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Review Article

MRI Evaluation of Stroke: Differentiating Haemorrhagic and Ischemic Pathologies

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Abstract: *Stroke remains one of the leading causes of death and long-term neurological disability worldwide. Differentiating ischemic stroke from haemorrhagic stroke is the first and most urgent imaging priority because management pathways differ substantially. Ischemic stroke may require intravenous thrombolysis, endovascular thrombectomy, antiplatelet therapy, or anticoagulation in selected cases, whereas haemorrhagic stroke requires urgent blood pressure control, reversal of anticoagulation when indicated, neurosurgical assessment, and prevention of hematoma expansion. Although computed tomography is widely used in emergency stroke pathways because of its rapid availability, magnetic resonance imaging provides superior tissue characterization and can identify early ischemia, blood products, vascular occlusion, perfusion abnormality, haemorrhagic transformation, and stroke mimics.* ***Objective:*** *This review summarizes the role of MRI in differentiating haemorrhagic and ischemic stroke, with emphasis on MRI sequences, temporal lesion evolution, diagnostic pitfalls, advanced imaging techniques, structured reporting, and clinical decision-making.* ***Methods:*** *Recent peer-reviewed literature, including original studies, meta-analyses, imaging-based stroke studies, and contemporary guidelines published within the last five years, was reviewed. Key MRI sequences discussed include diffusion-weighted imaging, apparent diffusion coefficient mapping, fluid-attenuated inversion recovery, gradient echo imaging, susceptibility-weighted imaging, perfusion MRI, arterial spin labeling, magnetic resonance angiography, and vessel wall imaging.* ***Results:*** *Diffusion-weighted imaging with ADC mapping remains the most sensitive MRI method for detecting acute ischemic injury, whereas GRE and SWI are central to identifying intracranial haemorrhage, microbleeds, superficial siderosis, thrombus susceptibility, and haemorrhagic transformation. FLAIR supports lesion-age assessment and DWI-FLAIR mismatch evaluation in unknown-onset stroke. Perfusion MRI and arterial spin labeling provide information about tissue-at-risk, delayed transit, collateral physiology, and post-recanalization haemodynamic changes. MRA detects large-vessel occlusion, stenosis, dissection, and vascular malformation, while vessel wall MRI improves etiological classification in selected patients.* ***Conclusion:*** *MRI is a highly valuable modality for differentiating ischemic and haemorrhagic stroke when performed using a rapid, structured, and treatment-oriented protocol. The most useful acute MRI protocol includes DWI/ADC, FLAIR, GRE or SWI, and MRA, with perfusion imaging, ASL, and vessel wall imaging added according to clinical need.*

Keywords: Magnetic Resonance Imaging; Stroke; Ischemic Stroke; Cerebral Hemorrhage; Diffusion Magnetic Resonance Imaging; Susceptibility Weighted Imaging; Magnetic Resonance Angiography; Perfusion Imaging

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Introduction

Stroke is a heterogeneous neurological emergency caused by either interruption of cerebral blood flow or bleeding within the intracranial compartment. The two major pathological categories are ischemic stroke and haemorrhagic stroke. Ischemic stroke results from arterial occlusion or critically reduced perfusion, leading to cytotoxic edema, infarction, and later tissue necrosis. Haemorrhagic stroke results from rupture of cerebral vessels with bleeding into the brain parenchyma, ventricular system, subarachnoid space, or other intracranial compartments. These entities may present with overlapping clinical features, including acute weakness, speech disturbance, facial deviation, visual field loss, altered consciousness, headache, vertigo, seizures, or ataxia. Clinical assessment alone is therefore insufficient to distinguish ischemic from haemorrhagic stroke with adequate reliability.

The global burden of stroke remains substantial. Recent Global Burden of Disease analyses continue to identify stroke as a major cause of mortality and disability worldwide, with ischemic stroke representing the majority of incident and prevalent stroke cases (1,2). Intracerebral haemorrhage is less frequent than ischemic stroke but is associated with higher early case fatality, more severe disability, and greater need for intensive management (3). This epidemiological difference explains why emergency imaging protocols must be highly sensitive for ischemia while also reliably excluding haemorrhage before reperfusion therapies are considered.

Neuroimaging is central to modern stroke care. The first imaging question is whether intracranial haemorrhage is present. The second is whether there is acute ischemic injury, large-vessel occlusion, tissue-at-risk, or a stroke mimic. The third is whether imaging suggests a specific etiology, such as cardioembolism, intracranial atherosclerosis, small-vessel disease, arterial dissection, vasculitis, cerebral amyloid angiopathy, venous thrombosis, vascular malformation, or tumour-related haemorrhage. MRI is particularly valuable because it can answer several of these questions within a single examination by combining tissue, vascular, haemodynamic, and blood-product information (4,5).

Computed tomography remains widely used as the first-line emergency imaging modality because it is fast, widely available, and excellent for detecting acute intracranial haemorrhage. However, MRI provides superior tissue characterization and is more sensitive for early ischemia, small infarcts, posterior fossa infarction, microbleeds, superficial siderosis, haemorrhagic transformation, and stroke mimics. MRI is especially useful in wake-up stroke, unknown-onset stroke, posterior

circulation stroke, young patients with suspected dissection or vasculitis, suspected venous thrombosis, and cases where CT findings are equivocal.

