

Susceptibility weighted imaging in acute stroke with co-morbid: Magnetic resonance imaging protocol revisited

Abstract

Introduction:

We aim to investigate penumbra mainly Diffusion weighted imaging- Susceptibility weighted imaging mismatch using Alberta Stroke Program Early Computed tomography Stroke Score scoring in patients with multiple co morbid.

Methods:

From January 2011 to December, 2017; 70 consecutive patients (50 men,20 women; mean age 64.5; range 45-82 years) with acute infarct on Diffusion weighted imaging(DWI) were selected for the study. Stroke protocol performed including DWI and susceptibility weighted imaging(SWI) on first day and repeat within three days. All initial MR images were interpreted by one Neuroradiologist with more than ten years blind to the clinical findings of each patient. The definition of an acute infarct area was high signal intensity on DWI with dark signal intensity on Apparent diffusion weighted imaging (ADC). The infarct extent was scored using the Alberta Stroke Program Early CT Score (ASPECTS) system. Infarct growth was defined as any new or larger lesion on the second DWI.

For correlation with infarct growth, the same topographic system was used to record the extent of the Prominent vessel sign(PVS) on SWI.

Spearman's rank correlation test was used to examine the correlations between PVS score and infarct growth score. Regression was computed, with $P < 0.05$ considered significant.

Results:

The MCA territory infarct was on the right side in 9 patients and on the left in 13. The mean ASPECTS score was 4.3 (range 0–9). PVS was detected in 15 patients (mean score 4.1, range 0–10).

The second MRI revealed no infarct growth in 9 patients and infarct growth in 13 (ASPECTS mean score 3.95, range 0–9; mean infarct growth score 7.4, range 0–10).

Of 7 patients without PVS on the first MRI, none had infarct growth on the second. Of 15 patients with PVS on the first MRI, 13 (87%) had infarct growth. The PVS score and infarct growth score were well correlated ($r = 0.86, P < 0.001$).

Conclusion:

PVS seen in infarcted territory is related to poor prognosis and this can be reliably used as a surrogate marker of oxygen extraction in penumbra.

SWI can predict tissue at risk and can be a replacement for perfusion scan in clinical scenerio of acute ischaemic infarct.

Keywords stroke, magnetic resonance imaging, diffusion magnetic resonance imaging

35

36 Introduction

37 The estimated annual incidence of stroke in Pakistan is 250/100,000 population which is
38 projected to an estimate of 350,000 new cases each year [1].

39 The role of imaging in triage of acute stroke is to rule out haemorrhage or ischemic infarction
40 and selection of ischaemic stroke patients for available reperfusion therapies [2]. The imaging
41 modality of choice for stroke triage should enable patient selection for thrombolytic therapy in
42 terms of both safety and efficacy.

43 Routine IV tissue plasminogen activator (IVTPA) treatment within the 3-hour window is still
44 typically administered according to the protocol of the 1995 National Institute of Neurological
45 Disorders and Stroke (NINDS) trial, which focused on exclusion of hemorrhage with unenhanced
46 CT [3].

47 However, infarct core and penumbra is desired for selection of reperfusion options as well as to
48 prognosticate [4]. Options for penumbra detection currently used and have several controversies
49 are CT or MR with perfusion. These were particularly advised when mechanical
50 thrombectomy is the plan [5].

51 The mismatch between PWI and DWI predicts a favorable response to thrombolysis after early
52 reperfusion, and so may be a surrogate for ischemic penumbra [6] but for predicting infarct
53 growth or clinical outcome is controversial and has been challenged [7].

54 In addition, Perfusion studies requires administration of contrast agent, which limits its
55 application in patients with renal insufficiency.

56 Susceptibility-weighted imaging (SWI) is a potential alternative for predicting infarct growth. In
57 the ischemic brain, the increased oxygen extraction fraction and slow flow contribute to a higher
58 level of deoxyhemoglobin and vein dilatation, which increases the conspicuity of vessels on
59 SWI [8].

