

RING ENHANCING LESIONS IN BRAIN – IMAGING EXPERIENCE AT A TERTIARY CARE CENTER IN WESTERN INDIA

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ABSTRACT

Ring-enhancing lesions in the brain can be due to neoplastic and non-neoplastic diseases. Infectious, vascular and inflammatory aetiologies are non-neoplastic and primary brain tumour and metastasis are important aetiologies for neoplastic disorders that cause ring enhancing lesions. The aim of this study was to evaluate the imaging characteristics of ring enhancing lesions identified using Magnetic Resonance Imaging (MRI) at a single tertiary care center in Western India.

INTRODUCTION

Ring-enhancing lesions in the brain can be due to neoplastic and non-neoplastic diseases.^[1,2,3] Infectious, vascular and inflammatory

aetiologies are non-neoplastic and primary brain tumour and metastasis are important aetiologies for neoplastic disorders that cause ring enhancing lesions. The common differential diagnosis includes abscess, tuberculoma, neurocysticercosis, glioma, metastasis, and radiation necrosis. The most common aetiology for multiple ring enhancing lesions in developed countries is neoplastic, however, in a developing country like India, infectious diseases are more frequently seen than neoplasms. Neurocysticercosis and tuberculoma are endemic diseases in India. Both diseases may be similar in clinical manifestations and radiological appearances. It is necessary to diagnose and differentiate these cases for definitive management and for a better outcome. The aim of this study was to evaluate the

imaging characteristics of ring enhancing lesions identified using Magnetic Resonance Imaging (MRI) at a single tertiary care center in Western India.

MATERIALS AND METHODS

A retrospective observational case record based study was done over a period of 18 months from October 2021 to March 2023. Patients with recurrent seizures, visual impairment, focal neurological deficit, raised intracranial pressure (severe headache, vomiting, and papilledema), loss of sensorium, and posturing of limbs who underwent MRI evaluation during the study period were identified from the case records. These records were further filtered for the presence of ring enhancing lesions and the imaging characteristics were studied. Patients who did not have an MRI image or had contraindications for MRI or who were evaluated with MRI brain/MR angiography for symptoms not meeting inclusion criteria were excluded. The MRI studies were done using 1.5 Tesla MRI system (1.5T, achieva, Philips healthcare, best, The Netherlands). Imaging sequences included T1W, T2W, FLAIR, DWI, GRE images and pre and post contrast 3DT1W FS images. All MRI acquisitions were done by MR technicians experienced in MRI and evaluated by radiologist with more than 10 years of experience in the field.

Tuberculomas (Image 1): Tuberculomas are often multiple^[5] and are usually found at the corticomedullary junction. Histologically, tuberculomas consist of a central zone of caseous material surrounded by reactive epithelial cells, Langhans giant cells, polymorphonuclear cells, plasma cells and lymphocytes. On MRI, tuberculomas have a different appearance depending on their stage of evolution. At an early stage, the lesion is isointense on T1 and T2 weighted images, because it has abundance of giant cells and a capsule poor in collagen. At a later stage, the capsule of the lesion has become rich in collagen, and the lesion appears as a low signal intensity on T2WI, probably because of fibrosis, scar tissue and free radicals. Gadolinium enhanced MRI studies shows a pattern of ring like enhancement or areas of nodular enhancement or irregular nonhomogeneous enhancement.

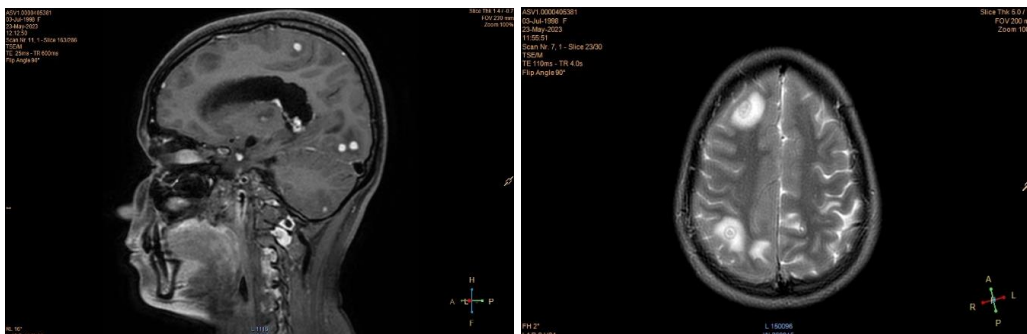


Image 1(a,b): Tuberculomas on MRI – a) Post contrast 3D T1 sag image shows multiple small ring enhancing lesions scattered in supra and infratentorial region in a patient of CNS tuberculosis. B) Axial T2WI shows T2 hyperintense lesions with hypointense ring and surrounding extensive edema.

