

## ORIGINAL RESEARCH



# Relationship between hyperintensity on MRI-T1WI and hemorrhagic transformation after cerebral infarction and its influencing factors

Fan Yu<sup>1,\*</sup>, Xiu Su<sup>2</sup>, Dan Liu<sup>3</sup>

<sup>1</sup>Department of Radiology, The First Affiliated Hospital of Chongqing Medical University, 400016 Chongqing, China

<sup>2</sup>Department of Radiology, Liupanshui Second People's Hospital of Guizhou Province, 553400 Liupanshui, Guizhou, China

<sup>3</sup>Department of Radiology, Science City Hospital, Jiulongpo District, 400050 Chongqing, China

**\*Correspondence**[fan\\_y66@163.com](mailto:fan_y66@163.com)

(Fan Yu)

**Abstract**

This study aims to investigate the correlation between hyperintensity on Magnetic Resonance Imaging-T1 weighted imaging (MRI-T1WI) and post-infarction hemorrhagic transformation (HT) after cerebral infarction (CI) and analyze the influencing factors. This retrospective study comprised 115 patients diagnosed with cerebral infarction at our hospital. Their clinical data were collected, and they were then divided into a hyperintensity and a non-hyperintensity group based on their T1WI image characteristics. Comparative analysis was performed and the diagnostic value of T1WI hyperintensity for HT and influencing factors were assessed. Lesions in the 115 cerebral infarction patients were distributed as follows: 52 in the cerebral cortex, 37 in the basal ganglia, 14 in the cerebellum, 7 in the thalamus, and 5 in the subcortex. Hyperintensity on T1WI was observed in 4 cases before treatment, which increased to 27 cases after treatment, including 16 affecting the cerebral cortex. These hyperintense signals manifested as spotty, patchy or linear patterns along the gyri. In the basal ganglia, 10 cases exhibited spotty or patchy signals, surrounded by an annular hypointense shadow. Additionally, 3 cases involved the cerebellum, 1 the thalamus, and 1 the subcortex, all with spotty or patchy hyperintensities. HT occurred in 17 out of 115 patients (14.78%) one month after treatment. The diagnostic performance of T1WI hyperintensity for HT showed sensitivity, specificity, accuracy, positive predictive value, and negative predictive value of 94.12%, 84.69%, 86.09%, 51.61% and 98.81%, respectively, with a Kappa value of 0.588. Multivariate logistic regression analysis revealed that age, atherosclerosis, and infarct size were significant risk factors for T1WI hyperintensity in cerebral infarction ( $p < 0.05$ ). Hyperintensity on T1WI in cerebral infarction primarily correlates with HT and could be a valuable diagnostic marker for HT, with age, atherosclerosis and infarct size identified as potential influencing factors.

**Keywords**

Cerebral infarction; Magnetic resonance imaging; T1-weighted hyperintensity; Hemorrhagic transformation of cerebral infarction; Diagnosis; Influencing factors

## 1. Introduction

Cerebral infarction is a highly prevalent condition in the middle-aged and elderly population, characterized by an acute onset and poor prognosis. Statistics indicate that in China, the incidence rate was approximately 145/100,000 in 2020, with a prevalence as high as 1700/100,000, representing a significant threat to the life safety of Chinese residents [1, 2]. Previous studies have reported that hemorrhagic transformation (HT) is a common complication in patients with cerebral infarction, with an incidence ranging from approximately 10% to 49.5%. HT can be broadly classified into two categories: spontaneous bleeding and bleeding after treatment. Its pathogenesis is currently complex and may be closely related to damage to blood-brain barrier (BBB) integrity caused by inflammation

and oxidative stress following cerebral infarction [3–5]. Imaging examinations play a pivotal role in the diagnosis and treatment of cerebral infarction. It is well-documented in literature that magnetic resonance imaging (MRI) offers several advantages, including excellent soft tissue contrast and absence of radiation, making it more widely employed in clinical practice. Conventional MRI findings typically show hypointensity on T1-weighted images (T1WI) and hyperintensity on T2-weighted images (T2WI). However, some patients may also exhibit hyperintensity on T1WI, which may be closely associated with laminar necrosis, macromolecular hydration effects and HT [6–8].