Pathophysiological Basis of MRI Findings

The MRI appearance of ischemic stroke is primarily determined by the evolution of cytotoxic edema, vasogenic edema, tissue necrosis, and blood-brain barrier disruption. In acute ischemia, arterial occlusion causes failure of oxidative metabolism and depletion of adenosine triphosphate. Sodium-potassium pump failure leads to intracellular water accumulation and restricted diffusion. This process is detected on diffusion-weighted imaging as high signal intensity with corresponding low signal intensity on the apparent diffusion coefficient map (6). This DWI-high/ADC-low pattern is the most important MRI signature of acute ischemic infarction. Over time, ischemic tissue evolves. FLAIR and T2 signal become more conspicuous as edema develops. ADC values gradually rise and may pseudonormalize during the subacute period. Contrast enhancement may occur because of blood-brain barrier disruption, and chronic infarction eventually appears as gliosis, encephalomalacia, volume loss, and ex-vacuo ventricular dilatation.

Haemorrhagic stroke follows a different biochemical trajectory. The MRI appearance depends on the stage of hemoglobin degradation. Hyperacute haemorrhage contains oxyhemoglobin, acute haemorrhage contains deoxyhemoglobin, early subacute haemorrhage contains intracellular methemoglobin, late subacute haemorrhage contains extracellular methemoglobin, and chronic haemorrhage leaves hemosiderin and ferritin. These blood products create characteristic signal changes on T1, T2, GRE, and SWI sequences. Susceptibility-sensitive sequences are therefore essential for identifying blood products and chronic haemorrhagic markers (7,8).

This temporal evolution is clinically important because haemorrhage is not always uniformly dark on SWI. Hyperacute and late-subacute haemorrhage may show mixed or high central signal, while acute and chronic blood products usually show prominent susceptibility-related low signal. Therefore, accurate differentiation between ischemic and haemorrhagic lesions requires interpretation of multiple MRI sequences rather than reliance on one sequence alone. (Figure 1).

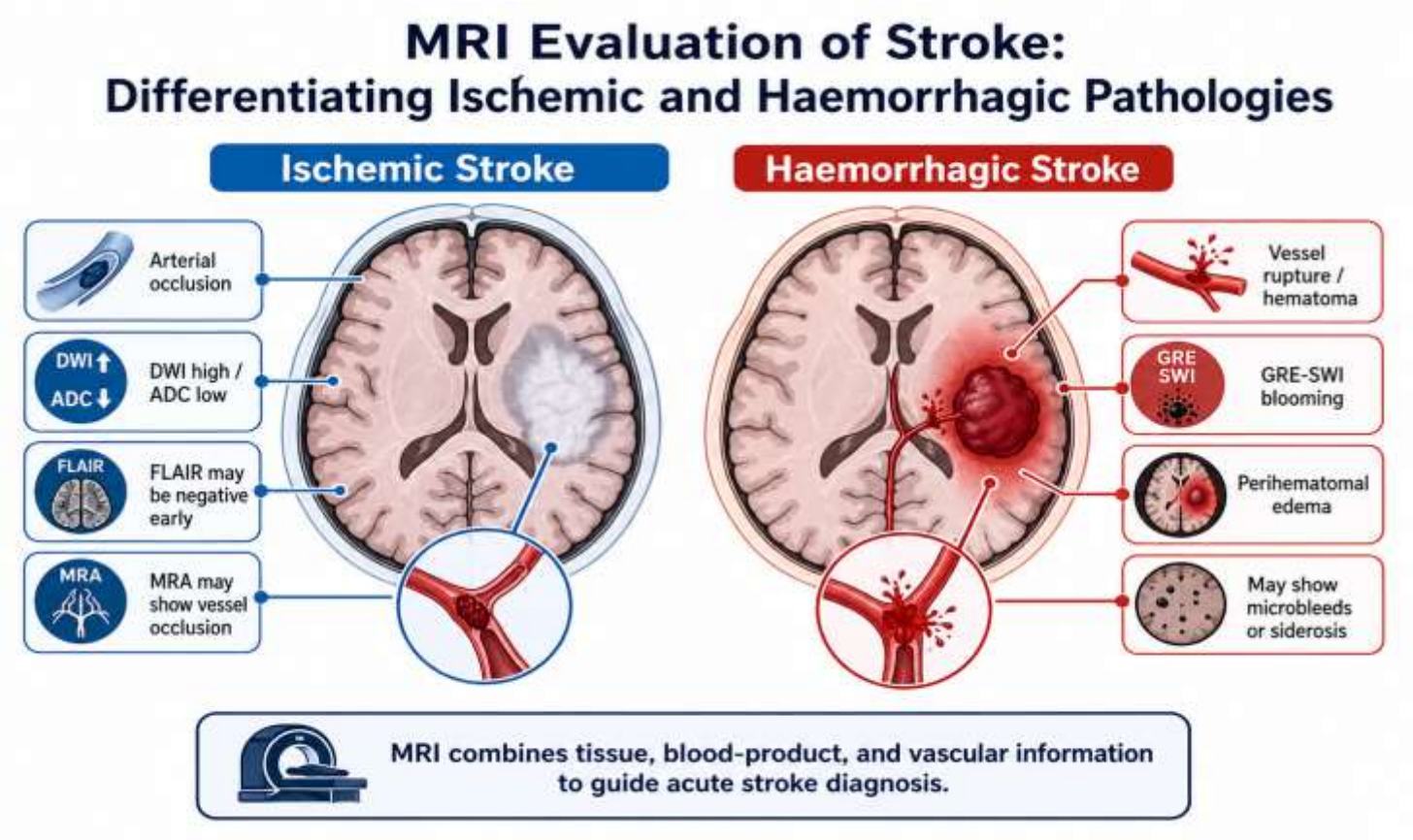

Figure 1: Schematic illustration of ischemic versus haemorrhagic stroke pathophysiology

Core MRI Sequences in Stroke Evaluation

MRI evaluation of stroke depends on a multiparametric protocol. Each sequence contributes different diagnostic information. DWI and ADC identify acute ischemia, FLAIR helps estimate lesion age and edema, GRE and SWI detect blood products, MRA identifies vascular occlusion or stenosis, perfusion imaging estimates tissue-at-risk, ASL provides non-contrast perfusion assessment, and vessel wall MRI improves etiological classification in selected patients.