60 Kaya, et al. [9] identified multiple hypointense vessels strictly in the ischemic territory during the
61 hyperacute phase of stroke on 3T and Haccke, et al. [10] on 1.5 T.

62 The region was larger than the lesion shown on DWI and correlated well with the final infarction
63 area after 72 hours.

64 SWI/DWI mismatch has also been recommended as a potential indicator of infarct growth in
65 some reports [11].

66 Using a similar approach, we aim to investigate pneumbra mainly DWI- SWI mismatch using
67 ASPECT scoring. To our knowledge no such study has been conducted so far on national level.

68 Materials & Methods

69 We created a data base from retrospective case material from our institution and then searched
70 the data base prospectively as a *hypothesis-driven scientific study*.

71 Study design was Cross-sectional analytical.

72 From January 2011 to December,2017; 70 consecutive patients(50 men,20 women; mean age
73 64.5; range 45-82 years) met the below mentioned criteria in our university hospital and were
74 selected for the study. The review was conducted in accordance with guidelines of the research
75 committee of our institution.

76 *Inclusion criteria:*

77 Acute infarct on DWI in MCA territory.

78 Stroke protocol performed including DWI and SWI on first day.

79 Repeat DWI with in three days.

80 *Exclusion criteria:*

81 Tissue plasminogen activator (TPA) given.

82 Haemorrhagic infarction on initial presentation.

83 Watershed infarcts/Posterior circulation infarcts

84 Sampling technique was Non-probability purposive

85 Imaging techniqueScanners: 1.5 T scanner (Magnetom Avanto; Siemens Medical Solutions,
86 Erlangen, Germany) and 3T (Titan,Toshiba)with a standard 12-channel head coil.

UNDER PEER REVIEW

87 Stroke protocol: After routine axial T2W Only DWI and SWI sequences were performed in first
88 encounter after triage.

89 For the DWI sequences, (with TR/TE = 3700/109 ms, b = 1000 s/mm², slice thickness = 5 mm,
90 slice number = 28, and matrix = 128x128) and generated ADC maps.

91 For the transverse 3-dimensional (3D) SWI sequences, TR/TE = 49/ 40 ms, flip angle = 15°,
92 slice thickness = 2 mm with 60 sections per slab, matrix = 224×256, 64 slices, and (integrated
93 parallel acquisition technique (iPAT) acceleration factor = 2. The phase, magnitude (mag),
94 minIP, and SWI images were uploaded and made available on a picture archiving and
95 communication (PACS) system (Rogan).

96 The total scan time for stroke protocol was 5-6 mins.

97 Follow up scan was performed within 3 days with axial T1W,T2W,Coronal FLAIR, DWI, SWI
98 AND Time Of flight MR angiogram of the circle of Willis.

99 All MRI images were interpreted by one Neuroradiologist with more than ten years of
100 experience and Neuroimaging fellow. All initial MR images were interpreted blind to the clinical
101 findings of each patient.

102 The definition of an acute infarct area was high signal intensity on DWI with dark signal
103 intensity on ADC.

104 The infarct extent was scored using the Alberta Stroke Program Early CT Score (ASPECTS)
105 system, a 10-point semiquantitative CT score system developed and tested as a reliable grading
106 system to assess the extent of ischemic change and to predict functional outcome in patients with
107 acute ischemic stroke [12].

108 This topographic system allots 1 point for each of 10 zones of the MCA territory. A score of 10
109 is normal while 0 indicates diffuse infarction [13].

110 The application of ASPECTS to DWI in stroke has been extended and contributes to outcome
111 prediction and quick risk assessment before thrombolytic therapy [14].

112 Infarct growth was defined as any new or larger lesion on the second DWI.

113 The infarct growth was scored from 10 (no growth) to 0 (growth in all 10 zones).

114 The PVS on SWI was defined as a local prominence of hypointense vessels with either increased
115 vessel number or diameter in the target area, when compared with the non-target area.