This present study was designed to assess the relationship between T1WI hyperintensity and HT after cerebral infarction

and its influencing factors to provide evidence-based medical insights to further clarify the diagnostic value of MRI for HT, which could help to improve clinical diagnosis and treatment.

## 2. Materials and methods

### 2.1 General data

This is a retrospective study conducted on a cohort of 115 patients (67 males and 48 females) diagnosed with cerebral infarction at our hospital, with age ranging from 37 to 91 years (mean age  $\pm$  standard deviation:  $66.70 \pm 10.76$  years). The study inclusion criteria were: A diagnosis of cerebral infarction based on clinical symptoms, signs and imaging findings [9]; Age  $\geq 18$  years; Admission within 24 hours from the onset of symptoms; Adherence to treatment guidelines; Completion of both pre-treatment and post-treatment MRI scans within a month; Follow-up for at least one month post-treatment to assess the presence of HT. The exclusion criteria comprised: (1) Coexistence of intracranial tumors, epilepsy, traumatic brain injuries, or other cerebral lesions; (2) Cerebral infarction complicated by cerebral hemorrhage; (3) A history of previous strokes; (4) Underwent intravascular interventional therapy; (5) Had incomplete clinical data which might have affected our study analysis.

### 2.2 Study method

#### 2.2.1 Baseline data

The collected baseline data comprised gender, age, body mass index (BMI), smoking history, drinking history, underlying medical conditions, history of prior cerebral infarction, time interval from symptom onset to admission, National Institute of Health Stroke Scale (NIHSS) score at admission, and Trial of Org 10 172 in acute stroke treatment (TOAST) classification [9].

#### 2.2.2 MRI findings

These were documented both before treatment and within 1 month after treatment for both study groups. The imaging was performed using the BRIVO MR355 1.5T magnetic resonance scanner from GE Company (Boston, MA, USA), and the findings of interest included the distribution characteristics of T1WI and T2WI signals. The infarct lesions were delineated on T2WI images, and the infarct size was calculated at the section of maximum involvement.

#### 2.2.3 Treatment

Treatment regimens included intravenous thrombolysis, arterial thrombolysis, administration of antiplatelet drugs, anticoagulant drugs, medications aimed at enhancing circulation and neurotrophic drugs.

#### 2.2.4 Incidence of HT

The occurrence of HT within 1 month after the procedure was followed up in both study groups. Diagnosis was established based on the absence of hemorrhagic lesions on the initial MRI, followed by the observation of hemorrhagic lesions upon subsequent MRI examinations. Cases that could not be definitively diagnosed on the MRI were verified through computed

tomography (CT) scans [10].

### 2.2.5 Data analysis

The patients were categorized into a hyperintensity or a non-hyperintensity group based on their T1WI image characteristics. Comparative analysis between these two groups was conducted, and the relationship between hyperintensity on T1WI and HT, as well as its diagnostic value, was assessed using the Kappa consistency test. Subsequently, factors influencing hyperintensity on T1WI were analyzed, and the potential factors affecting the diagnostic accuracy of hyperintensity on T1WI for HT were explored.

### 2.3 Statistical methods

Enumeration data are presented as  $n$  (%) and analyzed using the  $\chi^2$  test. For measurement data that followed a normal distribution, they are expressed as  $\bar{x} \pm s$  and analyzed using the independent samples  $t$ -test. The relationship between T1WI hyperintensity and HT, as well as its diagnostic value, was assessed using the Kappa consistency test. Multivariate logistic regression was used to analyze the factors influencing T1WI hyperintensity. Statistical analysis was conducted using the SPSS (BMI Corporation, Chicago, IL, USA) 25.0 software, with a significance level set at  $\alpha = 0.05$ .

