



THE ROLE OF DIFFUSION-WEIGHTED MAGNETIC RESONANCE IMAGING IN DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL-CYSTIC LESIONS

Radiodiagnosis

Dr. Akriti Gupta	Resident Department Of Radiodiagnosis, GMCH, Udaipur
Dr. Abhijit Patil*	Professor Department Of Radiodiagnosis, GMCH, Udaipur *Corresponding Author
Dr. N.C. Sharma	Sr. Professor Department Of Radiodiagnosis, GMCH, Udaipur
Dr. Shivam Batra	Resident Department Of Radiodiagnosis, GMCH, Udaipur

ABSTRACT

INTRODUCTION: Currently there is widespread use of MRI to determine the tumour extent for surgical and radiotherapy planning, as well as for post-therapy monitoring of tumour recurrence or progression. However, biopsy is still generally considered the gold standard for determining the cancer type and degree of malignancy.

OBJECTIVE: To compare the features of intracranial cystic lesions on various MRI sequences (T1 WI, T2 WI, FLAIR, DWI, ADC map, T2*GRE, etc.).

MATERIALS AND METHODS: The MRI was done on SEIMENS - Magnetom AVANTO 1.5 Tesla. The head coil was used for the scan. After intravenous administration of 10 ml of Gadolinium contrast, 5 mm T1W axial, sagittal, coronal images as well as 5 mm T1WI were obtained as and when required. Additionally, 0.9 mm thin post gadolinium 3D T1 weighted SPGR (spoiled gradient recoil).

RESULTS: DWI in MRI is capable of differentiating brain abscess from primary intracranial malignancy, High grade (IV) glioma and intracranial metastasis with accuracy of 87.1%, 90% and 89.4% respectively.

CONCLUSION: Contrast enhance MRI is an integral part in the initial evaluation of intracranial lesions. However, in some instances, contrast enhanced MRI alone without the use of DWI is not effective for the differentiation of intracranial cystic lesion type or for detection of tumour grade. DWI can increase both the sensitivity and specificity of MRI in the evaluation of these intracranial cystic lesions by providing information about cellularity and restricted movement of water molecules in the tissue, which may in turn improve the prediction of etiology of the lesion.

KEYWORDS

INTRODUCTION

Currently there is widespread use of MRI to determine the tumour extent for surgical and radiotherapy planning, as well as for post-therapy monitoring of tumour recurrence or progression. MRI provides an initial diagnosis of an intracranial mass lesion with a success rate of 30-90% depending on tumour types¹. However, biopsy is still generally considered the gold standard for determining the cancer type and degree of malignancy².

Differentiating brain infections from brain tumours (abscesses from necrotic or cystic brain tumours and encephalitis from diffuse gliomas) is a fundamental clinical issue as the strategies for their management and their prognosis differ completely. Unfortunately, this differentiation remains a diagnostic challenge for both neurologists and radiologists. It is shown that conventional MRI has only 61.4% sensitivity in differentiating brain cystic neoplasms from abscesses as their main differential diagnosis³.

Regions with restricted mobility of water molecules yield higher DW-MRI signals and appear bright. In Apparent diffusion coefficient maps, a region with higher water mobility appears bright. Till today, DWI has been used mainly being used for the detection of acute cerebral infarction. However, there are other pathologies in which DWI could facilitate diagnostic insight.

Diffusion-weighted imaging (DWI) provides a way to assess the diffusion properties of the water molecules in tissues of the brain that may be substantially altered by diseases. According to Fick's law, true diffusion is the net movement of molecules due to a concentration gradient. With MRI, molecular motion due to concentration gradients cannot be differentiated from molecular motion due to pressure gradients, thermal gradients, or ionic interactions. Therefore, when measuring molecular motion with DWI, only the ADC can be calculated⁴.

DWI is useful in providing a greater degree of confidence in distinguishing brain abscesses from cystic or necrotic brain tumours than conventional MRI. It may increase the diagnostic accuracy when combined with other sequences. Likewise, in Creutzfeldt-Jakob disease, DWI helps differentiate from infarct by showing persistent restricted diffusion⁵.

