an ischaemic stroke was found in three cases (4.5%) and two patients had an intratumoural haemorrhage (3%).

The ICH cause was considered unknown in 21 cases (31.3%) of patients despite extensive diagnostic workup, which included additional CT angiography and extensive blood examination.

The cases for cavernomas and tumours were histologically proven. The CAA cases were not histologically proven, but diagnosis was based on the Boston criteria for probable CAA-related ICH [12]. The diagnoses of arterial hypertension, respectively, haemorrhagic transformation of an ischaemic stroke as causes of the ICH were approved by the clinical history and patient follow-up.

Discussion

MRI with MRA proved to be an accurate diagnostic tool in detecting causative DSA-positive pathologies in patients with ICH; moreover, it revealed DSA-negative causes in a high percentage of patients.

Spontaneous ICHs account for 10–15% of all strokes. Increased age and arterial hypertension represent the most

Table 2 Patient characteristics and findings in ICH patients with DSA-positive pathologies

n	Sex	Age	Bleeding	Localization	Cause	Vessel(s) affected/feeding	DSA post onset	MRI post onset	MRI sequences available							
									MRA	T2	PD	FLAIR	T1	T1ce	T2*	DWI
1	f	40	ICH	l Basal ganglia	Vasculitis	All ic vessels	12	16	+	+	+	+	+	+	+	+
2	m	71	ICH	r Pons	AVM	r PCA	2	3	+	+	+	+	–	–	–	–
3	m	65	ICH	Cerebellar vermis	AVM	r PCA	1	1	+	+	+	–	+	+	+	+
4	m	59	ICH + IVH	r Pons	Aneurysm	BA	0	15	+	+	+	+	+	+	+	–
5	m	36	ICH	r Parietal	AVM	r PCA	0	2	+	+	+	–	–	–	+	+
6	m	64	ICH + IVH	l Frontoparietal	AVM	l MCA	1	1	+	+	+	+	+	–	+	+
7	m	72	ICH + SAH	Mesencephalon, pons	dAVF	l ICA	2	2	+	+	+	+	–	–	+	+

All MRI investigations were performed on a 1.5-T scanner

n number of patient, *DSA* digital subtraction angiography, *f* female, *m* male, *MRI* magnetic resonance imaging, *MRA* time-of-flight magnetic resonance angiography, *T1* non-enhanced T1-weighted images, *T1ce* contrast-enhanced T1-weighted images, *T2* T2-weighted images, *PD* proton-density-weighted images, *FLAIR* fluid-attenuated inversion recovery-weighted imaging, *T2** T2*-weighted gradient echo images, *DWI* diffusion-weighted images, *ICH* intracerebral haemorrhage, *IVH* intraventricular haemorrhage, *SAH* subarachnoid haemorrhage, *AVM* arteriovenous malformation, *dAVF* dural arteriovenous fistula, *ic* intracranial, *r* right, *l* left, *PCA* posterior cerebral artery, *BA* basilar artery, *MCA* middle cerebral artery, *ICA* internal cerebral artery, + sequence was available, – sequence was not available

Table 3 Diagnostic accuracy of MRI in the detection of DSA-positive pathologies in ICHs (n=67) evaluated by two readers (n=134)

	DSA confirmed related vascular lesion Reader 1/Reader 2	DSA confirmed no related vascular lesion Reader 1/Reader 2	Total
Positive MRI res. MRA for related vascular lesion	7/7	0/1	7/8
Negative MRI res. MRA for related vascular lesion	0/0	60/59	60/59
Total	7/7	60/60	67

DSA digital subtraction angiography, *MRI* magnetic resonance imaging, *MRA* magnetic resonance angiography, *res.* respectively

important risk factors for ICH. Further independent risk factors include alcohol abuse, male sex and anticoagulant treatment [13–15]. In the elderly, arterial hypertension constitutes by far the most important modifiable risk factor for non-traumatic ICHs [13, 15, 16]. In patients between 40 and 70 years of age, arterial hypertension accounts for the underlying cause of an ICH in over 50% of cases [17, 18]. In young adults, intracranial vascular malformations, including AVMs, dAVFs and cavernous malformations, represent the most common causes of ICH [19].

