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Research article

# Wall enhancement ratio determined by vessel wall MRI associated with symptomatic intracranial aneurysms

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ABSTRACT

*Purpose:* To study the association of the enhancement ratio (ER) of aneurysmal wall enhancement (AWE) with symptomatic intracranial aneurysms (IAs), we hypothesized that the ER of AWE would be stronger in symptomatic IAs than in asymptomatic IAs, as assessed by high-resolution magnetic resonance imaging (HRMRI).
*Materials and methods:* Between February 2016 and February 2018, 80 consecutive patients with 89 unruptured IAs were reviewed. Patients and IAs were divided into symptomatic and asymptomatic groups. In addition to the clinical characteristics, the IA features (e.g., size, shape) were evaluated via computed tomography angiography, while the ER and enhanced patterns were evaluated by HRMRI. Multiple logistic regression analysis was performed to determine the independent risk factors for symptomatic IAs. Receiver operating characteristic curve analysis was used for the final model to obtain the optimal thresholds.
*Results:* Multiple logistic regression analysis indicated that only the ER was associated with symptomatic IAs. The threshold value of the ER was 60.5%.
*Conclusions:* A higher ER was more frequently identified in symptomatic IAs. More attention should be paid to this factor in the management of IAs.

## 1. Introduction

Several studies in experimental animals and humans have demonstrated that inflammation plays a major role in the formation, growth, and rupture of intracranial aneurysms (IAs) [1–4]. Aneurysmal wall enhancement (AWE) visualized using gadolinium-enhanced high-resolution magnetic resonance imaging (HRMRI), an emerging technique for intracranial vascular disease assessment, has been reported in several studies [5–11]. AWE is thought to reflect wall inflammation and is a novel imaging biomarker for risk evaluation [3,6–11]. Previous studies have indicated that AWE is more frequently present in ruptured IAs [6–8]; however, a major limitation of these studies is that unruptured and ruptured IAs represent two different physiologic states: the shape or size of IAs may change and may generate an inflammatory response around the wall due to the rupture, giving false positive results. Clinical neurologic symptoms, such as a sudden headache, third nerve palsy or blepharoptosis usually occur prior to the rupture of IAs. Previous studies suggested that neurologic symptoms (e.g., sudden headache or blepharoptosis) relate to the location of the aneurysm and are defined as unstable IAs, while fortuitous and asymptomatic IAs are defined as stable IAs [[3,6–11],12–15]. Symptomatic IAs may be more prone to rupture than asymptomatic IAs. Recently, one study reported that AWE can distinguish between symptomatic and asymptomatic IAs [16]. However, several previous studies have reported that stable or asymptomatic IAs also exhibit AWE [6–10,16]. Furthermore, wall enhancement can even be observed in normal arteries [17]. Therefore, we hypothesized that the enhancement ratio (ER) of AWE would be stronger in symptomatic IAs than in asymptomatic IAs. We measured the wall signal intensity (SI) on pre- and post-enhancement images and investigated whether the ER of AWE could distinguish between symptomatic and asymptomatic IAs.

## 2. Materials and methods

### 2.1. Patients

This retrospective study was approved by our Institutional Ethics Committee, who waived the requirement for informed consent from

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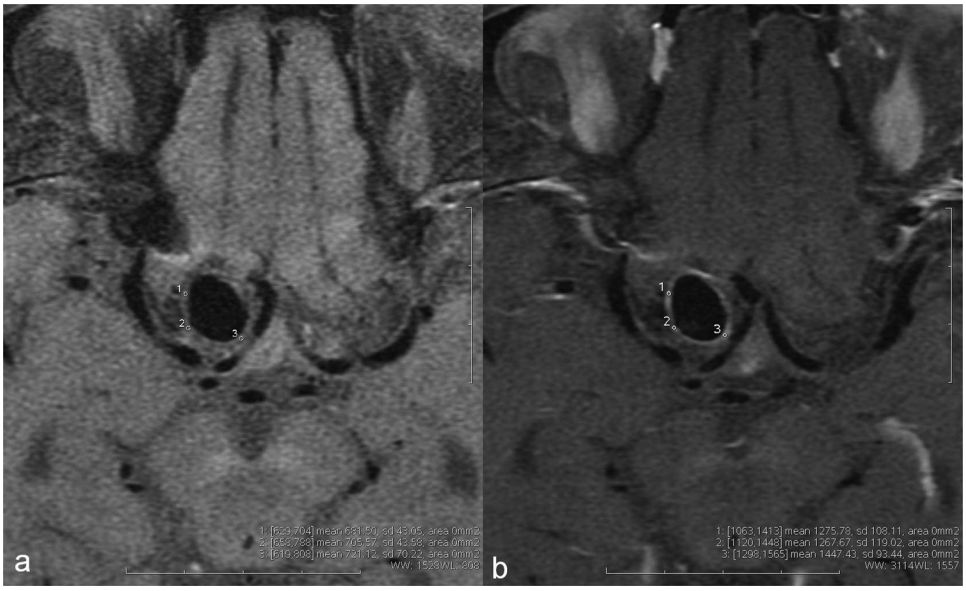