## 3. Results

### 3.1 MRI image characteristics of cerebral infarction and HT

In this study, among the 115 patients diagnosed with cerebral infarction, the lesion distribution was as follows: 52 in the cerebral cortex, 37 in the basal ganglia, 14 cases in the cerebellum, 7 cases in the thalamus, and 5 cases in the subcortex. Prior to treatment, MRI examinations identified 4 cases exhibiting T1WI. Following treatment, 27 cases displayed hyperintensity on T1WI, including 16 cases within the cerebral cortex. These hyperintense signals were characterized by spotty, patchy or linear patterns along the gyri. Specifically, 10 cases were located in the basal ganglia, showing spotty or patchy signals that were encircled by an annular hypointense shadow. Additionally, 3 cases had lesions in the cerebellum, 1 in the thalamus, and 1 in the subcortex, all demonstrating spotty or patchy hyperintensities.

### 3.2 Diagnostic value of hyperintensity on T1WI for HT

The outcomes of the 1-month follow-up revealed that among the 115 patients, 17 cases (14.78%) developed HT. The diagnostic sensitivity of T1WI hyperintensity for detecting HT was 94.12% (16/17), while the specificity was 84.69% (83/98). The overall accuracy was 86.09% (99/115), with a positive predictive value of 51.61% (15/31) and a negative predictive value of 98.81% (83/84). The degree of agreement, as indicated by the kappa value, was 0.588 (Table 1).

**TABLE 1. Diagnostic value of hyperintensity on T1WI for HT.**

T1WI hyperintensity	HT follow-up results		Total
	+	–	
+	25	6	31
–	1	83	84
Total	17	98	115

*T1WI: T1-weighted images; HT: hemorrhagic transformation.*

### 3.3 Comparison of clinical data between the hyperintensity group and the non-hyperintensity group on T1WI

Our analysis revealed significant differences between the T1WI hyperintense and non-hyperintense groups in terms of age, drinking history, atherosclerosis, cerebral infarction area, and thrombolytic therapy ( $p < 0.05$ ) (Table 2).

### 3.4 Influencing factors of T1WI hyperintensity

Multivariate logistic regression analysis demonstrated that age, atherosclerosis and infarct size were significant risk factors associated with T1WI hyperintensity in cerebral infarction ( $p < 0.05$ ) (Table 3).

## 4. Discussion

HT in cerebral infarction typically manifests between 36 hours and 1 month after the onset of the illness. Due to the absence of standardized diagnostic criteria across various regions, its statistics in epidemiological reports often vary significantly. For instance, findings from the French randomized trial indicated that the incidence of HT in endovascular treatment was 46%, with a symptomatic HT rate of 2.0% [11]. In addition, the incidence of HT in cerebral infarction patients from Asian populations appears to be notably higher compared to that observed in Europe and the United States. According to a Chinese multicenter study, the incidence was reported as 49.5%, with symptomatic hemorrhage rates reaching as high as 9.9%, surpassing data reported in Western literature [12, 13].

Imaging technology plays a pivotal role in diagnosing HT in cerebral infarction. Currently, the most commonly employed techniques are MRI and CT, with MRI being the preferred choice for assessing cerebral infarction due to its excellent soft tissue contrast, ability for multi-parameter imaging, and quantitative analysis capabilities [14–16]. T1WI hyperintensity is recognized as a distinctive abnormal imaging feature in the assessment of cerebral infarction. Previous literature has proposed various causes for T1WI hyperintensity, including neuronal necrosis [17], gliosis [18] and vascular rupture and hemorrhage [19]. In cerebral infarction, HT gradually leads to the breakdown of red blood cells over time, resulting in methemoglobinemia characterized by paramagnetic features during the deposition and degradation of hemoglobin. Additionally, macromolecular proteins can induce hydration effects, causing MRI to reveal T1WI hyperintensity [7, 20].

