Study	Terminology	Definition	Appearance
Stroke studies			
Acute ischemic stro	oke		
Choi et al., 2017	HARM	Cortical and sulcal contrast retention	Appearance: linear cortical or sulcal enhancement. Location: 82% in stroke vascular territory, 18% in multiple vascular territories ipsilateral or contralateral regardless of infarction
Dechambre et al., 2000	High signal from the CSF	High signal in subarachnoid space (higher signal intensity compared to cortex)	Appearance/location: diffuse bilateral (n=2); diffuse limited to stroke hemisphere (n=2); focal next to ischemic lesion (n=1); enhancement progressively decreased in extent and intensity and disappeared completely within 3 (n=2), and 6 (n=2) days.
Forster et al., 2016	HARM	Focal enhancement in the subarachnoid space and/or the ventricles	Appearance: focal in the subarachnoid space and/or the ventricles. Location: occipital sulci (n=7), ambient cistern (n=1), in all cases in the same vascular territory but remote from acute ischemic lesion.

Supplemental table e-4: Terminology, definition and appearance of post-contrast FLAIR CSF space enhancement.

Gupta et al., 2017	HARM	The presence of gadolinium in	Appearance and location not specified
		the sulci or parenchyma	
Henning et al.,	HARM	Positive signal enhancement;	Location: within the vascular territory of the acute stroke, appearance not specified.
2008 ±		verification of enhancement in	
		the CSF space (not parenchyma)	
Hjort et al., 2008	HARM	Hyperintensity in the CSF	Appearance and location not specified; Enhancement was observed both 2h and 24h after
			thrombolytic therapy
Kim et al., 2005	Sulcal hyperintensity	Presence of sulcal hyperintensity	Appearance: sulcal hyperintensity. Location: focally near the infarcted area in all cases;
			disappeared within 3 days after symptom onset.
Latour et al., 2004	HARM	Delayed gadolinium	Location/appearance: focally in the sulcal space in the vascular territory of the acute
\pm , Warach et al.,		enhancement of CSF space	stroke (n=21), both focally and diffusely within the ventricles (n=20) and only diffusely
2004 ±			in the ventricles (n=6), median time from onset of ischemia to observation of HARM
			was 13h; CSF enhancement was observed as late as 5 days after prior CA. HARM was
			not evident within 10min after CA.
Luby et al., 2019	HARM	Enhancement in the CSF spaces,	HARM was rated on a semi-quantitative scale; none, present but not severe, severe
		especially in the sulci and on the	occupying at least 10 slices but focal to the acute diffusion-weighted imaging ischemic
		cortical surface	

lesion, or severe occupying at least 10 slices but diffuse and not just limited to the territory where the acute ischemic lesion was present.

Nadareishvili et al., HARM	Enhancement of the CSF space	Appearance: 82.5% had enhancement occupying at least 10 slices. Location not
2018	(sulci, ventricles, background, or	specified.
	vitreous appears hyperintense	
	and continuous across >10	
	slices)	
Ostwaldt et al., HARM	CSF enhancement in the sulcal	Appearance and location not specified
2014 #	space in the affected vascular	
	territory or within the ventricles	
Ostwaldt et al., HARM	Hyperintensities in sulci or	Location: within the vascular territory of the acute stroke. HARM was present on all
2015 #	ventricles on >2 consecutive	follow-up images in most cases (n=6), occurred after 3 doses of CA (28.5h after symptom
	slices	onset) in 1 case, and appeared after 2 doses of CA (7.2h after symptom onset) and had
		disappeared 12 hours later (n=1)
Villringer et al., HARM	-	Appearance and location not specified
2017 #		