**Fig. 1.** The signal intensity at the body of the IA was measured manually 3 times on pre- (a) and post-enhancement (b) images. The SI values were determined in circular ROIs placed in the aneurysm wall, and the average values were used.

patients. One hundred–twenty patients with saccular unruptured IAs diagnosed by computed tomography angiography (CTA) were recruited to undergo enhanced HRMRI between February 2016 and February 2018. The exclusion criteria were as follows: (1) pregnancy and breast feeding, children, and patients with contraindications to MRI or use of contrast agent (n = 3); (2) patients with subarachnoid hemorrhage due to ruptured IAs diagnosed by CT, operative findings or lumbar puncture (n = 21); (3) patients with multiple unruptured IAs and clinical symptoms; in these cases, we could not determine which IAs caused the symptoms or whether other IAs caused the symptoms (n = 4) (patients with multiple IAs but no clinical symptoms or for whom we knew which IAs caused their symptoms were included); (4) patients with a recent history of using aspirin or nonsteroidal anti-inflammatory drugs because these drugs may reduce the degree of AWE (n = 4). and (5) patients with fusiform, traumatic, dissecting IAs, and those with arteriovenous malformations or poor image quality (n = 8).

Finally, 80 patients and 89 IAs met the inclusion criteria. Thirty-seven IAs were treated because of neurologic symptoms or because they had an irregular shape, and 52 were observed. One IA ruptured 19 days later, and the remaining were followed-up with CTA or magnetic resonance angiography (MRA) (mean 17.02 months, range 3–36 months) and did not develop symptoms or rupture. Based on previous literature [8,12–15], the patients and IAs were divided into two groups: a symptomatic group including patients with neurologic symptoms (e.g., sudden headache or blepharoptosis) related to the location of the aneurysm and an asymptomatic group including patients with fortuitous and asymptomatic IAs.

The patients' clinical data, such as sex, age and history of hypertension and heart disease, were collected using electronic medical records by one of the assessors, who was the only individual who faithfully recorded clinical data. The neurologic symptoms, whether or not they were related to the aneurysm, were determined by two readers (one with 25 years of experience in neurosurgery and the other with 15 years of experience in neurology).

### 2.2. Imaging protocol and analysis

#### 2.2.1. CTA

All patients initially underwent CTA on a 64-slice CT machine (GE LightSpeed VCT; GE Healthcare, Milwaukee, Wisconsin, USA). After the contrast agent was injected into the antecubital vein at a rate of 4–4.5 ml/s, three-dimensional (3D) volume-rendered (VR) images were obtained. All images were analyzed by 2 experienced readers (one with 20 years of experience in neuroradiology and the other with five years of experience in vascular imaging). The readers identified the best view angle to measure the IA morphological indices, including the size (neck width, depth, width and maximum size), flow angle, location, bifurcation (presence or absence) and shape. The location of the IAs was divided into two groups: anterior circulation and posterior circulation. Bifurcation aneurysms were defined as lesions that originated from major bifurcations. If an aneurysm originated from only one parent vessel or at a branch that was substantially smaller than the parent vessel (< 1/5th of the diameter) visible on CTA, the aneurysm was defined as a sidewall aneurysm. The shape of the IAs was categorized as simple lobed or irregular, and IAs with lobular or daughter sacs were classified as irregular. These variables were identified and are described in our previous studies [18–21].

#### 2.2.2. MRI

All patients underwent MR examination on a 3.0 T scanner (Signa HDx, GE Healthcare, Milwaukee, Wisconsin, USA) with an 8-channel head coil. The examination protocol included 3D time-of-flight (TOF) MRA and pre- and post-contrast T1-weighted HRMRI.

3D-TOF-MRA was performed using the following parameters for the localization of subsequent scans: TR/TE = 25/3.4 ms; flip angle = 15°; field of view = 22 × 19.8 cm; acquired matrix = 384 × 160; slice thickness = 1.2 mm; and layer spacing = 0 mm. The images generated 3D VR and a maximum intensity projection (MIP). The imaging time was approximately 3 min 42 s.