In this study, we conducted a retrospective analysis of MRI follow-up results from 115 patients diagnosed with cerebral infarction, which showed that 4 cases exhibited T1WI hyperintensity before treatment, and an additional 27 cases displayed T1WI hyperintensity 1 month after treatment, resulting in a total of 31 cases. These hyperintensity signals were predominantly distributed in a patchy, spotty or linear pattern and were distributed across various brain regions, including 16 cases in the cerebral cortex, 10 in the basal ganglia, 3 in the cerebellum, 1 in the thalamus, and 1 in the subcortical region. These findings were consistent with previous literature reports [21]. Additionally, our study identified 17 patients (14.78%) diagnosed with HT through 1-month follow-up, a prevalence lower than what has been reported in epidemiological studies both in China and internationally, which might have been attributed to the patient selection criteria used in our present study. Upon further investigation into the relationship between T1WI hyperintensity and HT, we determined that the kappa value signifying agreement between the two variables was 0.588, indicating a moderate level of correlation. When T1WI hyperintensity was used for the diagnosis of HT in patients with cerebral infarction, the sensitivity and specificity were 94.12% and 84.69%, respectively, indicating a valuable reference for clinical diagnosis. The negative predictive value was high at 98.81%, signifying strong exclusion capabilities for HT. However, the positive predictive value was relatively lower at 51.61%, mainly due to an increased number of false positives. This limitation was attributed to the presence of numerous interfering factors in the assessment of T1WI hyperintensity for HT diagnosis.

MRI imaging operates on the fundamental principle of observing the vibrational signal of H<sup>+</sup> (protons) within a magnetic field. Research has demonstrated that the freedom of water molecule activity plays a central role in influencing signal characteristics. Typically, fluids, tumors or infarcted areas exhibit hypointensity on T1WI. Conversely, when water molecule activity is restricted, the T1 relaxation time shortens, leading to hyperintensity [22–24]. However, the pathophysiological manifestations of cerebral infarction are intricate, and various factors, including the location, type and severity of the infarction, can significantly interfere with the signal characteristics of T1WI. Furthermore, systemic factors such as blood pressure, blood composition, blood glucose levels, and therapeutic interventions may also contribute to variations in T1WI signal characteristics, thereby affecting the accuracy of T1WI hyperintensity in HT diagnosis. In our study, the results of multivariate logistic regression analysis revealed that age, atherosclerosis and infarct size were risk factors associated with T1WI hyperintensity in cerebral infarction. As individuals age, brain tissue can undergo irreversible degeneration, potentially exacerbated by conditions such as hypertension and diabetes. This aging process can result in the gradual hardening of cerebral vessels and increased permeability. Additionally, inflammation and oxidative stress following cerebral infarction, among other factors, may lead to neuronal necrosis, which may in turn cause the accumulation of lipids and proteins or the formation of calcifications. These processes can result in the adsorption of water molecules, ultimately leading to T1WI hyperintensity [25]. Atherosclerosis remains one of the important

**TABLE 2. Comparison of clinical data between the hyperintensity and non-hyperintensity group on T1WI.**

Clinical data	Hyperintensity group (n = 31)	Non-hyperintensity group (n = 84)	$\chi^2/t$	<i>p</i>
Gender (n (%))				
Male	19 (61.29)	48 (57.14)	0.160	0.689
Female	12 (38.71)	36 (42.86)		
Age (n (%))				
≥60 years	28 (90.32)	57 (67.86)	5.927	0.015
<60 years	3 (9.68)	27 (32.14)		
BMI (kg/m <sup>2</sup> )	23.20 ± 2.84	22.64 ± 2.76	0.958	0.340
Smoking history (n (%))	7 (22.58)	18 (21.43)	0.018	0.894
Alcohol history (n (%))	11 (35.48)	15 (17.86)	4.021	0.045
Underlying disease (n (%))				
Hypertension	13 (41.94)	29 (34.52)	0.537	0.464
Diabetes	17 (56.67)	35 (41.67)	2.005	0.157
Hyperlipidemia	18 (58.06)	36 (42.86)	2.102	0.147
Atherosclerosis	19 (61.29)	31 (36.90)	5.479	0.019
History of cerebral infarction (n (%))	6 (19.35)	9 (10.71)	1.490	0.222
Onset to admission (h)	10.97 ± 3.46	10.81 ± 2.83	0.237	0.813
NIHSS score (points)	9.74 ± 2.13	9.73 ± 2.01	0.070	0.944
TOAST typing				
Large artery atherosclerosis type	15 (48.39)	31 (36.90)	2.832	0.418
Other/unknown etiology	12 (38.71)	38 (45.24)		
Arteriolar occlusion	4 (12.90)	10 (11.90)		
Cardioembolism	0 (0)	5 (5.95)		
Cerebral infarction area (cm <sup>2</sup> )	3.84 ± 0.97	3.39 ± 0.98	2.191	0.031
Thrombolytic therapy (n (%))	19 (61.29)	32 (38.10)	4.936	0.026