116 In this study, the target area was defined as the MCA territory of the infarct side.

117 For correlation with infarct growth, the same topographic system was used to record the extent of
118 the PVS.

119 The PVS of the insular cortical vessels was recorded as I, of the lower MCA-territory cortical or
120 medullary vessels (M1, M2, or M3), of the higher MCA-territory cortical or medullary vessels
121 (M4, M5, or M6), and of the thalamostriate vein (C, L, or IC) because this vein drains the
122 caudate nucleus, lentiform nucleus, and internal capsule.

123 After the two readers had reached a consensus, the extent of the PVS was scored from 10 (no
124 PVS) to 0 (PVS in M1, M2, M3, M4,M5, M6, I, C, L, or IC).

125 Statistical analysis used SPSS, version 17.0 (SPSS, Chicago, IL, USA). Mean and standard
126 deviation of PVS scores, DWI ASPECTS scores, and infarct growth scores were calculated.

127 Spearman's rank correlation test was used to examine the correlations between PVS score and
128 infarct growth score. Regression was computed, with $P < 0.05$ considered significant.

129 Results

130 The study included 12 women and 10 men, (mean age 67.1 years).

131 First MRI images were all acquired in the acute stage of stroke (mean 12 hours) and second
132 images within 3 days after stroke.

133 The MCA territory infarct was on the right side in 9 patients and on the left in 13. The mean
134 ASPECTS score was 4.3 (range 0–9). PVS was detected in 15 patients (mean score 4.1, range
135 0–10).

136 The second MRI revealed no infarct growth in 9 patients and infarct growth in 13 (ASPECTS
137 mean score 3.95, range 0–9; mean infarct growth score 7.4, range 0–10).

138 Of 7 patients without PVS on the first MRI, none had infarct growth on the second. Of 15
139 patients with PVS on the first MRI, 13 (87%) had infarct growth. The PVS score and infarct
140 growth score were well correlated ($r = 0.86, P < 0.001$).

141 Our results were consistent with those of recently published studies on pediatric arterial ischemic
142 stroke Polan, RM, et al [15] in which we restricted the analytic sample to adults, indicating
143 SWI/DWI mismatch is useful for predicting ischemic stroke progression and study by Chia-
144 YuenChen on adults Chia, Yuen, et al. [16].

145 Discussion

146 Our study showed that the PVS on SWI is a signature of salvageable ischemic tissue that will
147 become infarcted if blood perfusion cannot be established in time.

148 This finding is consistent with the results of previous studies Kesavadas C, et
149 al. J Neurool [17]. Kao HW et al. Euro Radiol [18], Huang P, et al. Neurool [19], Baik et al.
150 Cerebrovasc Dis [20], Yamashita E, et al. Acta Radiol [21].

151 The PVS had a positive predictive rate of 87% and a negative predictive rate of 100%.

152 PVS might reflect not only veins but also small arteries with deoxyhemoglobin blood in the
153 penumbra area. Consistent with previous SWI studies, our study of 22 patients showed PVS in
154 15, microbleed in 6, and intra-arterial thrombus in 9. A lower microbleed rate would be
155 expected, with parenchymal hemorrhage used as an exclusion criterion.

156 Only two patients (25%) with infarct growth in the lentiform nucleus, internal capsule, or
157 caudate nucleus had PVS, which can be explained by the admixed venous flow in the
158 thalamostriate vein, which drains not only these structures, but also the thalamus

159 Good spatial correlation between infarct growth and the extent of PVS was also observed. Of 57
160 zones of infarct growth, all 46 in the insula or M1–M6 zones of the MCA territory matched the
161 extent of PVS, consistent with previous reports that PVS on SWI can predict stroke evolution
162 and spatially correlate with DWI/PWI mismatch.

163 In Baik's study, clinical outcome improved with the apparent normalization of PVSs in veins
164 after successful recanalization. One case report [22] in the literature described that SWI iso- or
165 hyperintensity of the draining veins might suggest hyperperfusion, which contributed further to
166 an increased risk of developing post-ischemic malignant edema [23-25]. In our study, given that
167 the extent of PVS indicates the extent of penumbra, that patients with more extensive PVS can
168 be expected to have a larger volume of salvageable tissue to be rescued.