Table 1: MRI sequences used for differentiating ischemic and haemorrhagic stroke

MRI sequence	Role in ischemic stroke	Role in haemorrhagic stroke	Key diagnostic value
DWI	Detects acute cytotoxic edema within minutes	May show perihematomal or remote ischemic lesions	Most sensitive marker of acute infarction
ADC map	Confirms true diffusion restriction	Helps distinguish restriction from T2 shine-through	Essential companion to DWI

FLAIR	Shows edema, lesion age, and DWI-FLAIR mismatch	Shows perihematomal edema and subarachnoid/intraventricular signal	Useful for timing and lesion conspicuity
GRE T2*	Detects haemorrhagic transformation and thrombus susceptibility	Detects acute and chronic blood products	Rapid susceptibility-sensitive sequence
SWI	Shows susceptibility vessel sign, venous congestion, and microbleeds	Highly sensitive for blood products, microbleeds, and siderosis	Best sequence for subtle haemorrhagic markers
TOF-MRA	Detects large-vessel occlusion and stenosis	May detect vascular malformation or aneurysmal cause	Non-contrast arterial assessment
Perfusion MRI	Identifies tissue-at-risk and perfusion mismatch	May show perihematomal perfusion changes	Supports treatment selection
ASL	Non-contrast perfusion and collateral-flow assessment	May show hyperperfusion or delayed transit	Useful when gadolinium is avoided
Vessel wall MRI	Detects plaque, dissection, and vasculitis	Evaluates underlying vasculopathy	Adds etiological information

A practical acute stroke MRI protocol should be short and treatment-oriented. Delayed acquisition may reduce the benefit of MRI in hyperacute stroke. Minimal imaging requirements for acute stroke emphasize rapid acquisition and direct relevance to treatment decisions (9). Newer abbreviated MRI protocols have shown promising diagnostic performance, including one-minute and 2.5-minute multi-contrast protocols for acute ischemic stroke (10,11). Deep-learning-based MRI reconstruction has also been investigated to shorten acquisition time while preserving diagnostic utility (12).

Diffusion-Weighted Imaging and ADC Mapping

DWI is the most important MRI sequence for detecting acute ischemic stroke. It is highly sensitive to restricted water motion caused by cytotoxic edema. In acute infarction, the lesion appears hyperintense on DWI and hypointense on ADC. The ADC map is essential because DWI hyperintensity alone may reflect T2 shine-through rather than true diffusion restriction.

DWI can detect ischemic injury within minutes and is particularly valuable in small cortical infarcts, lacunar infarcts, embolic infarcts, and posterior circulation strokes. It is more sensitive than CT for early ischemic injury, especially in the posterior fossa where CT artifacts may obscure brainstem and cerebellar infarcts. However, a normal DWI does not completely exclude ischemic stroke. A recent systematic review and meta-analysis reported that DWI-negative ischemic stroke may occur, particularly in minor stroke and posterior circulation events (13). This limitation is important because overreliance on a negative DWI may delay diagnosis in clinically suspicious cases.

ADC values also help determine lesion timing. ADC is typically reduced in acute infarction, rises during the subacute stage, and becomes elevated in chronic infarction because of tissue loss and gliosis. ADC pseudonormalization during the subacute period can complicate interpretation, particularly when the clinical history is unclear. Therefore, ADC must be interpreted along with DWI, FLAIR, vascular imaging, and clinical onset time.

FLAIR Imaging and DWI-FLAIR Mismatch

FLAIR imaging suppresses cerebrospinal fluid signal and improves visualization of cortical, periventricular, and subarachnoid abnormalities. In acute ischemic stroke, FLAIR may remain normal during the earliest hours, whereas DWI is already positive. This mismatch, known as DWI-FLAIR mismatch, can help estimate lesion age in patients with unknown onset or wake-up stroke.

The European Stroke Organisation guideline supports use of MRI-based DWI-FLAIR mismatch in selected patients with wake-up or unknown-onset ischemic stroke when considering intravenous thrombolysis and when mechanical thrombectomy is not planned (14). Studies evaluating FLAIR vascular hyperintensity-DWI mismatch and DWI-FLAIR mismatch have also suggested that these markers may provide prognostic information in anterior circulation large-vessel occlusion (15). Deep learning models have also been investigated for stroke onset time prediction using DWI-FLAIR mismatch concepts (16).

In haemorrhagic stroke, FLAIR helps demonstrate surrounding edema, intraventricular extension, subarachnoid signal abnormality, and chronic tissue injury. Although FLAIR is less specific than SWI or GRE for blood products, it provides important anatomical information about lesion extent, mass effect, edema, and associated parenchymal changes.

GRE and Susceptibility-Weighted Imaging

GRE T2* and SWI are central to the MRI evaluation of haemorrhage. These sequences detect magnetic susceptibility effects caused by deoxyhemoglobin, methemoglobin, ferritin, hemosiderin, and other paramagnetic substances. SWI is generally more sensitive than GRE for microbleeds, superficial siderosis, small haemorrhagic foci, venous abnormalities, and susceptibility vessel signs.

In ischemic stroke, SWI can identify the susceptibility vessel sign, which represents thrombus within an occluded artery. Prominent venous signs may reflect increased oxygen extraction in hypoperfused tissue and have been associated with poor functional outcome in acute ischemic stroke (17). Susceptibility vessel sign characteristics may also provide information about thrombus burden and possible stroke mechanism (18).