Acute ischemic stroke or transient ischemic attack

Lee et al., 2015 * HARM	Linear contrast enhancement	Appearance: focal linear contrast leakage. Location: Both cerebral sulci (n=4), both
	(sulcal enhancement)	occipital sulci (n=2), cerebellar sulcus (n=1), both parieto-occipital sulci (n=1), frontal
		and occipital sulci (n=1), both high frontoparietal sulci (n=1). Focal enhancement
		became diffuse on $<24h$ FU MRIs in all cases (n=5). Cases with $>24h$ FU MRI showed
		a small amount of contrast leakage (n=1) or no leakage (n=5)
Lee et al., 2016 * HARM	Linear contrast enhancement	Appearance: linear contrast enhancement along the cortical surface near acute
	(abnormal cortical or sulcal	infarctions. Location: multifocal (n=10, covering multiple vascular territories in 8 cases
	enhancement)	and in the same vascular territory as the infarction in 2 cases), focal in the same vascular
		territory as the acute infarction (n=1), and focal in the absence of infarction (n=1); initial
		linear enhancement spread diffusely to fill the subarachnoid space 2.5-19h later, and
		disappeared in the subset of cases who were again imaged 3-5 days later.
Lee et al., 2018 HARM	-	Appearance and location not specified
Rozanski et al., HARM	CSF enhancement in sulci and/or	Appearance and location not specified
2010 #	ventricles	
Spontaneous intracerebral hem	orrhage	
Kidwell et al., HARM	Hyperintense signal within the	Sulcal enhancement was noncontiguous with the hematoma in all cases. 50% showed
2011	CSF spaces	moderate to severe HARM

Jolink et al., 2019	HARM	Hyperintense signal in normal-	Location: cortical (94%), deep and cortical (6%), never infratentorial; Both hemispheres
		appearing brain or CSF, visually	(53%), symptomatic hemisphere (12%), contralateral hemisphere (35%). Appearance:
		distinct and anatomically non-	24% single punctate, 29% multi-punctate, 18% focal sulcal enhancement, 29% bilateral
		contiguous with the hematoma	and diffuse; anatomically non-contiguous with the hematoma
Acute ischemic str	oke, spontaneous intrace	rebral hemorrhage or transient iso	chemic attack (mixed cases)
Barr et al., 2010	HARM	Enhancement of the CSF space	Appearance and location not specified
		(sulci, ventricles, background, or	
		vitreous appears hyperintense	
		and continuous across >10	
		slices)	

Post-cardiovascular and intracranial vascular surgery studies

Treatment for carotid artery disease

Cho et al., 2014 HARM

Delayed

gadolinium Appearance and location not specified

enhancement of the CSF space

Ogami et al., 2011	Enhancement of the	Hyperintensity or obscuration of	Location: all CSF enhancements were distributed within the MCA territory at the core
	CSF space	the CSF space	of the watershed area between the MCA and the PCA. Three lesions were recognized in
			the watershed area between the MCA and the ACA. 0% in the basal cistern
Wilkinson et al.,	LME	Abnormal accumulation of	Location: 50% unilateral enhancement of the leptomeninges primarily MCA territory,
2000		contrast media in the pia and/or	50% focal enhancement adjacent to areas of old ischemic damage; 0% showed LME in
		arachnoid mater	ACA territory, LME in PCA territory was minimal; Ipsilateral to the stented carotid
			artery (n=11), contralateral to the stent in 1 case (the opposite carotid artery was
			occluded). In all cases LME became more prominent after 2nd CA.
Aneurysm treatme	nt		
Li et al., 2018	LME	Leptomeningeal enhancement	38% focal, 47% lobar, 15% hemispheric (based on rater 1)
Suthiphosuwan et	CSHF (cortical and	Cortical or sulcal hyperintensity	Location: right ACA territory (47%), right MCA territory (56%), left ACA territory
al., 2018	sulcal hyperintensity on	equal to or greater than a	(13%), left MCA territory (13%), right PCA territory (19%), left PCA territory (13%),
	gadolinium-enhanced	superficial cortical vein	left cerebellar hemispheric territory (6%); enhancement was observed in the arterial
	FLAIR)		territories exposed to iodinated contrast media during angiography.