169 • Limitations:

UNDER PEER REVIEW

- 170 – small patient number.
- 171 – did not include performing PWI or arterial spin labeling
- 172 – Patients with the worst clinical outcomes or who died were not recruited
- 173 – Bias in interpreting the images was possible, because PVS in this study was defined by
174 observation and comparison rather than objective measurement of vessel number or diameter.
175 Using ASPECTS for PVS semiquantification is arguable [26].
- 176 – Quantitative susceptibility mapping is a development of SWI that utilizes phase data to
177 obtain information on local susceptibility [27-30]
- 178 – Future directions:
- 179 It may in future be possible to provide fully quantitative and noninvasive information on oxygen
180 metabolism

181 Conclusions

182 Venous congestion seen in infarcted territory is related to poor prognosis and this can be reliably
183 used as a surrogate marker of oxygen extraction in penumbra.

184 SWI can predict tissue at risk and can be a replacement for perfusion scan in clinical scenerio of
185 acute ischaemic infarct.

186 MRI stroke protocol can be a one stop shop with initial first day DWI-SWI sequences to detect
187 core and penumbra with multiple co-morbids and in settings were reperfusion is planned.

188 189 References

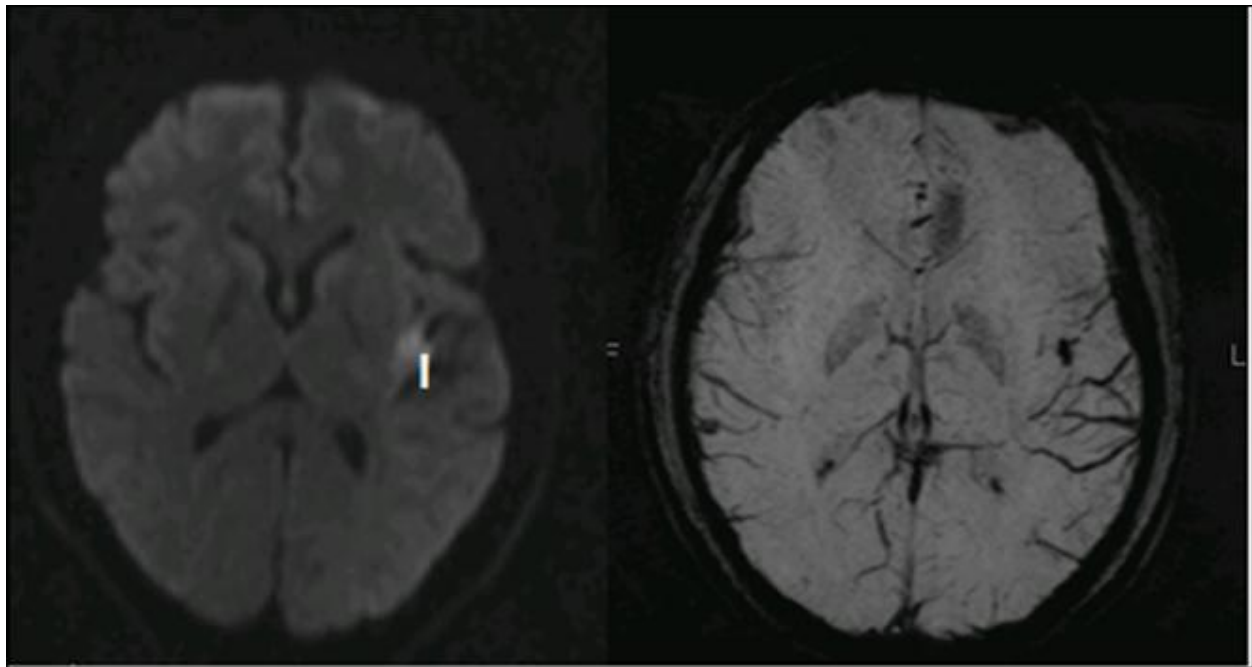
- 190 1. Bhojo A. Khealani¹, Bilal Hameed², Uzma U. Mapari³ Department of Medicine¹,
191 Aga Khan University Hospital, Karachi, Department of Medicine^{2,3}, University of
192 Alberta, Edmonton, Alberta, Canada. : [Stroke in Pakistan](#). J Pak Med Assoc .
193 2008, 58:400-403.
- 194 2. Max Wintermark, MD MAS, Associate Professor,¹ Pina Sanelli, MD MPH,
195 Associate Professor,² Gregory W. Albers, MD, Professor,³ Jacqueline Bello, MD,
196 FACR, Professor,⁴ Colin Derdeyn, MD, FACR, Professor,⁵ Steven W. Hetts, MD,
197 Associate Professor,⁶ Michele H. Johnson, MD, Associate Professor,⁷ Chelsea
198 Kidwell, MD, Professor,⁸ Michael H. Lev, MD FAHA FACR, Associate
199 Professor,⁹ David S. Liebeskind, MD FAHA FAAN, Neurology Director,¹⁰