In haemorrhagic stroke, GRE and SWI demonstrate acute and chronic blood products through blooming artifacts and low-signal foci. Microbleed distribution may suggest underlying small-vessel pathology. Deep microbleeds are commonly associated with hypertensive arteriopathy, whereas lobar microbleeds and cortical superficial siderosis raise suspicion for cerebral amyloid angiopathy. Importantly, not all haemorrhage appears black on SWI, and the signal depends on hemoglobin stage and lesion timing (8).

Magnetic Resonance Angiography

MRA provides non-invasive evaluation of arterial patency. Time-of-flight MRA is widely used in acute stroke MRI because it can identify intracranial large-vessel occlusion, high-grade stenosis, and flow limitation without contrast. Contrast-enhanced MRA can provide additional information about extracranial carotid and vertebral arteries, tandem lesions, and aortic arch anatomy.

In ischemic stroke, MRA helps identify large-vessel occlusion and supports endovascular thrombectomy decisions. It may also identify intracranial atherosclerosis, embolic occlusion, dissection, and collateral-flow patterns. In haemorrhagic stroke, MRA may reveal secondary vascular causes such as aneurysm, arteriovenous malformation, dural arteriovenous fistula, or vascular malformation. However, CT angiography or digital subtraction angiography may still be required when vascular malformation is strongly suspected or when MRI findings are inconclusive.

Slow flow can mimic occlusion on TOF-MRA, and small distal vessel occlusions may be missed. Therefore, MRA findings should be interpreted with DWI lesion pattern, perfusion imaging, clinical deficit, and, when required, CT angiography or catheter angiography.

Perfusion MRI and Arterial Spin Labeling

Perfusion imaging estimates haemodynamic status and helps distinguish infarct core from tissue-at-risk. Contrast-based perfusion MRI evaluates cerebral blood flow, cerebral blood volume, mean transit time, time-to-maximum, and related parameters. In acute ischemic stroke, perfusion-diffusion mismatch may suggest salvageable tissue that has not yet undergone irreversible infarction.

Arterial spin labeling is a non-contrast perfusion technique that uses magnetically labeled arterial blood water as an endogenous tracer. It may be useful in patients with renal impairment, contrast allergy, or need for repeated follow-up imaging. ASL can demonstrate hypoperfusion, delayed arterial transit, collateral flow, and hyperperfusion after recanalization (22,23).

In haemorrhagic stroke, perfusion methods are not usually required for first-line diagnosis but may provide information about perihematomal physiology or post-treatment haemodynamic changes. ASL has also been studied for evaluating haemorrhagic transformation after endovascular recanalization in subacute ischemic stroke (21). These advanced techniques should be used selectively and should not delay urgent treatment decisions.

Vessel Wall MRI

High-resolution vessel wall MRI provides information beyond the arterial lumen by directly evaluating the vessel wall. It can identify intracranial atherosclerotic plaque, plaque enhancement, intraplaque haemorrhage, dissection, intramural hematoma, vasculitis, and reversible cerebral vasoconstriction syndrome.

In young adults with ischemic stroke or transient ischemic attack, high-resolution vessel wall imaging can improve etiological classification and reduce the proportion of cases labelled as undetermined (24). Vessel wall MRI is also increasingly used to evaluate intracranial arterial diseases and characterize plaque vulnerability (25). In symptomatic intracranial atherosclerotic steno-occlusive disease, vessel wall imaging characteristics may be associated with recurrent stroke risk (26). In suspected internal carotid artery dissection, high-resolution vessel wall MRI can directly demonstrate wall abnormalities and intramural hematoma (27).

Vessel wall MRI is usually not part of the first emergency sequence set unless dissection, vasculitis, or unusual vasculopathy is strongly suspected. It is often most useful after initial stabilization for etiological classification and secondary prevention planning.

Temporal MRI Evolution of Ischemic Stroke

The MRI appearance of ischemic stroke changes over time. In the hyperacute stage, DWI becomes positive early, while FLAIR may still be normal. ADC is low because of cytotoxic edema. During the acute stage, FLAIR and T2 signal become increasingly visible as edema develops. In the subacute stage, ADC values rise and may pseudonormalize, while DWI may remain bright due to T2 shine-through. In the chronic stage, infarction evolves into gliosis, encephalomalacia, volume loss, and increased ADC.

Table 2: Temporal MRI evolution of ischemic stroke

Stage	Approximate timing	DWI/ADC pattern	FLAIR/T2 pattern	Interpretation
Hyperacute	Minutes to 6 hours	DWI high, ADC low	May be normal	Early ischemia
Acute	6 hours to 7 days	DWI high, ADC low	FLAIR/T2 positive	Established acute infarct
Early subacute	7 to 14 days	DWI may remain high; ADC rises	Edema and FLAIR signal persist	Evolving infarct
Late subacute	2 to 3 weeks	ADC pseudonormalization possible	Enhancement may appear	Timing may be difficult
Chronic	More than 3 weeks	DWI usually normal or shine-through	Gliosis, encephalomalacia, volume loss	Old infarct

Temporal MRI Evolution of Haemorrhagic Stroke

The MRI signal of haemorrhage depends on the biochemical stage of hemoglobin. Hyperacute blood may be difficult to interpret on susceptibility imaging because oxyhemoglobin is relatively diamagnetic. Acute blood containing deoxyhemoglobin usually shows prominent low signal and blooming on GRE/SWI. Early subacute blood with intracellular methemoglobin becomes T1 hyperintense but may remain T2 hypointense. Late subacute blood with extracellular methemoglobin is usually hyperintense on both T1 and T2. Chronic haemorrhage leaves hemosiderin and ferritin, producing persistent susceptibility hypointensity.