DSA-positive pathologies

AVMs are defined as vascular malformations with arteriovenous shunting in the absence of an intervening capillary bed. Angiographically, AVMs show a so-called ‘nidus’, a conglomeration of numerous small arteriovenous shunts, typically including enlarged feeding arteries and draining veins, which are also significantly enlarged and can show associated varixes, stenoses and aneurysms. Dural arteriovenous fistulas are defined as arteriovenous shunts that are located within the dura mater and most commonly involve the transverse sinus, followed by the cavernous sinus. Typically, a thrombotic occlusion of the involved dural venous sinus is found [20].

In recent years, several studies have demonstrated that, compared with DSA and intraoperative findings, multidetector CTA is highly accurate for the detection of vascular malformations in patients with ICH, with reported sensitivities ranging from 89% to 96% and specificities from 92% to 100% [21–24]. Furthermore, MRI with MRA emerged as an effective diagnostic modality to identify vascular causes of ICHs [25–27]. Thus, despite the considerable recent advances in CTA and MRA techniques, DSA is still commonly considered the gold standard in ruling out an underlying vascular malformation in ICH patients [8].

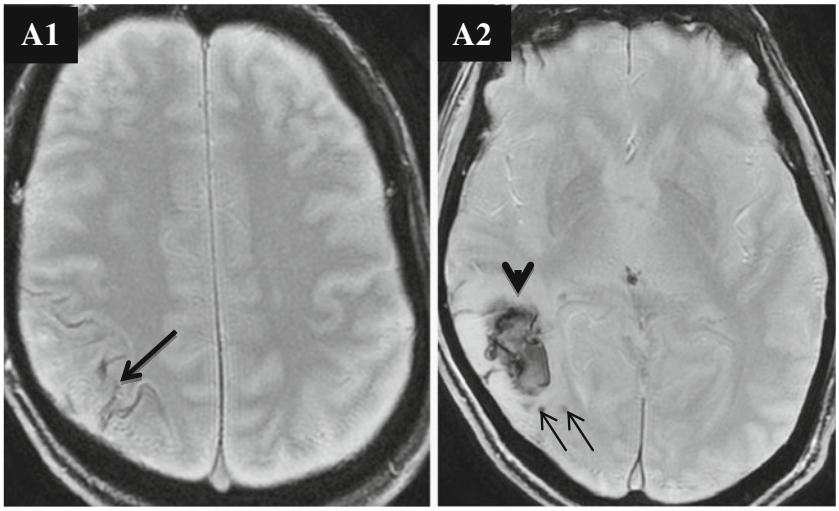

Fig. 3 T2*-weighted images of a 63-year-old male with a parietotemporal ICB (*arrowhead*). Because of the presence of additional microbleeds (*thin arrows*) and superficial siderosis (*fat arrow*) as well as a negative DSA, CAA was suspected as the probable underlying cause of the ICH

With regard to AVMs, a conventional time-of-flight (TOF)-MRA allows the gross depiction of AVMs but does not provide sufficient information on their angioarchitecture [28]. Regarding dAVFs, TOF-MRA can be negative, especially in small or slow flow shunts [29]. However, new high-resolution, contrast-enhanced and time-resolved MRA techniques have strongly improved the value of MRA in the evaluation of AVMs [25–27] and the detection of dAVFs [30]. Furthermore, conventional MRI sequences (T1, T2 and fluid-attenuated inversion recovery (FLAIR)) can be very helpful in the detection of AVMs or dAVFs. The AVM's vessels present as prominent hypointense flow voids resembling a 'bag of black worms' with little or no mass effect on T2-weighted images, and the FLAIR sequence might show a high signal in some of the adjacent brain parenchyma, indicating gliotic tissue. In dAVFs, T2, T1 and FLAIR sequences demonstrate the thrombosed dural sinus and eventually an adjacent oedema if a venous congestion is present. In rare cases, a diffuse dural enhancement is observed on contrast-enhanced T1-weighted images. Previous studies on MRA in AVMs and dAVFs did not evaluate the diagnostic yield of MRI in the detection of a bleeding cause in ICHs but focused on the appearance of already diagnosed AVM or dAVF using MRI [25–37].

In our patient population, vascular malformations were identified in 10.5% of patients, with a sensitivity of 100% and a specificity of 98.3% compared to DSA. In one case, the second reader made one false-positive diagnosis of a cerebral arteriovenous malformation based on MRI images, which turned out not to be a vascular lesion on DSA. There was no instance in which the DSA could detect a causative vascular pathology that was not seen on MRI.