BMI: body mass index; NIHSS: National Institute of Health Stroke Scale; TOAST: Trial of Org 10 172 in acute stroke treatment.

**TABLE 3. Influencing factors of T1WI hyperintensity.**

Risk factors	$\beta$ value	SE	$\chi^2$	OR value	95% CI	<i>p</i>
Age	0.092	0.027	11.610	1.096	1.040–1.156	<0.001
Atherosclerosis	1.265	0.489	6.692	3.543	1.359–9.239	0.010
Infarct size	0.143	0.051	7.862	1.154	1.044–1.275	0.005

SE: Standard Error; OR: Odds Ratio; CI: Confidence interval.

factors causing cerebral infarction and cerebral hemorrhage. Vascular lesions and abnormal cerebral perfusion may also lead to neuronal and myelin structural damage, destroy the BBB and promote gliosis. It can also cause complex T1WI signal changes, some of which show T1WI hyperintensity [26–28]. The extent of the cerebral infarction area represents a significant risk factor for the occurrence of hyperintensity on T1WI. Some studies have established a connection between the cerebral infarction area and the development of hyperintensity on T1WI, which is closely associated with HT. For instance, Liu *et al.* [29] reported that extensive cerebral infarction serves as a crucial risk factor for HT following thrombolysis. Additionally, severe edema and hypoxia resulting from extensive infarction can give rise to laminar necrosis, which constitutes

another important underlying cause of T1WI hyperintensity [30].

## 5. Conclusions

In conclusion, T1WI hyperintensity often manifests in cerebral infarction as the disease progresses and is primarily associated with HT, offering valuable diagnostic insights. Nevertheless, this phenomenon can be influenced by factors such as age, atherosclerosis and infarct size. Thus, to enhance diagnostic accuracy, future studies may consider excluding or refining and quantifying T1WI signal characteristics while addressing these factors. This study has two primary limitations. Firstly, the sample size was relatively small (115 cases) and had a

limited number (17 cases) of HT patients, potentially introducing statistical bias. Expanding the sample size in subsequent studies would be essential to validate the results. Secondly, the exclusion of intracranial tumors, epilepsy, traumatic brain injuries and other brain lesions or injuries may not fully account for the practical clinical interference that these factors may pose to T1WI images of HT patients. As research advances and MRI technology continues to evolve, the relationship between T1WI hyperintensity and HT in patients with cerebral infarction is becoming clearer, thereby providing valuable insights for HT prediction and diagnosis; however, the full clinical implications and significance of these findings remain to be analyzed and further elucidated.

## AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

## AUTHOR CONTRIBUTIONS

FY—designed the study and carried them out; FY, XS—analyzed the data; FY, XS, DL—supervised the data collection, interpreted the data, prepare the manuscript for publication and reviewed the draft of the manuscript. All authors have read and approved the manuscript.

## ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of the First Affiliated Hospital of Chongqing Medical University (No.: 202103284). Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

## ACKNOWLEDGMENT

Not applicable.