Pre- and post-contrast T1-weighted HRMRI were performed using the following parameters: TR/TE = 580/11 ms; field of view = 16 × 16 cm; acquired matrix = 384 × 224; slice thickness = 1.2 mm; and layer spacing = 0 mm. All patients were administered an intravenous injection of a single dose (0.1 mmol/kg) of Gd-BOTPA (MultiHance; Bracco, Shanghai, China). After the contrast agent was injected, each aneurysm scan was performed four times: axial, coronal, sagittal and axial planes. The total scan time was 3 min 8 s per sequence.

The two readers, who were blinded, subsequently determined the SI of the IA manually on the pre- and post-enhancement images magnified 3-fold on the PACS system [22]. The regions of interest (ROIs) were placed at the IA neck, body and dome portion to measure SI, and at each area SI was measured 3 times, respectively (Fig. 1). Then, as a total

9 times SI were measured to calculate the average SI for each enhancement sequence. The maximum average SI was selected from the four-enhancement sequence after 36 times SI were measured. The ER was calculated as follows: ER = (SImax–SIpre)/SIpre × 100% [22]. The wall enhancement patterns included no, entire and partial wall enhancement as described in our previous study [21]. The average values of the continuous data between the two readers were used for the subsequent statistical analyses.

### 2.3. Statistical analysis

Data analysis was performed using SPSS 17.0 (Inc., Chicago, IL, USA), and a *P* value less than 0.05 was considered statistically significant. The agreement between two observers for the neurologic symptoms of the IAs was evaluated by a kappa value. Inter-observer agreement of the ER measurements was evaluated by Mann–Whitney U test analysis. Categorical data were expressed as numbers with percentages and were compared using Fisher's exact test. Continuous data were presented as the means ± standard deviation and were analyzed by independent-samples Student's *t*-test (for normally distributed data) or the Mann–Whitney U test (for non-normal distributed data). All variables were entered into a univariate analysis if $P \leq 0.2$. The variables that achieved univariate analysis significance ($P < 0.05$) were further analyzed using forward multiple logistic regression to calculate the 95% confidence interval (CI) and odds ratio (OR) for the likelihood of unstable IAs. Receiver operating characteristic (ROC) curve analysis was performed for the final model; the cut-off point was determined using the Youden index when the value of (sensitivity + specificity-1) reached its maximum.

## 3. Results

The inter-observer agreement was excellent for the neurologic symptoms ($\kappa = 0.877$, $P < 0.001$). Eighty patients and 89 IAs met the inclusion criteria. Of these patients, 54 were female, and 26 were male (female:male ratio of 2.08:1). The mean age was 57.18 ± 9.19 years. There were 50 asymptomatic patients and 30 symptomatic patients. The clinical characteristics of the 80 patients are listed in Table 1. No associations were identified between the clinical characteristics and the symptomatic patients.

There was no significant difference between two observers for the ER measurements ($P = 0.972$). The geometric and morphological characteristics of the asymptomatic and symptomatic IAs are presented in Table 2. IAs with thrombus, irregular shape, depth, width, maximum size, ER, enhanced patterns and flow angle were associated with symptomatic IAs.

These variables ($P \leq 0.2$) were subsequently entered into an univariate logistic regression model. Then, the variables ($P \leq 0.05$) were entered into a forward conditional multiple logistic regression model to determine the risk factors for symptomatic IAs. Only the ER was associated with symptomatic IAs (Table 3).

The threshold value of the ER was 60.5%, and the area under the curve was 0.903 (Fig. 2). The sensitivity and specificity for the detection of symptomatic IAs were 90.3% and 87.9% (Table 4).

**Table 1**
The clinical characteristics of patients with aneurysms.

| Clinical data | Asymptomatic patients (n = 50), n (%) | Asymptomatic patients (n = 30), n (%) | *P* |
|---|---|---|---|
| Female | 35(70.0) | 19(63.3) | 0.624 |
| Age (Y) | 58.36 ± 9.75 | 55.87 ± 8.76 | 0.227 |
| Hypertension | 22(44.0) | 12(40.0) | 0.817 |
| Heart disease | 7(14.0) | 1(3.3) | 0.247 |
| Diabetes mellitus | 4(8.0) | 2(6.9) | 1.000 |
| Cerebral atherosclerosis | 13(26.0) | 4(13.3) | 0.260 |
| Current alcohol | 7(14.0) | 5(16.7) | 0.756 |
| Current smoking | 8(16.0) | 7(23.3) | 0.555 |
| Multiple aneurysms | 7(14.0) | 2(6.9) | 0.471 |