- 200 Howard Rowley, MD, Professor,11 Pamela W. Schaefer, MD, Associate
 201 Director,12 Jeffrey L. Sunshine, MD, PhD, Professor,13 Greg Zaharchuk, MD,
 202 PhD, Associate Professor,14 and Carolyn C. Meltzer, MD, Professor15: [Imaging](#)
 203 [Recommendations for Acute Stroke and Transient Ischemic Attack Patients: A](#)
 204 [Joint Statement by the American Society of Neuroradiology, the American](#)
 205 [College of Radiology and the Society of NeuroInterventional Surgery](#). AJNR Am
 206 J Neuroradiol. 2013, 34:117-127. [10.3174/ajnr.A3690](#)
- 207 3. Monitoring Editor: Bart M. Demaerschalk Natalie T. Cheng, MD1 and Anthony S.
 208 Kim, MD, MASCORresponding author1: [Intravenous Thrombolysis for Acute](#)
 209 [Ischemic Stroke Within 3 Hours Versus Between 3 and 4.5 Hours of Symptom](#)
 210 [Onset](#). Neurohospitalist. 2015, 5:101-109. [10.1177/1941874415583116](#)
- 211 4. Yannan Yu,1 Quan Han,1 Xinfu Ding,2 Qingmeng Chen,1 Keqi Ye,1 Sheng
 212 Zhang,1 Shenqiang Yan,1 Bruce C. V. Campbell,3 Mark W. Parsons,4 Shaoshi
 213 Wang,5 and Min Loua,1 : [Defining Core and Penumbra in Ischemic Stroke: A](#)
 214 [Voxel- and Volume-Based Analysis of Whole Brain CT Perfusion](#). Sci Rep. 2016,
 215 6:[10.1038/srep20932](#)
- 216 5. Campbell BCV, Donnan GA, Mitchell PJ, et al : [Endovascular thrombectomy for](#)
 217 [stroke: current best practice and future goals](#) . Stroke and Vascular Neurology.
 218 2016, 6:Accessed: [10.1038/srep20932](#): [10.1136/svn-2015-000004](#)
- 219 6. Feng Chen and Yi-Cheng Ni : [Magnetic resonance diffusion-perfusion mismatch](#)
 220 [in acute ischemic stroke: An update](#). World J Radiol. 2012, 4:63-
 221 74. [10.4329/wjr.v4.i3.63](#)
- 222 7. Chia-Yuen Chen, Chin-I Chen, Fong Y. Tsai, Ping-Huei Tsai, Wing P. Chan
 223 : [Prominent Vessel Sign on Susceptibility-Weighted Imaging in Acute Stroke:](#)
 224 [Prediction of Infarct Growth and Clinical Outcome](#). PLOS. 2015, 1-
 225 12. [10.1371/journal.pone.0131118](#)
- 226 8. Atay Vural Rahsan Gocmen Kader Karli Oguz Mehmet Akif Topcuoglu Ethem
 227 Murat Arsava: [Bright and dark vessels on stroke imaging: different sides of the](#)
 228 [same coin?](#). Diagn Interv Radio. 2016, 22:284-290. [10.5152/dir.2015.15271](#)
- 229 9. Kaya D, Dinçer A, Yildiz ME, Cizmeli MO, Erzen C. : [Acute ischemic infarction](#)
 230 [defined by a region of multiple hypointense vessels on gradient-echo T2* MR](#)
 231 [imaging at 3T..](#) Am J Neuroradiol. 2009, 30:1227-32. [10.3174/ajnr.A1537](#)
- 232 10. Haacke EM, Xu Y, Cheng YC, Reichenbach JR. : [Susceptibility-weighted](#)
 233 [imaging \(SWI\)](#). Magn Reson Med. 2004, 52:612-18. [10.1002/mrm.20198](#)
- 234 11. Tsui YK, Tsai FY, Hasso AN, Greensite F, Nguyen BV. : [Susceptibility-weighted](#)
 235 [imaging for differential diagnosis of cerebral vascular pathology: a pictorial](#)
 236 [review](#). . J Neurol Sci . 2009, 287:7-16.
- 237 12. Barber PA1, Hill MD, Eliasziw M, Demchuk AM, Pexman JH, Hudon ME,
 238 Tomanek A, Frayne R, Buchan AM : [Imaging of the brain in acute ischaemic](#)
 239 [stroke: comparison of computed tomography and magnetic resonance diffusion-](#)
 240 [weighted imaging..](#) J Neurol Neurosurg Psychiatry.. 2005, 76:1528-
 241 33. [10.1136/jnnp.2004.059261](#)
- 242 13. Polan RM1, Poretti A1, Huisman TA1, Bosemani T2.: [Susceptibility-weighted](#)
 243 [imaging in pediatric arterial ischemic stroke: a valuable alternative for the](#)