AIMS AND OBJECTIVES

- To compare the features of intracranial cystic lesions on various MRI sequences (T1 WI, T2 WI, FLAIR, DWI, ADC map, T2*GRE, etc.).
- To study the role of DW - MRI in differentiating brain abscess and other intracranial cystic lesions.

MATERIALS AND METHODS

The study was performed in Department of Radiodiagnosis of Geetanjali Medical College and Hospital, Udaipur to describe imaging features of intracranial cystic lesions on MRI with contrast administration whenever necessary. This study consists of 68 patients identified with intracranial cystic lesions on MRI.

The referred patients underwent MRI scans after an informed consent was obtained and MRI contraindications were excluded. The MRI was done on SEIMENS - Magnetom AVANTO 1.5 Tesla. The head coil was used for the scan.

TECHNIQUE OF MRI EXAMINATION:

All patients were screened before entry into the MRI scanning room for ferromagnetic objects, cardiac pacemakers, aneurysm clips etc. Upper body clothing with metallic trim or any clothing likely to create static electricity were removed. The patients were given disposable earplugs to attenuate the gradient switching noise.

Initial topogram of the head was obtained and sequences were planned according to the MRI Brain protocol. Initial localizer of the head was obtained and sequences were planned according to the MRI protocol ordered by the referring doctor. IV contrast agent was used wherever necessary. MRI protocol at 1.5 T included:

NON-CONTRAST IMAGES:

- 5 mm thick TSE T1 weighted images axial and sagittal,
- 5 mm thick TSE T2 weighted images axial and coronal,
- 5 mm thick TSE T2 weighted FLAIR (Fluid attenuation inversion recovery),
- 5 mm thick TSE T2 weighted EPI diffusion weighted & ADC (Apparent Diffusion Coefficient) images.
- 5 mm thick TSE T2 x weighted GRE images (Gradient echo images)

After intravenous administration of 10 ml of Gadolinium contrast, 5 mm T1W axial, sagittal, coronal images as well as 5 mm T1WI were

obtained as and when required. Additionally, 0.9 mm thin post gadolinium 3D T1 weighted SPGR (spoiled gradient recoil) Inversion recovery prepared sequences were obtained as when required.

Further special imaging technique like MRI spectroscopy are used, if lesions are larger and located away from bone and base of skull.

The scans were studied in detail on monitor and finally films were taken for permanent record. Every effort was made to make sure of high-quality scans and to avoid artifacts.

Dosage of contrast used was 0.1 mg/kg wt. As a precautionary measure, resuscitation apparatus and emergency drugs were kept ready MRI findings with provisional diagnosis (In case of differential diagnosis given in MRI report, the first differential was assumed as MRI diagnosis) and clinical history were recorded as per the proforma.

RESULTS

Table 1: Etiological classification of intracranial cystic lesions in the studied population

Etiological Classification	No.	%
Congenital	17	25
Infectious	20	29.41
Encephalomalacia	2	2.94
Primary malignancy	20	2.94
Secondary malignancy	9	13.23

Here, the most common intra-cranial cysts were in infectious in nature and primary malignancy (29.4%) each followed by congenital (25%) and secondary malignancy was seen in 13.2% lesions.

Table 2: Age wise distribution of individual intracranial lesions.

MRI diagnosis	<10 years		11-30 years		31-50 years		51-70 years		>70 years	
	No.	%	No.	%	No.	%	No.	%	No.	%
Congenital Lesions	1	1.5	4	5.9	6	8.8	4	5.9	2	2.9
Brain Abscess	1	1.5	7	10.2	2	2.9	1	1.5	0	0
Encephalomalacia	0	0	2	2.8	0	0	0	0	0	0
Grade I/II Glioma	0	0	2	2.9	0	0	2	2.8	0	0
Grade II/III Glioma	0	0	4	5.8	2	2.9	0	0	1	1.5
Grade IV Glioma	0	0	1	1.5	2	2.9	5	7.3	1	1.5
Metastasis	0	0	1	1.5	3	4.4	5	7.3	0	0
NCC	0	0	5	7.3	2	2.9	2	2.9	0	0
Total	2	2.9	26	38.2	17	25	19	27.9	4	5.8

Intracranial lesions were most common in age group 11-30 years followed by 51-70 years age group. Here, the lesions were most prevalent in 3rd and 4th decade of life. In concordance with this study by Reddy et al⁸ reported that Intra cranial lesions were found in patients of all age groups.