Table 3: Temporal MRI evolution of intracerebral haemorrhage

Stage	Hemoglobin state	T1 signal	T2 signal	GRE/SWI appearance	Diagnostic implication
Hyperacute	Oxyhemoglobin	Iso/hypointense	Hyperintense	May be subtle or mixed	Very early blood may not be uniformly dark
Acute	Deoxyhemoglobin	Iso/hypointense	Hypointense	Marked blooming	Acute haemorrhage
Early subacute	Intracellular methemoglobin	Hyperintense	Hypointense	Blooming persists	Subacute blood
Late subacute	Extracellular methemoglobin	Hyperintense	Hyperintense	Mixed; rim may remain dark	Resolving hematoma
Chronic	Hemosiderin/ferritin	Variable	Hypointense rim	Persistent dark rim/foci	Old haemorrhage or microbleeds

Differentiating Ischemic and Haemorrhagic Stroke on MRI

The most reliable distinction between ischemic and haemorrhagic stroke is based on integrated pattern recognition. Ischemic stroke usually shows vascular-territory diffusion restriction, reduced ADC, possible vessel occlusion, and sometimes perfusion mismatch. Haemorrhagic stroke usually shows hematoma-centered signal abnormality, susceptibility blooming, mass effect, surrounding edema, and stage-dependent blood-product signal.

Table 4: MRI features differentiating ischemic and haemorrhagic stroke

Feature	Ischemic stroke	Haemorrhagic stroke
Dominant pattern	Vascular territory infarction	Hematoma-centered lesion
DWI/ADC	DWI high with ADC low in acute phase	Variable; may show adjacent or remote ischemia
FLAIR	May be negative early and positive later	Shows edema, mass effect, ventricular/subarachnoid extension
GRE/SWI	Thrombus sign, prominent veins, petechial haemorrhagic transformation	Blood products, blooming, microbleeds, siderosis

MRA	Occlusion, stenosis, embolus, dissection	May show aneurysm, AVM, or vascular malformation
Perfusion	Hypoperfusion, mismatch, penumbra	Variable perihematomal perfusion
Evolution	Cytotoxic edema to gliosis	Hemoglobin degradation to hemosiderin
Treatment implication	Reperfusion and antithrombotic decisions	Avoid thrombolysis; reverse anticoagulation; control bleeding risk

Haemorrhagic transformation is an important mixed pattern. It occurs when ischemic tissue bleeds, often after reperfusion, thrombolysis, anticoagulation, large infarct volume, cardioembolism, or severe blood-brain barrier disruption. MRI shows both diffusion restriction and susceptibility abnormality. Petechial haemorrhage may be subtle on CT but visible on SWI. Larger parenchymal hematoma shows susceptibility blooming, edema, and mass effect.

Diffusion-positive lesions may also occur after primary intracerebral haemorrhage. A study evaluating diffusion-weighted lesions after intracerebral haemorrhage reported that such lesions can be seen in a subset of patients and may occur both ipsilateral and contralateral to the hematoma (19). This finding highlights the need for careful interpretation rather than assuming that all DWI lesions represent the primary stroke mechanism.

MRI in Haemorrhagic Transformation

Haemorrhagic transformation is a clinically important complication of ischemic stroke. It may range from small petechial haemorrhage to large parenchymal hematoma with mass effect. MRI is highly sensitive for detecting this complication because SWI and GRE can identify small blood products that may not be visible on CT.

After thrombectomy, MRI may detect intracranial haemorrhage not seen on CT, although the clinical significance of MRI-only haemorrhage requires careful interpretation (20). ASL has also been investigated for evaluating haemorrhagic transformation after endovascular recanalization in subacute ischemic stroke (21). In practice, haemorrhagic transformation should be described according to location, extent, mass effect, and relationship to the infarcted territory.

MRI in Stroke Mimics

Stroke mimics are non-vascular disorders that present with acute neurological deficits. These include seizure-related changes, migraine aura, demyelination, encephalitis, abscess, tumour, hypoglycemia, posterior reversible encephalopathy syndrome, cerebral venous thrombosis, and functional neurological disorders. MRI is valuable because many mimics have characteristic patterns that differ from arterial-territory infarction.

Seizure-related changes may show cortical DWI hyperintensity and FLAIR abnormality but often do not respect a vascular territory and may show hyperperfusion. Tumours may demonstrate mass-like enhancement and vasogenic edema. Demyelinating lesions may be ovoid, periventricular, juxtacortical, or infratentorial. Cerebral venous thrombosis may produce haemorrhagic venous infarction crossing arterial boundaries and requires MR venography or CT venography.

Recognition of stroke mimics is important because inappropriate thrombolysis or delayed treatment of the true underlying condition can worsen outcomes.

MRI Protocol for Acute Stroke

A practical emergency MRI protocol should be brief, reproducible, and directly linked to treatment decisions. The core protocol should include DWI/ADC, FLAIR, GRE or SWI, and MRA. Perfusion MRI or ASL may be added when tissue-at-risk assessment is required. Vessel wall MRI should be reserved for selected cases where etiological clarification is necessary.