- 244 noninvasive evaluation of altered cerebral hemodynamics.. AJNR Am J
245 Neuroradiol. . 2015, 36:783-8. [10.3174/ajnr.A4187](https://doi.org/10.3174/ajnr.A4187)
- 246 14. Haacke EM, Tang J, Neelavalli J, Cheng YC. : [Susceptibility mapping as a](#)
247 [means to visualize veins and quantify oxygen saturation..](#) J Magn Reson
248 Imaging. 2010, 32:663-76. [2081506510.3174/ajnr.A4187](https://doi.org/10.3174/ajnr.A4187)
- 249 15. Viratsinh Vaghela, Chandrasekharan Kesavadas, Bejoy Thomas: Year : 2010 |
250 Volume : 58 | Issue : 6 | Page : 879-885 Functional magnetic resonance imaging
251 of the brain: A quick review. Neurology India. 2010, 58:879-885. [10.4103/0028-](https://doi.org/10.4103/0028-3886.73735)
252 [3886.73735](https://doi.org/10.4103/0028-3886.73735)
- 253 16. Kao HW, Tsai FY, Hasso AN. 2012;22:1397–1430: [Predicting stroke evolution:](#)
254 [comparison of susceptibility-weighted MR imaging with MR perfusion..](#) Euro
255 Radiol. 2012, 22:1397-1430.. [10.1007/s00330-012-2387-4](https://doi.org/10.1007/s00330-012-2387-4)
- 256 17. Huang P, Chen CH, Lin WC, Lin RT, Khor GT, Liu CK. 2012;259:1426–32
257 pmid:22186853: [Clinical applications of susceptibility weighted imaging in](#)
258 [patients with major stroke. .](#) J Neurol. 2012, 259:1426-32.. [10.1007/s00415-011-](https://doi.org/10.1007/s00415-011-6369-2)
259 [6369-2](https://doi.org/10.1007/s00415-011-6369-2).
- 260 18. Baik SK, Choi W, Oh SJ, Park KP, Park MG, Yang TI, et al. : [Change in cortical](#)
261 [vessel signs on susceptibility-weighted images after full recanalization in](#)
262 [hyperacute ischemic stroke..](#) J Neurol. 2012, 34:206-12. [23006622](https://doi.org/10.1007/s00415-011-6369-2)
- 263 19. Yamashita E, Kanasaki Y, Fujii S, Tanaka T, Hirata Y, Ogawa T: [Comparison of](#)
264 [increased venous contrast in ischemic stroke using phase-sensitive MR imaging](#)
265 [with perfusion changes on flow-sensitive alternating inversion recovery at 3](#)
266 [Tesla..](#) Acta Radiol. 2011, 52:905-10. [2184411](https://doi.org/10.1007/s00415-011-6369-2)