Compared to reports of previous CT and DSA studies, which found vascular pathologies as the cause of ICH in 27.5% to 52.5%, the number of vascular causes in our population was quite low (10.4%) [31–33]. This discrepancy is due to the following reasons, which result in a biased patient selection in this retrospective study. In our institution, CT is still the emergency imaging modality of choice in patients with acute onset of neurological deficits and, thus, in the detection of ICHs. For the evaluation of a vascular pathology as the cause of an ICH, a CTA or DSA is initially performed. Causative vascular lesions are often immediately treated; only in unclear cases or in cases in which immediate treatment is not necessary is MRI performed. That is why not all patients who presented in our emergency department with an ICH in the given time period could be included in this study.

DSA-negative pathologies

The most important DSA-negative bleeding causes include arterial hypertension and CAA, which account for approximately 80% of cases [1, 2, 13]. Other important DSA-negative bleeding causes are cavernous malformations, haemorrhagic ischaemic stroke and intracranial neoplasms. In the majority of cases, these pathologies present with the following typical MR imaging findings: Hypertensive ICHs are typically located within the basal ganglia and/or thalami. This location is so pathognomonic that angiographical techniques (MRA, CTA and DSA) are rarely necessary in establishing this diagnosis in older patients with long-standing arterial hypertension [34]. CAA represents the second most important non-vascular cause of ICH in elderly patients. In contrast to hypertensive ICHs, ICHs due to CAA are located more superficially in the cerebral lobes in the majority of cases. However, the diagnosis of CAA as the underlying aetiology of an ICH cannot be made based on localisation alone. The definite diagnosis of CAA still requires histopathologic demonstration of vascular amyloid. However, the Boston diagnostic criteria can be used to determine the probability of CAA as the bleeding source [12,

35]. The sensitivity of these criteria can be enhanced to about 95% by including microbleeds and superficial siderosis as typical imaging findings of CAA [34, 36].

Cavernous malformations are DSA-negative vascular malformations. They have a very characteristic MRI appearance with a central core of mixed signal intensity and a peripheral hypointense rim on T2-weighted images and are detected best on gradient-echo T2*-weighted sequences [37–39].

Intratumoural haemorrhage must be suspected in cases of pronounced perilesional oedema adjacent to an ICH immediately after the onset of the clinical symptoms and in cases of significant contrast enhancement of parts of the lesion on contrast-enhanced T1-weighted imaging early in the clinical course.

In our study population, we found the above-mentioned typical MRI signs of these different DSA-negative ICH causes in 58.2% of cases. Our results are in accordance with previous reports that the large majority of spontaneous ICHs are 'primary', i.e. caused either by longstanding hypertension, CAA or both [1, 2, 40]. The high prevalence of CAA (25.4%) compared to arterial hypertension (11.9%) arises from the fact that, in 'typical' cases of an acute basal ganglia or thalamic haemorrhage in an elderly, hypertensive patient, DSA is usually not accomplished because it is not necessary to establish the diagnosis. Thus, the majority of these cases did not meet the inclusion criteria of our study.

Due to the characteristic MRI findings of DSA-negative lesions and based on our and others' results, we recommend that in addition to the obligatory MRA, an MRI protocol should include at least the following sequences to identify these ICH causes: a T2-weighted sequence (for cavernomas), a FLAIR sequence (for accompanying SAH or IVH), a T2*-weighted gradient echo sequence (for CAA and cavernomas), diffusion weighted imaging (DWI; for haemorrhagic transformation of ischaemic stroke), and T1-weighted sequences before and after application of contrast agent (intratumoural haemorrhage) [41, 42].

Overall, the results of our study suggest that the routine use of MRI is beneficial in ICH patients because the unique parenchymal information provided by this technique reveals ICH causes that cannot be identified on vessel-only imaging studies such as DSA or CTA in a considerably high percentage.

In 31.3% of the patients investigated in this study, the ICH cause was considered unknown despite extensive diagnostic workup. This percentage is slightly higher than in former studies (11–16%), most likely because of the biased patient selection in this study [24, 40].

Limitations

Because of the retrospective study design, only routine data sets without a standardised MRI protocol were available in the patients, and most data sets were performed on a 1.5-T scanner. The use of an optimised sequence protocol with more advanced MRI sequences and higher field strength (e.g. contrast-enhanced and time-resolved MRA and susceptibility weighted imaging at 3-T) would most likely enhance the usefulness of MRI in ICH patients. Furthermore, prospective studies with a standard MRI protocol should be performed to further address this issue. Another limitation is the wide time frame of the MRI examination. However, whether MRI can be performed routinely in the majority of acute ICH patients remains uncertain.