Cardiac surgery

Merino et al., 2013	HARM	Enhancement of the CSF	HARM was present as mild/moderate background signal (n=4),
			sulcal(severe)+background (severe)+ventricular(mild/moderate) (n=1),
			sulcal(severe)+background (mild/moderate) (n=1) or only sulcal(mild/moderate) (n=3)
Multiple sclerosis s	tudies		
Absinta et al., 2015	LME	Signal intensity within the	109 foci were detected in 74 cases. Location: 19% cerebral convexity 56% within a
		subarachnoid space substantially	sulcus, 17% along a dural fissure, 7% traversing several of these areas (8/109, 7%). In
		greater than that of brain	one case high resolution T2* images were acquired at 7T MRI, which showed that LME
		parenchyma	was perivascular. Of 62 foci that were measured at FU imaging, 53 were stable in shape
			and size, 1 disappeared, 2 fluctuated over time, and 6 new foci were detected in 4 cases.
			1 control case presented with a single focus of LME in the right frontal region.
Bergsland et al.,	LMCE	Signal intensity within the	80 foci detected in 58 cases. Appearance: 65% nodular, 20% plate-like, 15% linear.
2019 \$		subarachnoid space that was	
		substantially greater than that of	
		brain parenchyma	
Eisele et al., 2015	Cortical or	Sulcal or leptomeningeal	In one case focal CSF enhancement was observed in the right temporal lobe.
	leptomeningeal contrast	hyperintensity	
	enhancement		

Harrison et al., LME	Hyperintensities on subtraction	Median of 2 foci per case (range 0-8). Appearance: nodular foci in 51% of cases;
2017 &	images within the	spread/fill foci in 76% of cases; control cases (n=2) had two or three nodular foci.
	leptomeningeal space (pia,	
	subarachnoid, or subarachnoid	
	space)	
Ighani et al., 2020 LME	Hyperintensities on pcFLAIR	Median of 3 foci per case (range 0-14). Appearance: spread/fill-gyral foci in 61% of
&	images and subtraction images	cases, spread/fill-sulcal foci in 59%, nodular in 32%. Locaton: infratentorial in 10% of
	within the leptomeningeal space	cases
Jonas et al., 2018 & Meningeal	Hyperintensities in the	284 foci detected in 31 cases. Appearance: 40% subarachnoid spread/fill foci, 5%
enhancement	meningeal space	subarachnoid nodular foci, 38% vessel wall pattern, 16% dural nodular foci; Location:
		44% frontal, 31% parietal, 15% occipital, 8% temporal lobe, 1% cerebellar. At 1-year
		FU, persistence was noted in 89% overall foci, and in 82% at 2-year FU.
Zivadinov et al., LMCE	Signal intensity within the	61 foci detected in 25 cases. Appearance: 80% nodular, 13% plate-like, 7% linear.
2017 s	subarachnoid space that was	Location: 21% in sulci, 79% cortical convexity; 41% right hemisphere, 59% left
	substantially greater than that of	hemisphere; 93% supratentorial, 7% infratentorial; lobar foci: 20% frontal, 18% parietal,
	brain parenchyma	16% temporal, 39% occipital lobe.

Zivadinov et al., LMCE	Signal intensity within the	61 foci were detected in 39 cases using a subtraction method. Appearance: 68% nodular,
2018 \$	subarachnoid space that was	11% plate-like, 20% linear. Location: 70% sulci, 30% cortical convexity; 64% right
	substantially greater than that of	hemisphere, 34% left hemisphere, all supratentorial. 30% frontal, 36% parietal, 8%
	brain parenchyma	temporal, and 25% occipital lobe.
Zurawski et al., LME	Hyperintense signal on	54 foci were identified in 20 cases. Appearance: all cases had at least 1 nodular LME
2020	pcFLAIR with no signal on pre-	focus, 8 cases also had spread/fill LME. Location: 43% left hemisphere, 57% right
	contrast FLAIR	hemisphere; 52% frontal, 31% parietal, 13% temporal, 4% occipital.
Studies in meningitis		
Ahmad et al., 2005 LME	Thick, long or nodular	Appearance and location not specified
	Thick, long or nodular meningeal enhancement, noted	Appearance and location not specified
		Appearance and location not specified
	meningeal enhancement, noted	Appearance and location not specified
	meningeal enhancement, noted on >3 contiguous images, and extending deep into sulcal bases	Appearance and location not specified Distinct leptomeningeal enhancement along each cerebral lobe of both hemispheres and
Ahmad et al., 2005 LME	meningeal enhancement, noted on >3 contiguous images, and extending deep into sulcal bases	