Table 3: Categorization on basis of number of lesions among studied population acquired intracranial cystic lesions.

MRI diagnosis	Solitary		2-4 no.		Multiple (>4)	
	No.	%	No.	%	No.	%
Brain Abscess	7	13.7	3	5.8	1	1.9
Neutocysticercosis	0	0	0	0	9	17.6
Grade I/II Glioma	4	7.8	0	0	0	0
Grade II/III Glioma	7	13.7	0	0	0	0
Grade IV Glioma	7	13.7	1	1.9	1	1.9
Metastasis	1	1.9	4	7.8	4	7.8
Encephalomalacia	2	3.9	0	0	0	0

In our study the most of the lesions were single (61.7%) followed by 2-4 lesions (19.1%) and multiple lesions (11.9%). Out of them single lesion were most common in brain abscess, Grade II/III Glioma and Grade IV Glioma with 13.7 % each followed by Grade I/II Glioma (7.8%) other constitute less than 5%. 2-4 lesion were most common in metastasis (7.8%) & >4 lesions were most common NCC (17.6%) followed by metastasis (7.8%). In study by Jindal et al⁷ Solitary lesions were present in 58 patients (72.5%) & multiple lesions in 22 patients (27.5%). Garg et al⁹ found 19(63.3%) patients had a single lesion. 2-4 lesions were noted in 8 (26.6%) cases and > 4 RELs were seen in 3 (10%) cases.

Table 4: Categorization on basis of wall thickness of acquired intracranial cystic lesions among studied population.

MRI diagnosis	<5mm		>5mm	
	No.	%	No.	%
Brain Abscess	8	15.69%	3	5.88%
NCC	9	17.65%	0	0.00%
Grade I/II Glioma	0	0.00%	4	7.84%
Grade II/III Glioma	2	3.92%	5	9.80%
Grade IV Glioma	2	3.92%	7	13.73%
Metastasis	4	7.84%	5	9.80%
Encephalomalacia	2	3.92%	0	0.00%

Mostly intracranial neoplastic lesion had size greater than 20mm (58.5%). And there were 41.2% lesion had size less than 20 mm. Garg et al⁸ found that Maximum number of patients (66.6%) had lesion size ranging between 2-4 cm. 20% patients had lesion size measuring less than 2cms. 13.3% of the total patients had lesion size measuring more than 4cms. We also found that According to thickness of wall Brain abscess (15.6%), metastasis (9.8%) were having wall thickness was greater than 5mm and lesions less than 5 mm wall thickness includes NCC (17.5%) followed by Grade- IV glioma (13.7%), Grade II/III glioma (9.8%).

Table 5: Imaging characteristics of intracranial cystic lesions on T2W, FLAIR and DWI sequences.

MRI diagnosis		T2W		FLAIR		DWI	
		No	%	No	%	No	%
Congenital lesions	Hypointense	2	11.6	14	81.2	11	63.8
	hyperintense	15	88.2	2	11.6	1	8.5
	Isointense	0	0	1	5.8	5	29
Brain Abscess	Hypointense	0	0	0	0	0	0
	hyperintense	11	100	11	100	11	100
	Isointense	0	0	0	0	0	0
Encephalomalacia	Hypointense	0	0	2	100	2	100
	hyperintense	2	100	0	0	0	0
	Isointense	0	0	0	0	0	0
Grade I/II Glioma	Hypointense	0	0	0	0	0	0
	hyperintense	4	100	4	100	0	0
	Isointense	0	0	0	0	4	100
Grade II/III Glioma	Hypointense	0	0	0	0	0	0
	hyperintense	7	100	7	100	2	28.5
	Isointense	0	0	0	0	5	71.5
Grade IV Glioma	Hypointense	0	0	0	0	6	66.7
	hyperintense	9	100	9	100	1	11.1
	Isointense	0	0	0	0	2	22.2
Metastasis	Hypointense	0	0	2	22.2	4	44.5
	hyperintense	9	100	7	77.8	3	33.3
	Isointense	0	0	0	0	2	22.2
NCC	Hypointense	0	0	9	100	9	100
	hyperintense	9	100	0	0	0	0
	Isointense	0	0	0	0	0	0