Table 5: Suggested emergency MRI protocol for suspected stroke

Protocol component	Recommended sequence	Purpose
Tissue injury	DWI and ADC	Detect acute ischemia
Lesion timing	FLAIR	Estimate lesion age and edema
Blood products	GRE or SWI	Detect haemorrhage, microbleeds, thrombus
Arterial status	TOF-MRA head	Detect large-vessel occlusion
Cervical vessels	Neck MRA	Detect carotid/vertebral stenosis or dissection
Tissue at risk	Perfusion MRI or ASL	Identify mismatch or haemodynamic compromise
Etiology	Vessel wall MRI	Assess plaque, dissection, vasculitis
Follow-up	DWI/ADC, FLAIR, SWI	Assess evolution and complications

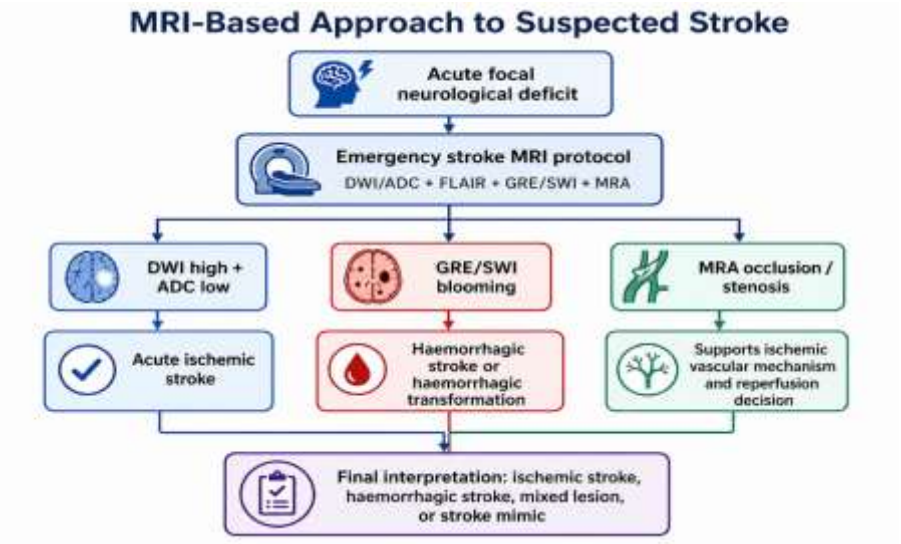

Figure 3: Suggested MRI-based stroke differentiation pathway

Diagnostic Pitfalls

Several pitfalls may lead to incorrect interpretation. First, DWI-negative ischemic stroke can occur, especially in minor stroke and posterior circulation infarction (13). Therefore, persistent clinical suspicion should prompt repeat imaging or additional vascular evaluation.

Second, T2 shine-through can mimic restricted diffusion. ADC maps must always be reviewed. True acute ischemia requires DWI hyperintensity with corresponding ADC hypointensity.

Third, haemorrhage is not always uniformly dark on SWI. Hyperacute and late-subacute haemorrhage may show variable signal, and interpretation requires knowledge of hemoglobin evolution (8).

Fourth, calcification, air, iron deposition, and vascular structures may mimic blood products on susceptibility imaging. CT correlation or phase images may be helpful in uncertain cases.

Fifth, slow flow on TOF-MRA may mimic occlusion. Contrast-enhanced MRA, CTA, or catheter angiography may be needed when findings are unclear.

Sixth, haemorrhagic transformation may be mistaken for primary haemorrhage unless the arterial-territory infarct pattern is recognized.

Clinical Impact of MRI-Based Differentiation

MRI affects acute treatment, prognosis, and secondary prevention. In ischemic stroke, MRI confirms infarct core, identifies large-vessel occlusion, estimates tissue-at-risk, and supports treatment decisions regarding thrombolysis and thrombectomy. In unknown-onset stroke, DWI-FLAIR mismatch may help identify selected patients eligible for reperfusion therapy (14).

In haemorrhagic stroke, MRI helps identify underlying causes and chronic bleeding markers. Microbleeds and superficial siderosis may influence antithrombotic decisions. Detection of vascular malformation, tumour, venous thrombosis, dissection, or vasculitis may substantially change management.

Advanced MRI is increasingly relevant. ASL provides non-contrast perfusion information (22,23). Vessel wall MRI can identify plaque, dissection, vasculitis, and intracranial arterial disease (24-27). Artificial intelligence has also been investigated for MRI-based stroke detection, although external validation and workflow integration remain important limitations (28). Imaging-selection strategies for endovascular treatment continue to evolve, and MRI may be feasible in selected early and extended time-window pathways when it does not delay treatment (29,30).

Structured MRI Reporting

A structured MRI report improves communication between radiologists, neurologists, emergency physicians, and neurosurgeons. The report should directly answer the acute clinical question and highlight treatment-relevant findings.

Table 6: Suggested structured MRI report for suspected stroke

Report section	Required details
Clinical information	Onset time, last known well, neurological deficit, treatment question
Technique	MRI sequences performed, contrast use, MRA/perfusion/vessel wall imaging
Acute ischemia	Presence, location, vascular territory, DWI/ADC pattern, lesion burden
Haemorrhage	Primary haemorrhage, haemorrhagic transformation, microbleeds, siderosis
Mass effect	Edema, midline shift, herniation, ventricular compression
Vascular findings	Occlusion, stenosis, dissection, aneurysm, AVM, collateral information
Perfusion findings	Core-penumbra mismatch, hypoperfusion, hyperperfusion
Etiological clues	Small-vessel disease, amyloid pattern, plaque, vessel wall enhancement
Impression	Final diagnosis, stage, treatment relevance, urgent findings

The impression should be concise and treatment-oriented. For example, it should state whether there is acute ischemia, whether haemorrhage is present, whether large-vessel occlusion exists, whether haemorrhagic transformation is present, and whether imaging supports immediate reperfusion or haemorrhage-specific management.