Fukuoka et al.,	LME	Abnormal leptomeningeal	Abnormal leptomeningeal enhancement in the sulci.
2010		enhancement	
Splendiani et al.,	Pathological	Abnormal enhancement in sulci,	Abnormal enhancement was marked and circumscribed or diffuse.
2005	leptomeningeal altered	cisterns, ventricles, or any	
	signals	combination of these	

Studies in other diseases

Memory clinic patients

Freeze et al., 2017	Pericortical	Hyperintense signal	32 pericortical foci were detected in 21 cases (including control cases). Location: 31%
@	enhancement	substantially brighter than the	PCA, 28% MCA, 41% ACA; 47% left hemisphere, 53% right hemisphere; 38% on the
		proximal parenchymal signal	cortical convexity, 38% within a sulcus, 3% traversed several sulci, 22% within the
		intensity, occurring in the	longitudinal fissure. 1 focus in 23 cases, 2 foci in five cases, 3 foci in three cases. In
		subarachnoid space	addition, 1 focus was found in a perivascular space. At FU all foci remained stable over
			time.

Freeze et al., 2019	Pericortical	Hyperintense signal	All pericortical foci had remained stable since baseline, one new leakage focus was
@	enhancement	substantially brighter than the	detected. Foci appeared visually more intense on heavily T2w pcT2wFLAIR images
		proximal parenchymal signal	compared to regular pcT2wFLAIR images. In one case, widespread signal enhancement
		intensity, occurring in the	within the sulci became visible on the heavily T2w pcT2wFLAIR scan several hours
		subarachnoid space	after CA, which was not present on the scan shortly after CA.
Susac syndrome			
Coulette et al.,	LME	Hyperintensity within the	All cases with Susac syndrome had at least 3 LME lesions of which at least one in the
2019		subarachnoid space substantially	posterior fossa. MS cases never had more than 2 lesions and LME was never detected
		greater than that of the brain	within the posterior fossa. LME appeared as punctate, linear, or mixed. On FU MRIs
		parenchyma	(n=53) LME was seen in 7 of 9 cases with Susac syndrome on 40 follow-up MRIs. LME
			lesion load remained unchanged in 36%, was decreased in 23%, and was increased in
			41% of MRIs.
Familial amyloid p	oolyneuropathy		
Hirai et al., 2005	CSF enhancement	Contrast enhancement of the	LME was seen immediately after CA especially in the superior cerebellar cisterns,
		leptomeninges	Sylvian fissures, interhemispheric fissures, and cerebral sulci. 3 and 6 hours after CA
			there was marked and diffuse CSF enhancement. At 24 hours there was mild CSF
			enhancement in the sulci.

Mixed (non-MS) infectious and non-infectious diseases

Absinta et al., 2017 LME	Signal intensity within the	99 foci were detected in 56 cases, 75% had a single focus and 25% had multiple foci.
	subarachnoid space substantially	Appearance: linear or nodular. Location: mostly supratentorial.
	greater than that of brain	
	parenchyma	

#*±&\$@ Studies with (suspected) overlapping study samples.

Abbreviations: CSF, cerebrospinal fluid; FU, follow-up; HARM, Hyperintense acute reperfusion (injury) marker; LME, leptomeningeal enhancement; LMCE, leptomeningeal contrast enhancement; FLAIR, fluid-attenuated inversion recovery

; -, not available.