In our study among the congenital lesions 88.2% show hyperintense on T2W, 81.2% hypointense on FLAIR and only one of congenital lesion show restriction on DWI, i.e. epidermoid cyst. Intracranial epidermoid cyst and arachnoid cyst give equal signal intensity with CSF on all conventional MR sequences. Therefore, by the means of DW, the differentiation between the two can be done. Y. Bukte et al⁹, studied 5 cases of epidermoid cyst, out of which all showed diffusion restriction when compared with other intracranial cystic lesion.

Similarly, in brain abscess 100% showed hyperintensity on T2W, DWI & FLAIR and 90.9% showed central intense hyperintensity on DWI, while 9% (1 out of 11) showed peripheral restriction. Cystic Low grade (I/II/III) gliomas appeared hyperintense on T2W and FLAIR and 18% of low-grade glioma showed patchy restriction on DWI in solid part. Cystic High-grade glioma appeared hyperintense on T2W and on FLAIR, only 11.1% of the high-grade glioma showed patchy restriction on DWI in solid component with corresponding low ADC value, which could be also contributed due to cytotoxic edema. ADC Rana et al¹⁰ showed that central part of brain abscesses gives a high signal on DWI and have low ADC, whereas cystic or necrotic brain tumors have the opposite pattern. 100% cases of brain abscess showed

discrete T2 Hypointense rim. Haimes et al¹¹ showed 9 out of the 10 brain abscess showed discrete capsule appearing iso to hypointense capsule to which matter on T2W imaging.

Metastasis appear hyperintense to heterogenous on T2W & FLAIR and 33.3% of metastasis show restriction on DWI, which is peripheral with corresponding low ADC value, while the central necrotic part showed facilitated diffusion & appeared hyperintense on. Holtas et al¹² reported a metastatic brain tumor that showed a bright signal on DWI which is peripheral. Reddy et al¹³ found that Ninety-three abscess lesions showed central hyperintensity on T2-WI and DWI. Bukte et al¹⁴ found All 22 abscesses were hyperintense on DWI. The cystic or necrotic areas of all primary tumors were hypo- (90%, $n_z = 17$) or isointense (10%, $n_z = 2$) on DWI. Of the metastatic tumors, 27% ($n_z = 3$) occurred in the same patient and were hyperintense (peripheral) on DWI.

Table 6: Efficacy of DWI in MRI in differentiating brain abscess from intracranial malignancy among studied population.

	Sensitivity	Specificity	PPV	NPV	Accuracy
Abscess Vs Primary Malignancy	100%	85%	75%	94.4%	87.1%
Abscess Vs High Grade Glioma	100%	88%	90%	88.8%	90%
Abscess Vs Metastasis	100%	70%	75%	87.5%	89.4%

DWI in MRI is capable of differentiating brain abscess from primary intracranial malignancy, High grade (IV) glioma and intracranial metastasis with accuracy of 87.1%, 90% and 89.4% respectively.

CONCLUSION

Contrast enhance MRI is an integral part in the initial evaluation of intracranial lesions. However, in some instances, contrast enhanced MRI alone without the use of DWI is not effective for the differentiation of intracranial cystic lesion type or for detection of tumour grade. DWI can increase both the sensitivity and specificity of MRI in the evaluation of these intracranial cystic lesions by providing information about cellularity and restricted movement of water molecules in the tissue, which may in turn improve the prediction of etiology of the lesion.