## FUNDING

This research received no external funding.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## REFERENCES

- [1] Muratova T, Khramtsov D, Stoyanov A, Vorokhta Y. Clinical epidemiology of ischemic stroke: global trends and regional differences. *Georgian Medical News*. 2020; 83–86.
- [2] Stuntz M, Busko K, Irshad S, Paige T, Razhkova V, Coan T. Nationwide trends of clinical characteristics and economic burden of emergency department visits due to acute ischemic stroke. *Open Access Emergency Medicine*. 2017; 9: 89–96.
- [3] Wang H, Zi W, Hao Y, Yang D, Shi Z, Lin M, *et al*. Direct endovascular treatment: an alternative for bridging therapy in anterior circulation large-vessel occlusion stroke. *European Journal of Neurology*. 2017; 24: 935–943.
- [4] Spronk E, Sykes G, Falcione S, Munsterman D, Joy T, Kamtchum-Tatuene J, *et al*. Hemorrhagic transformation in ischemic stroke and the role of inflammation. *Frontiers in Neurology*. 2021; 12: 661955.
- [5] Arba F, Piccardi B, Palumbo V, Biagini S, Galmozzi F, Iovene V, *et al*. Blood-brain barrier leakage and hemorrhagic transformation: the reperfusion injury in ischemic stroke (RISK) study. *European Journal of Neurology*. 2021; 28: 3147–3154.
- [6] Lee KY, Suh SH, Ahn SJ. Significance of hyperintense arteries on Gd-enhanced 3D T1W black-blood imaging in acute stroke. *European Radiology*. 2019; 29: 1329–1337.
- [7] Potreck A, Mutke MA, Weyland CS, Pfaff JAR, Ringleb PA, Mundiyanapurath S, *et al*. Combined perfusion and permeability imaging reveals different pathophysiologic tissue responses after successful thrombectomy. *Translational Stroke Research*. 2021; 12: 799–807.
- [8] Kersemans V, Wallington S, Allen PD, Gilchrist S, Kinchesh P, Browning R, *et al*. Manganese-free chow, a refined non-invasive solution to reduce gastrointestinal signal for T<sub>1</sub>-weighted magnetic resonance imaging of the mouse abdomen. *Lab Animal*. 2020; 54: 353–364.
- [9] Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, *et al*. 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline from the American Heart Association/American Stroke Association. *Stroke*. 2021; 52: e364–e467.
- [10] Chen G, Wang A, Zhao X, Wang C, Liu L, Zheng H, *et al*. Frequency and risk factors of spontaneous hemorrhagic transformation following ischemic stroke on the initial brain CT or MRI: data from the China National Stroke Registry (CNSR). *Neurological Research*. 2016; 38: 538–544.
- [11] Bracard S, Ducrocq X, Mas JL, Soudant M, Oppenheim C, Moulin T, *et al*. Mechanical thrombectomy after intravenous alteplase versus alteplase alone after stroke (THRACE): a randomised controlled trial. *The Lancet Neurology*. 2016; 15: 1138–1147.
- [12] Shen AY, Yao JF, Brar SS, Jorgensen MB, Chen W. Racial/ethnic differences in the risk of intracranial hemorrhage among patients with atrial fibrillation. *Journal of the American College of Cardiology*. 2007; 50: 309–315.
- [13] Hao Y, Yang D, Wang H, Zi W, Zhang M, Geng Y, *et al*. Predictors for symptomatic intracranial hemorrhage after endovascular treatment of acute ischemic stroke. *Stroke*. 2017; 48: 1203–1209.
- [14] Ande SR, Grynspan J, Aviv RI, Shankar JJS. Imaging for predicting hemorrhagic transformation of acute ischemic stroke—a narrative review. *Canadian Association of Radiologists Journal*. 2022; 73: 194–202.
- [15] Tian B, Tian X, Shi Z, Peng W, Zhang X, Yang P, *et al*. Clinical and imaging indicators of hemorrhagic transformation in acute ischemic stroke after endovascular thrombectomy. *Stroke*. 2022; 53: 1674–1681.
- [16] Gil-Garcia CA, Flores-Alvarez E, Cebrian-Garcia R, Mendoza-Lopez AC, Gonzalez-Hermosillo LM, Garcia-Blanco MD, *et al*. Essential topics about the imaging diagnosis and treatment of hemorrhagic stroke: a comprehensive review of the 2022 AHA guidelines. *Current Problems in Cardiology*. 2022; 47: 101328.
- [17] Qiu L, Fu F, Zhang W, He J, Zhan Z, Cheng Z. Prevalence, risk factors, and clinical outcomes of remote intracerebral hemorrhage after intravenous thrombolysis in acute ischemic stroke: a systematic review and meta-analysis. *Journal of Neurology*. 2023; 270: 651–661.
- [18] Qiu L, Cai Y, Geng Y, Yao X, Wang L, Cao H, *et al*. Mesenchymal stem cell-derived extracellular vesicles attenuate tPA-induced blood-brain barrier disruption in murine ischemic stroke models. *Acta Biomaterialia*. 2022; 154: 424–442.
- [19] Goncalves A, Su EJ, Muthusamy A, Zeitelhofer M, Torrente D, Nilsson I, *et al*. Thrombolytic tPA-induced hemorrhagic transformation of ischemic stroke is mediated by PKC $\beta$  phosphorylation of occludin. *Blood*. 2022; 140: 388–400.
- [20] Roelz R, Scheiwe C, Grauvogel J, Csok I, Coenen VA, Beck J, *et al*. Early cisternal fibrinolysis is more effective than rescue spasmolysis for the prevention of delayed infarction after subarachnoid haemorrhage. *Stroke and Vascular Neurology*. 2022; 7: 108–113.