Future Directions

MRI stroke imaging is moving toward faster acquisition, automated interpretation, quantitative perfusion, artificial intelligence, and integrated tissue-based triage. Abbreviated protocols may reduce scan time while preserving diagnostic value (10-12). These methods may make MRI-first stroke triage more feasible in selected centers.

Artificial intelligence has shown promising performance for MRI-based stroke detection, but further validation is needed across scanner types, populations, stroke subtypes, haemorrhagic lesions, and workflow settings (28). Future AI systems may integrate DWI, ADC, FLAIR, SWI, MRA, perfusion, and clinical variables to generate treatment-oriented decision support.

Advanced vessel wall imaging and ASL may also improve etiological diagnosis and recurrence-risk stratification. These techniques may be particularly useful in young stroke, cryptogenic stroke, intracranial atherosclerosis, dissection, vasculitis, and recurrent stroke despite standard therapy.

Conclusion

MRI provides a comprehensive and highly sensitive approach to differentiating ischemic and haemorrhagic stroke. Acute ischemic stroke is best identified by DWI hyperintensity with corresponding ADC reduction, vascular-territory distribution, and supportive MRA or perfusion findings. Haemorrhagic stroke is best identified by GRE/SWI susceptibility patterns, hematoma-centered morphology, mass effect, edema, microbleeds, and stage-dependent hemoglobin signal evolution. The distinction is most reliable when MRI is interpreted as a multiparametric examination rather than through any single sequence. A rapid emergency MRI protocol consisting of DWI/ADC, FLAIR, GRE or SWI, and MRA can differentiate most ischemic and haemorrhagic lesions, while perfusion imaging, ASL, and vessel wall MRI provide additional treatment and etiological information. MRI should be applied in a structured and time-conscious manner so that diagnostic detail supports rather than delays acute stroke management.

Declarations

Consent for publication

Approved

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Conflict of interest

The authors declared the absence of a conflict of interest.

Author Contribution

RR, MM and MA contributed to the conception and design of the review, developed the literature search strategy, performed literature screening, extracted relevant information from included studies, interpreted the available evidence, and drafted the initial manuscript. **FI, RN and MI** assisted in literature searching, study selection, data organization,

reference management, manuscript formatting, proofreading, and revision of the manuscript. **AK** supervised the overall review process, provided methodological and intellectual guidance, critically reviewed the manuscript for important academic content, coordinated among all authors, finalized the manuscript, and approved the final version for submission.

All authors reviewed the results and approved the final version of the manuscript. They are also accountable for the integrity of the study.