**Table 2**
The morphological characteristics of aneurysms.

| Morphologic parameters | Asymptomatic IAs (n = 58), n(%) | Symptomatic IAs (n = 31), n(%) | *P* |
|---|---|---|---|
| Anterior circulation | 56(96.6) | 30(96.8) | 1.000 |
| Bifurcation | 20(34.5) | 16(51.6) | 0.173 |
| Thrombus | 0(0.0) | 4(100) | 0.013 |
| Irregular Shape | 14(24.1) | 20(64.5) | < 0.001* |
| Neck width (mm) | 4.24 ± 1.19 | 5.50 ± 2.72 | 0.065* |
| Depth (mm) | 3.90 ± 1.43 | 10.49 ± 9.09 | < 0.001* |
| Width (mm) | 4.16 ± 1.66 | 9.41 ± 7.34 | < 0.001* |
| Maximum Size (mm) | 5.11 ± 1.60 | 12.04 ± 9.92 | < 0.001* |
| Enhanced ratio | 0.32 ± 0.30 | 1.12 ± 0.87 | < 0.001* |
| Enhanced patterns | | | |
| No | 25(43.1) | 0(0.0) | < 0.001* |
| Partial | 2(3.4) | 5(16.1) | 0.047* |
| Entire | 31(53.5) | 26(83.9) | 0.005* |
| Flow angle (°) | 107.72 ± 27.32 | 124.40 ± 28.39 | < 0.001* |

* Variables showing significant difference by univariate analysis ($P < 0.05$).

**Table 3**
Multiple logistic regression analysis for all aneurysms.

| Variable | Odds ratio | *P* | 95% CI | β |
|---|---|---|---|---|
| ER | 29.443 | < 0.001 | 4.670–185.628 | 3.382 |

ER, enhancement ratio; CI, Confidence interval; β, partial regression coefficient.

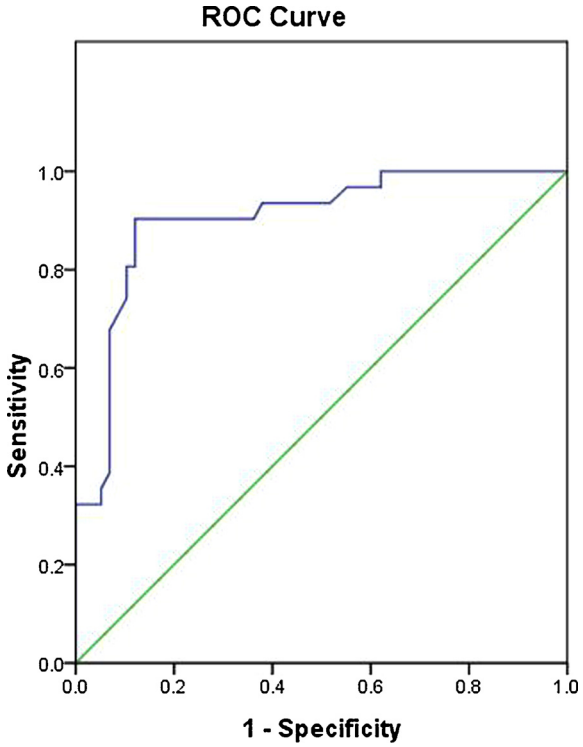


**Fig. 2.** According to the clinical risk, the area under the receiver operating characteristic curve for the ER was 0.903 (95% confidence interval, 0.836–0.970). The cut-off point for the ER was 60.5%, the sensitivity was 90.3%, and the specificity was 87.9%.

**Table 4**
Area under the curve for enhancement ratio.

| Characteristics | Area | Threshold value (%) | *P* | Sen (%) | Spe (%) | 95% CI |
|---|---|---|---|---|---|---|
| ER | 0.903 | 60.5 | < 0.001 | 90.3 | 87.9 | 0.836–0.970 |

ER, enhancement ratio; Sen, sensitivity; Spe, specificity; CI, confidence interval; Threshold value, the cut-off for the enhancement ratio.

## 4. Discussion

Recently, several studies have reported that AWE can be visualized on enhanced HRMRI and is thought to reflect wall inflammation [3–11]. However, AWE has not been sufficiently investigated. For example, the relationship between the degree of enhancement and symptomatic IAs remains unclear. In this study, we found that the ER is the only independent factor associated with symptomatic IAs.