- [21] Kidwell CS, Chalela JA, Saver JL, Starkman S, Hill MD, Demchuk AM, *et al.* Comparison of MRI and CT for detection of acute intracerebral hemorrhage. *JAMA*. 2004; 292: 1823–1830.
- [22] Glover GH. Overview of functional magnetic resonance imaging. *Neurosurgery Clinics of North America*. 2011; 22: 133–139.
- [23] Jayachandran Preetha C, Meredig H, Brugnara G, Mahmutoglu MA, Foltyn M, Isensee F, *et al.* Deep-learning-based synthesis of post-contrast T1-weighted MRI for tumour response assessment in neuro-oncology: a multicentre, retrospective cohort study. *The Lancet Digital Health*. 2021; 3: e784–e794.
- [24] Paing MP, Tungjitkusolmun S, Bui TH, Visitsattapongse S, Pintavirooj C. Automated segmentation of infarct lesions in T1-weighted MRI scans using variational mode decomposition and deep learning. *Sensors*. 2021; 21: 1952.
- [25] Lindroth H, Nair VA, Stanfield C, Casey C, Mohanty R, Wayer D, *et al.* Examining the identification of age-related atrophy between T1 and T1 + T2-FLAIR cortical thickness measurements. *Scientific Reports*. 2019; 9: 11288.
- [26] Gutierrez J, Turan TN, Hoh BL, Chimowitz MI. Intracranial atherosclerotic stenosis: risk factors, diagnosis, and treatment. *The Lancet Neurology*. 2022; 21: 355–368.
- [27] Shen M, Gao P, Zhang Q, Jing L, Yan H, Li H. Middle cerebral artery atherosclerosis and deep subcortical infarction: a 3T magnetic resonance vessel wall imaging study. *Journal of Stroke and Cerebrovascular Diseases*. 2018; 27: 3387–3392.
- [28] Shenoy AS, Bhosale MN, Amonkar GP. An autopsy study of cerebrovascular atherosclerosis in the elderly. *Neurology India*. 2022; 70: 2411–2415.
- [29] Liu L, Luo GQ, Liu Q, Yang ZY. Hemorrhagic risk factors after rt PA thrombolysis in acute cerebral infarction. *European Review for Medical and Pharmacological Sciences*. 2023; 27: 5542–5551.
- [30] Cannella R, Sparacia G, Lo Re V, Oddo E, Mamone G, Miraglia R. Advanced magnetic resonance imaging of cortical laminar necrosis in patients with stroke. *The Neuroradiology Journal*. 2019; 32: 431–437.

**How to cite this article:** Fan Yu, Xiu Su, Dan Liu. Relationship between hyperintensity on MRI-T1WI and hemorrhagic transformation after cerebral infarction and its influencing factors. *Signa Vitae*. 2024; 20(1): 106-111. doi: 10.22514/sv.2024.005.