Furthermore, in this investigation, MRIs were evaluated concerning the presence or absence of an underlying vascular aetiology in ICH patients. However, other important features related to AVMs/AVFs, such as the number and origin of arterial feeders, presence of intranidal and/or feeding artery aneurysms, and the presence of draining vein stenoses and deep versus superficial venous drainage, were not measured.

Conclusions

MRI with MRA proved to be an accurate diagnostic tool in detecting causative vascular lesions in patients with ICHs, and it has the potential to replace DSA, the current gold standard. In addition, MRI has the ability to provide valuable additional information with regard to DSA-negative ICH causes; thus, MRI has a high diagnostic yield in the detection of the bleeding source in patients with ICHs and should be more routinely obtained in ICH patients in the future.

Conflict of interest We declare that we have no conflict of interest.

References

1. Qureshi AI, Tuhrim S, Broderick JP et al (2001) Spontaneous intracerebral hemorrhage. NEJM N Engl J Med 344:1450–1460
2. Foulkes MA, Wolf PA, Price TR et al (1988) The Stroke Data Bank: design, methods, and baseline characteristics. Stroke 19:547–554
3. Linn J, Herms J, Dichgans M et al (2008) Subarachnoid hemosiderosis and superficial cortical hemosiderosis in cerebral amyloid angiopathy. AJNR Am J Neuroradiol 29:184–186
4. (1999) Arteriovenous malformations of the brain in adults: the Arteriovenous Malformation Study Group. N Engl J Med 340:1812–1818
5. Kidwell CS, Chalela JA, Saver JL et al (2004) Comparison of MRI and CT for detection of acute intracerebral hemorrhage. JAMA 292:1823–1830
6. Fiebach JB, Schellinger PD, Gass A et al (2004) Stroke magnetic resonance imaging is accurate in hyperacute intracerebral hemorrhage: a multicenter study on the validity of stroke imaging. Stroke 35:502–506
7. Kidwell CS, Wintermark M (2008) Imaging of intracranial haemorrhage. Lancet Neurol 7:256–267