Symptomatic IAs are more prone to rupture than asymptomatic IAs [16]. Hu et al. [7] reported that wall enhancement had a highly consistent correlation with symptomatic IAs; however, their study also included ruptured IAs. Edilali et al. [8] reported that an AWE was observed in 6 of 9 symptomatic IAs, but they did not analyze the relationship between the AWE and clinical symptoms. A previous study suggested that IAs with AWE had a significantly higher International Study of Unruptured Intracranial Aneurysms (ISUIA) grade than that of IAs without AWE, though they did not include clinical symptoms as risk factors [9]. Recently, one study investigated 45 unruptured IAs and found that the AWE was associated with sentinel headaches and third nerve palsy [16]. All of these previous studies demonstrated that AWE was more frequently found in symptomatic IAs. In our study, the enhancement was found to be higher in the symptomatic IAs, but we observed partial or entire enhancement in 33 asymptomatic IAs, and multiple analysis revealed no correlation, which could be due to the sample size and the source of the sample.

Most previous studies used qualitative assessments to demonstrate that AWE was more frequently found in ruptured or symptomatic IAs; however, some small asymptomatic IAs could also be enhanced. Thus, relying on the presence of AWE to determine whether the aneurysm is symptomatic may lead to false-positives. A previous study defined AWE as nonevident, faint or strong, indicating that strong enhancement had a high specificity for ruptured IAs [9]. However, this evaluation method may lead to different opinions because discriminating absent and faint enhancement from faint and strong enhancement is often difficult. To avoid these issues, Omodaka et al. [23] used a quantitative method and found that the wall enhancement index was significantly higher in ruptured versus unruptured IAs. Omodaka et al. found that the cut-off value was 0.53 and the sensitivity was 96%; however, the specificity was only 47%. In our previous study, we measured the wall SI on pre- and post-enhancement images and calculated the ER, and we found that the ER ($\geq$0.615) was a better predictor of rupture, with a sensitivity 89.5% and a specificity 63.2% [21]. In the present study, the threshold value of the ER ($\geq$0.605) was determined with high sensitivity and high specificity in symptomatic IAs. Although no consensus exists regarding a common threshold value, this finding also supports our hypothesis and the concept that the ER of AWE is stronger in symptomatic than in asymptomatic IAs.

Theoretically, a larger IA is more likely to compress surrounding structures and cause clinical symptoms. Previous study have reported that treatment decisions regarding unruptured IAs are based mainly on size and location [24]. However, Fu et al. [16] investigated 45 unruptured IAs in 37 patients and found no association between size and symptoms. In the present study, IAs were larger in the symptomatic group than in the asymptomatic group, though multiple analysis did not show a correlation. This may be related to the correlation between aneurysm size and wall enhancement, though wall enhancement is a better indicator than aneurysm size. Recently, several studies have demonstrated that aneurysm size is independently associated with AWE [9,25,26]. However, the authors also showed that some small IAs did exhibit AWE. This may be because AWE is associated not only with aneurysm size but also with the degree of inflammation, wall thickness and dense areas in the vasa vasorum, and wall permeability, stagnation, or leakage of contrast agent [[9,25,26],[18,20,21],14]. In our previous clinical experience, ruptured IAs tended to be larger than unruptured ones, while the size of IAs was not significantly different between the ruptured and unruptured groups [18,20,21], nor was there a difference between the unstable and stable groups [19]. These results were also consistent with a recent study [15]. Therefore, size may be not an independent factor for either the rupture of IAs or symptoms.

### 4.1. Limitations

This study has several limitations. First, it had a relatively small sample size. Second, the data were collected from a single center with a very high rate of symptomatic IAs in this consecutive series. This is because many patients who do not need treatment were unwilling to participate in the study. Third, this was a retrospective study, and some patients with asymptomatic IAs may still develop symptoms in the future. Fourth, we did not assess the aneurysmal wall thickness, in part because the aneurysmal wall thickness was that the HRMRI resolution was not sufficiently high to precisely measure the pre-enhancement wall. In addition, the wall thickness on post-enhancement images that was visualized reflected the true wall plus other tissue components [27]. In the future, a prospective study and larger sample size are necessary.

## 5. Conclusion

Based on HRMRI findings, a higher ER correlated with symptomatic IAs. The threshold value of the ER was 60.5%. Moreover, increased attention in clinical practice should be paid to the wall of IAs using this characteristic, actively follow-up or surgery is recommended. Nevertheless, further research is needed to confirm these findings.

## Competing interests statement

The authors have declared that no competing interests exist.

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All authors listed have read the complete manuscript and have approved submission of the paper. All authors declare that there are no conflicts of interest.

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