References

1. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973-1003. https://doi.org/10.1016/S1474-4422(24)00369-7
2. Li XY, Kong XM, Yang CH, Cheng ZF, Lv JJ, Guo H, et al. Global, regional, and national burden of ischemic stroke, 1990–2021: an analysis of data from the Global Burden of Disease Study 2021. EClinicalMedicine. 2024;75:102758. https://doi.org/10.1016/j.eclinm.2024.102758
3. Wang S, Zou XL, Wu LX, Zhou HF, Xiao L, Yao T, et al. Epidemiology of intracerebral hemorrhage: a systematic review and meta-analysis. Front Neurol. 2022;13:915813. https://doi.org/10.3389/fneur.2022.915813
4. Frias P, Khangura RS, Varjavand B, Alexander MD. Imaging in acute ischaemic stroke: assessing findings in light of evolving therapies. Br J Radiol. 2024;97(1158):1078-87. https://doi.org/10.1093/bjr/tqae050
5. Colasurdo M, Chen H, Gandhi D. MR imaging techniques for acute ischemic stroke and delayed cerebral ischemia following subarachnoid hemorrhage. Neuroimaging Clin N Am. 2024;34(2):203-14. https://doi.org/10.1016/j.nic.2023.12.002
6. Nagaraja N. Diffusion weighted imaging in acute ischemic stroke: a review of its interpretation pitfalls and advanced diffusion imaging application. J Neurol Sci. 2021;425:117435. https://doi.org/10.1016/j.jns.2021.117435
7. Greenberg SM, Ziai WC, Cordonnier C, Dowlatshahi D, Francis B, Goldstein JN, et al. 2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: a guideline from the American Heart Association/American Stroke Association. Stroke. 2022;53(7):e282-e361. https://doi.org/10.1161/STR.0000000000000407
8. Weerink LBM, Appelman APA, Kloet RW, van der Hoorn A. Susceptibility-weighted imaging in intracranial hemorrhage: not all bleeds are black. Br J Radiol. 2023;96(1148):20220304. https://doi.org/10.1259/bjr.20220304
9. Kawano H, Hirano T. Minimal imaging requirements. J Neuroendovasc Ther. 2023;17(11):243-56. https://doi.org/10.5797/jnet.ra.2023-0045
10. Kits A, De Luca F, Kolloch J, Müller S, Mazya MV, Skare S, et al. One-minute multi-contrast echo planar brain MRI in ischemic stroke: a retrospective observational study of diagnostic performance. J Magn Reson Imaging. 2021;54(4):1088-95. https://doi.org/10.1002/jmri.27641
11. Kits A, Al-Saadi J, De Luca F, Janzon F, Mazya MV, Lundberg J, et al. 2.5-minute fast brain MRI with multiple contrasts in acute ischemic stroke. Neuroradiology. 2024;66(5):737-47. https://doi.org/10.1007/s00234-024-03331-0
12. Altmann S, Grauhan NF, Brockstedt L, Kondova M, Schmidtmann I, Paul R, et al. Ultrafast brain MRI with deep learning reconstruction for suspected acute ischemic stroke. Radiology. 2024;310(2):e231938. https://doi.org/10.1148/radiol.231938
13. Alkhiri A, Alturki F, Alansari NM, Almaghrabi AA, Alghamdi BA, Alamri AF, et al. Prognosis and distribution of ischemic stroke with negative diffusion-weighted imaging: a systematic review and meta-analysis. Front Neurol. 2024;15:1376439. https://doi.org/10.3389/fneur.2024.1376439
14. Berge E, Whiteley W, Audebert H, De Marchis GM, Fonseca AC, Padiglioni C, et al. European Stroke Organisation guidelines on intravenous thrombolysis for acute ischaemic stroke. Eur Stroke J. 2021;6(1):I-LXII. https://doi.org/10.1177/2396987321989865
15. Kaewumporn U, Songsaeng D, Kunapinun A, Chaisinanunkul N. FLAIR vascular hyperintensity-DWI mismatch and DWI-FLAIR mismatch ASPECTS for prediction of good outcome after recanalization in anterior circulation stroke: multicenter observational study. Eur J Radiol. 2023;163:110837. https://doi.org/10.1016/j.ejrad.2023.110837
16. Akay EMZ, Rieger J, Schöttler R, Behland J, Schymczyk R, Khalil AA, et al. A deep learning analysis of stroke onset time prediction and comparison to DWI-FLAIR mismatch. Neuroimage Clin. 2023;40:103544. https://doi.org/10.1016/j.nicl.2023.103544
17. Xiang W, Liang Z, Zhang M, Wei H, Sun Z, Lv Y, et al. Prognostic value of susceptibility-weighted imaging of prominent veins in acute ischemic stroke: a systematic review and meta-analysis. Front Neurol. 2022;13:1052035. https://doi.org/10.3389/fneur.2022.1052035
18. Li J, Zhan Z, Zhang Z, Wang W, Xu K, Sun W. Susceptibility vessel sign in patients with subacute ischemic stroke. Quant Imaging Med Surg. 2024;14(6):3914-22. https://doi.org/10.21037/qims-23-1797
19. Wiegertjes K, Voigt S, Jolink WMT, Koemans EA, Schreuder FHBM, van Walderveen MAA, et al. Diffusion-weighted lesions after intracerebral hemorrhage: associated MRI findings. Front Neurol. 2022;13:882070. https://doi.org/10.3389/fneur.2022.882070
20. Suzuki K, Katano T, Numao S, Nishi Y, Kutsuna A, Kanamaru T, et al. Clinical significance of intracranial hemorrhage after thrombectomy detected solely by magnetic resonance imaging and not by computed tomography. J Neurol Sci. 2024;460:122999. https://doi.org/10.1016/j.jns.2024.122999
21. Wu L, Liu Y, Zhu L, Li T, Wang L, Zhang Y, et al. MRI arterial spin labeling in evaluating hemorrhagic transformation following endovascular recanalization of subacute ischemic stroke. Front Neurosci. 2023;17:1105816. https://doi.org/10.3389/fnins.2023.1105816
22. Jaafar N, Alsop DC. Arterial spin labeling: key concepts and progress towards use as a clinical tool. Magn Reson Med Sci. 2024;23(3):352-66. https://doi.org/10.2463/mrms.rev.2024-0013
23. Togao O, Obara M, Yamashita K, Kikuchi K, Wada T, Murazaki H, et al. Arterial spin labeling-based MR angiography for cerebrovascular diseases: principles and clinical applications. J Magn Reson Imaging. 2024;60(4):1305-24. https://doi.org/10.1002/jmri.29119
24. He WW, Lu SS, Ge S, Gu P, Shen ZZ, Wu FY, et al. Impact on etiology diagnosis by high-resolution vessel wall imaging in young adults with ischemic stroke or transient ischemic attack. Neuroradiology. 2023;65(6):1015-23. https://doi.org/10.1007/s00234-023-03131-y
25. Kang DW, Kim DY, Kim J, Baik SH, Jung C, Singh N, et al. Emerging concept of intracranial arterial diseases: the role of high resolution vessel wall MRI. J Stroke. 2024;26(1):26-40. https://doi.org/10.5853/jos.2023.02481
26. Yuan W, Liu X, Yan Z, Wu B, Lu B, Chen B, et al. Association between high-resolution magnetic resonance vessel wall imaging characteristics and recurrent stroke in patients with intracranial atherosclerotic steno-occlusive disease: a prospective multicenter study. Int J Stroke. 2024;19(5):569-76. https://doi.org/10.1177/17474930241228203
27. Xie S, Ran Y, Wang X, Zhang Y, Fu Q, Ren Y, et al. Diagnostic potential of routine brain MRI and high-resolution, multi-contrast vessel wall imaging in the detection of internal carotid artery dissection. Front Neurol. 2023;14:1165453. https://doi.org/10.3389/fneur.2023.1165453
28. Bojsen JA, Elhakim MT, Graumann O, Gaist D, Nielsen M, Harbo FSG, et al. Artificial intelligence for MRI stroke detection: a systematic review and meta-analysis. Insights Imaging. 2024;15(1):160. https://doi.org/10.1186/s13244-024-01723-7
29. Wu J, Peng Z, Zhang H. Impact of stroke imaging selection modality on endovascular thrombectomy outcomes in the early and extended time windows: a meta-analysis. Brain Behav. 2024;14(8):e3530. https://doi.org/10.1002/brb3.3530
30. Bankole NDA, Mujanovic A, Dokponou YCH, Provost C, Pasi M, Redjem H, et al. MRI versus CT before endovascular thrombectomy in the early time window: a systematic review and meta-analysis. J Neuroradiol. 2025;52(6):101384. https://doi.org/10.1016/j.neurad.2025.101384