8. Broderick JP, Adams HP Jr, Barsan W et al (1999) Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 30:905–915
9. Heiserman JE, Dean BL, Hodak JA et al (1994) Neurologic complications of cerebral angiography. AJNR Am J Neuroradiol 15:1401–1407
10. Willinsky RA, Taylor SM, TerBrugge K et al (2003) Neurologic complications of cerebral angiography: prospective analysis of 2899 procedures and review of the literature. Radiology 227:522–528
11. Dawkins AA, Evans AL, Wattam J et al (2007) Complications of cerebral angiography: a prospective analysis of 2,924 consecutive procedures. Neuroradiology 49:753–759
12. Knudsen KA, Rosand J, Karluk D et al (2001) Clinical diagnosis of cerebral amyloid angiopathy: validation of the Boston criteria. Neurology 56:537–539
13. Fewel ME, Thompson BG Jr, Hoff JT (2003) Spontaneous intracerebral hemorrhage: a review. Neurosurg Focus 15:1–16
14. Juvela S, Hillbom M, Palomaki H (1995) Risk factors for spontaneous intracerebral hemorrhage. Stroke 26:1558–1564
15. Thrift AG, McNeil JJ, Forbes A et al (1996) Risk factors for cerebral hemorrhage in the era of well-controlled hypertension. Melbourne Risk Factor Study (MERFS) Group. Stroke 27:2020–2025
16. Brott T, Thalinger K, Hertzberg V (1986) Hypertension as a risk factor for spontaneous intracerebral hemorrhage. Stroke 17:1078–1083
17. Gomori JM, Grossman RI, Hackney DB et al (1988) Variable appearances of subacute intracranial hematomas on high-field spin-echo MR. AJR Am J Roentgenol 150:171–178
18. Viswanathan A, Chabriat H (2006) Cerebral microhemorrhage. Stroke 37:550–555
19. Al-Shahi R, Bhattacharya JJ, Currie DG et al (2003) Scottish Intracranial Vascular Malformation Study (SIVMS): evaluation of methods, ICD-10 coding, and potential sources of bias in a prospective, population-based cohort. Stroke 34:1156–1162
20. Cognard C, Gobin YP, Pierot L et al (1995) Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. Radiology 194:671–680
21. Yeung R, Ahmad T, Aviv RI et al (2009) Comparison of CTA to DSA in determining the etiology of spontaneous ICH. Can J Neurol Sci 36:176–180
22. Romero JM, Artunduaga M, Forero N et al (2009) Accuracy of CT angiography for the diagnosis of vascular abnormalities causing intraparenchymal hemorrhage in young patients. Emerg Radiol 16:195–201
23. Yoon DY, Chang SK, Choi CS et al (2009) Multidetector row CT angiography in spontaneous lobar intracerebral hemorrhage: a prospective comparison with conventional angiography. AJNR Am J Neuroradiol 30:962–967
24. Delgado Almandoz JE, Schaefer PW, Forero NP et al (2009) Diagnostic accuracy and yield of multidetector CT angiography in the evaluation of spontaneous intraparenchymal cerebral hemorrhage. AJNR Am J Neuroradiol 30:1213–1221
25. Hadizadeh DR, von Falkenhausen M, Gieseke J et al (2008) Cerebral arteriovenous malformation: Spetzler-Martin classification at subsecond-temporal-resolution four-dimensional MR angiography compared with that at DSA. Radiology 246:205–213
26. Unlu E, Temizoz O, Albayram S et al (2006) Contrast-enhanced MR 3D angiography in the assessment of brain AVMs. Eur J Radiol 60:367–378
27. Taschner CA, Gieseke J, Le Thuc V et al (2008) Intracranial arteriovenous malformation: time-resolved contrast-enhanced MR angiography with combination of parallel imaging, keyhole acquisition, and k-space sampling techniques at 1.5 T. Radiology 246:871–879
28. Ozsarlak O, Van Goethem JW, Maes M et al (2004) MR angiography of the intracranial vessels: technical aspects and clinical applications. Neuroradiology 46:955–972
29. Chen JC, Tsuruda JS, Halbach VV (1992) Suspected dural arteriovenous fistula: results with screening MR angiography in seven patients. Radiology 183:265–271
30. Meckel S, Maier M, Ruiz DS et al (2007) MR angiography of dural arteriovenous fistulas: diagnosis and follow-up after treatment using a time-resolved 3D contrast-enhanced technique. AJNR Am J Neuroradiol 28:877–884
31. Griffiths PD, Beveridge CJ, Gholkar A (1997) Angiography in non-traumatic brain haematoma. An analysis of 100 cases. Acta Radiol 38:797–802
32. Toffol GJ, Biller J, Adams HP et al (1986) The predicted value of arteriography in nontraumatic intracerebral hemorrhage. Stroke 17:881–883
33. Laissy JP, Normand G, Monroc M et al (1991) Spontaneous intracerebral hematomas from vascular causes. Predictive value of CT compared with angiography. Neuroradiology 33:291–295
34. Linn J, Halpin A, Demaerel P et al (2009) Prevalence of superficial siderosis in patients with cerebral amyloid angiopathy. Neurology 74:1346–1350
35. Greenberg SM, Briggs ME, Hyman BT et al (1996) Apolipoprotein E epsilon 4 is associated with the presence and earlier onset of hemorrhage in cerebral amyloid angiopathy. Stroke 27:1333–1337
36. van Rooden S, van der Grond J, van den Boom R et al (2009) Descriptive analysis of the Boston criteria applied to a Dutch-type cerebral amyloid angiopathy population. Stroke 40:3022–3027
37. Cooper AD, Campeau NG, Meissner I (2008) Susceptibility-weighted imaging in familial cerebral cavernous malformations. Neurology 71:382
38. de Souza JM, Domingues RC, Cruz LC et al (2008) Susceptibility-weighted imaging for the evaluation of patients with familial cerebral cavernous malformations: a comparison with t2-weighted fast spin-echo and gradient-echo sequences. AJNR Am J Neuroradiol 29:154–158
39. Lehnhardt FG, von Smekal U, Ruckriem B et al (2005) Value of gradient-echo magnetic resonance imaging in the diagnosis of familial cerebral cavernous malformation. Arch Neurol 62:653–658
40. Wijman CA, Venkatasubramanian C, Bruins S et al (2010) Utility of early MRI in the diagnosis and management of acute spontaneous intracerebral hemorrhage. Cerebrovasc Dis 30:456–463
41. Young N, Vladica P, Soo YS et al (1993) Acute intracerebral haematomas: assessment for possible underlying cause with MRI scanning. Australas Radiol 37:315–320
42. Dylewski DA, Demchuk AM, Morgenstern LB (2000) Utility of magnetic resonance imaging in acute intracerebral hemorrhage. J Neuroimaging 10:78–83