- 267 20. Haacke EM, Tang J, Neelavalli J, Cheng YC. : [Susceptibility mapping as a](#)
268 [means to visualize veins and quantify oxygen saturation. .](#) J Magn Reson
269 Imaging. 2010, 32:663-76. [20815065](https://doi.org/10.3174/ajnr.A4187)
- 270 21. Hermier M, Nighoghossian N, Derex L, Adeleine P, Wiart M, Berthezène Y, et al.
271 : [Hypointense transcerebral veins at T2*-weighted MRI: a marker of hemorrhagic](#)
272 [transformation risk in patients treated with intravenous tissue plasminogen](#)
273 [activator..](#) J Cereb Blood Flow Metab . 2003, 23:1362-70. [14600444](https://doi.org/10.1007/s00415-011-6369-2)
- 274 22. Sobesky J, Zaro Weber O, Lehnhardt FG, Hesselmann V, Neveling M, Jacobs A,
275 et al. : [Does the mismatch match the penumbra? MRI and PET in early ischemic](#)
276 [stroke..](#) Stroke. 2005, 36:980–5 . [15790950](https://doi.org/10.1161/01.STR.0000157909.52812.1d)
- 277 23. Rosso C, Hevia-Montiel N, Deltour S, Bardinet E, Dormont D, Crozier S, et al.
278 : [Prediction of Infarct growth based on apparent diffusion coefficients: penumbral](#)
279 [assessment without intravenous contrast material..](#) Radiology. 2009, 250:184–
280 [92. 19017923](https://doi.org/10.1148/radiol.20091017923)
- 281 24. Wechsler LR.: [Imaging evaluation of acute ischemic stroke..](#) Stroke . 2011,
282 [42:12–15. 21164129](https://doi.org/10.1161/01.STR.0000321164.12912.1d)
- 283 25. Rivers CS, Wardlaw JM, Armitage PA, Bastin ME, Carpenter TK, Cvorovic V, et al.
284 : [Do acute diffusion- and perfusion-weighted MRI lesions identify final infarct](#)
285 [volume in ischemic stroke?.](#) Stroke . 2006, 37:98–104. [16322499](https://doi.org/10.1161/01.STR.0000163224.99123.1d)
- 286 26. Tsui YK, Tsai FY, Hasso AN, Greensite F, Nguyen BV. : [Susceptibility-weighted](#)
287 [imaging for differential diagnosis of cerebral vascular pathology: a pictorial](#)
288 [review..](#) J Neurol Sci. 2009, 287:7–16. [19772973](https://doi.org/10.1016/j.jns.2009.07.073)