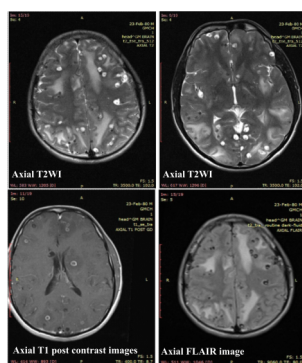


Figure 1 : There is a T1 hypointense, T2 hyperintense conglomerated lesion in left cerebral hemisphere with significant perilesional edema and blooming on T2* GRE sequence representing haemorrhage. There is no restriction on DWI

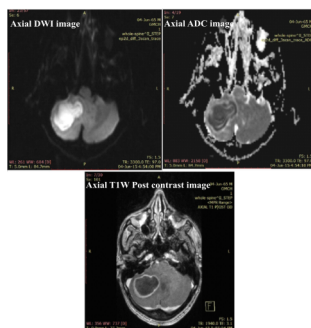


Figure 2 : Two well defined ring enhancing lesions are noted involving right cerebellar hemisphere and right frontal region

appearing hyperintense on T2WI and hypointense on T1W with marked hyperintense on DWI with corresponding hypointense ADC. These imaging findings are suggestive of brain abscess

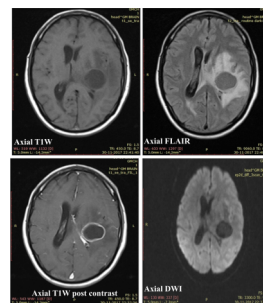


Figure 3 : Multiple well defined ring enhancing lesions are noted involving both cerebral and cerebellar hemispheres. The lesions appear hypo to hyperintense on T2WI and hypo to iso intense on T1WI & FLAIR with few of them showing central dot appearing hypointense on T2WI and iso to hyperintense on T1WI & FLAIR. Few lesions show blooming on GRE (T2*) images. Mild surrounding vasogenic edema is noted around the lesions, Above finding are SUGGESTIVE of NCC

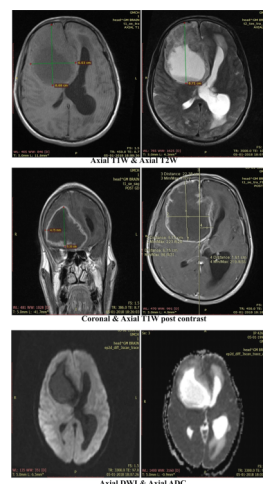


Figure 4 : Large well defined irregularly margined mass right frontal region appearing heterogeneously hyperintense on T2/FLAIR and hypointense on T1 with nodular wall contrast enhancement. Lesion shows significant perilesional edema, effacement of right lateral with significant midline shift & periventricular CSF seepage. There is no restriction on DWI

REFERENCES

- Coutinho JM, Ferro JM, Canhão P, Cantú C, Boussier M, Stam J. Cerebral Venous and Sinus Thrombosis in Women. Stroke 2009; 2356–61.
- Stam J. Cerebral venous and sinus thrombosis: incidence and causes. Adv Neurol. 2003; 92: 225–32.
- Masuhr F, Mehraein S, Einhaupl K. Cerebral venous and sinus thrombosis. J Neurol. 2004; 251(1): 11–23.
- Ameri A, Boussier MG. Cerebral venous thrombosis. Neurol Clin. 1992; 10(1): 87–111.
- Walecki J, Mruk B, Nawrocka-Laskus E, Piliszek A, Przelaskowski A, Sklinda K. Neuroimaging of cerebral venous thrombosis (CVT) – Old dilemma and the new diagnostic methods. Polish J Radiol. 2015 Jul 25;80(1):368–73.
- Altinkaya N, Demir S, Alkan O, Tan M. Diagnostic value of T2*-weighted gradient-echo MRI for segmental evaluation in cerebral venous sinus thrombosis. Clin Imaging. 2015 Jan 1;39(1):15–9.
- Yildiz ME, Ozcan UA, Turk A, Ulus OS, Erzen C, Dinçer A. Diffusion-Weighted MR Imaging Findings of Cortical Vein Thrombosis at 3T. Clin Neuroradiol. 2015;25(3):249–56.
- Chu K, Kang DW, Yoon BW, Roh JK. Diffusion-weighted magnetic resonance in cerebral venous thrombosis. Arch Neurol. 2001;58(10):1569–76.