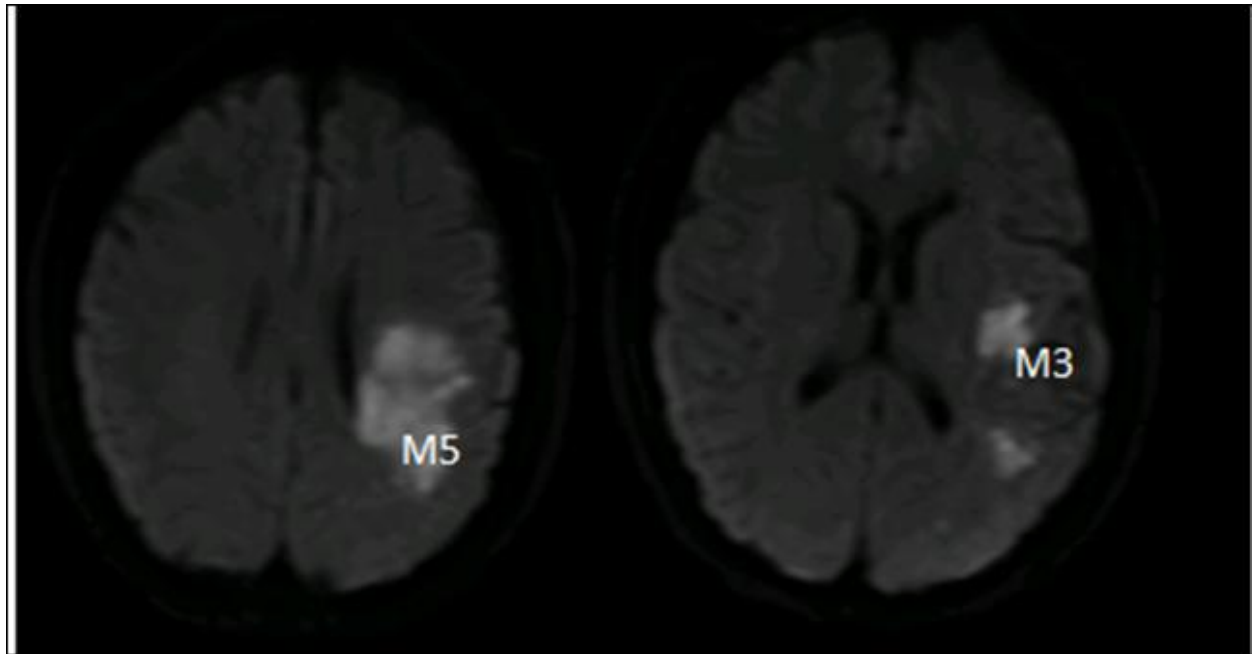
- 289 27. Hermier H, Nighoghossian N. : [Contribution of susceptibility-weighted imaging to](#)
290 [acute stroke assesement.](#). Stroke. 2004, 35:1989–94 . [15192245](#)
- 291 28. Mittal S, Wu Z, Neelavalli J, Haacke EM. : [Susceptibility-weighted imaging:](#)
292 [technical aspects and clinical applications. Part 2.](#) . Am J Neuroradiol . 2009,
293 30:232–52 . [19131406](#)
- 294 29. Tong KA, Ashwal S, Obenaus A, Nickerson JP, Kido D, Haacke
295 EM.: [Susceptibility-weighted MR imaging: a review of clinical applications in](#)
296 [children.](#) Am J Neuroradiol . 2008, 29:9–17 . [7925363](#)
- 297 30. Rovira A, Orellana P, Alvarez-Sabín J, Arenillas JF, Aymerich X, Grivé E, et al.
298 : [Hyperacute ischemic stroke: middle cerebral artery susceptibility sign at echo-](#)
299 [planar gradient-echo MR imaging.](#). Radiology. 2004, 232:466–73. [15215546](#)

301



302

303



304

305 A 63-year-old woman had a diagnosis of LEFT middle cerebral artery territory infarct.
306 Susceptibility-weighted imaging reveals prominent hypointense cortical and medullary vessels
307 diffusely seen in the insula and M1 to M6 zones of the left
308 Middle cerebral artery territory. Engorged deep veins and thalamostriate artery over the lesions
309 compared with the healthy side were also noted. Involved
310 M1 to M6 zones and insula lost 7 points and an engorged thalamostriate vein lost 3 points. The
311 prominent vessel sign score was 0 ($10 - 7 - 3 = 0$).
312 Susceptibility-weighted imaging (C, D) at the basal ganglia and suprabasal ganglion levels reveal
313 prominent vessel signs in the cortical veins (arrows),
314 medullary veins (arrows) and thalamostriate vein (arrowhead).