Brain abscess (Image 2): Abscess formation occurs secondary to local suppurative infection within the brain parenchyma. Multiple foci may be seen particularly in immune compromised patients. The mass usually appears at the corticomedullary junction. On T1WI, the capsule appears as rim that is isointense to mildly hyperintense, whereas this may be either hypo or hyperintense on T2WI. With contrast injection, enhancement of the abscess capsule occurs as thin/ thick walled shaggy ring enhancement. The thinnest part of the contrast enhancement is located along the medial margin in the direction of the white matter. This supposedly represents the less well-developed areas of the collagen layer within the abscess capsule. The central cavity is usually more intense than the CSF (expand CSF), but less than normal brain tissue on T1WI. It is of increased intensity on T2WI. Edema that surrounds the abscess appears as high signal intensity on both T2WI and FLAIR. The presence of satellite lesions is a unique feature of cerebral abscess, but was not found in our patients. Do try to expand the short forms when used for the first time with the short forms in parenthesis.

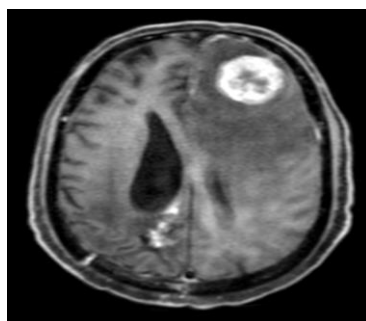


Image 2: Brain abscess in 38 year old drug addict. Axial post contrast T1WI reveals thick walled ring enhancing lesion in left frontal lobe with shaggy inner margin and extensive perilesional edema causing mass effect and midline shift.

Neurocysticercosis: Cysts is frequently found at the subarachnoid space, near the gray matter–white matter junction or in the basal ganglia, cerebellum, brainstem, cisterns, or ventricular system.^[5,6] On the basis of radiologic findings, neurocysticercosis is divided into five stages: non-cystic, vesicular, colloidal vesicular, granular nodular, and calcified nodular.

Non-cystic neurocysticercosis (active) is asymptomatic with negative imaging findings.

Vesicular neurocysticercosis has a cyst signal intensity similar to that of CSF on T1- and T2-weighted images. The cyst wall is well defined and thin, with little or no enhancement on gadolinium-enhanced images; scolex (hole with dot appearance) is iso- or hypointense relative to white matter on T1-weighted images; iso- to hyperintense relative to white matter on T2-weighted images and is best seen on proton-density–weighted images.

Colloidal vesicular neurocysticercosis (Image 3) – The cyst contents are hyperintense on T1- and T2-weighted images (proteinaceous fluid), cyst wall is thick and hypointense, pericystic edema (best seen on fluid attenuated inversion recovery images), with pericystic enhancement on gadolinium-enhanced images.

Granular nodular neurocysticercosis is similar to colloidal vesicular stage but with more edema and thicker ring enhancement.

Calcified nodular neurocysticercosis has hypointense nodules, no edema and no enhancement.

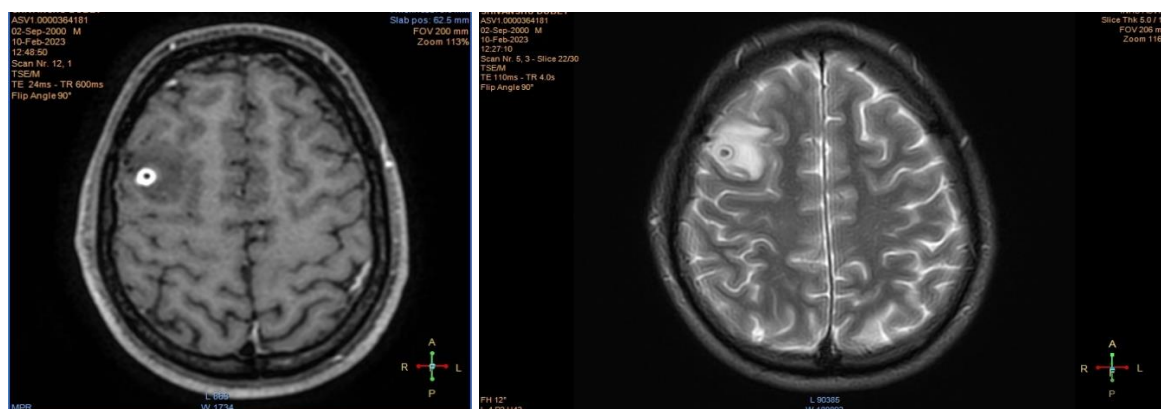


Image 3: MRI in neurocysticercosis: a) Post contrast 3DT1 axial image shows ring enhancing lesion in right frontal lobe with mild perilesional edema. B) Axial T2WI shows central hypointense nodule with surrounding hyperintensity and again hypointense rim followed by perilesional edema.

Brain metastases (Image 4): Parenchymal metastases are typically iso- to hypointense to brain on T1-weighted images, and variable in intensity on T2-weighted images. They tend to be roughly spherical in shape. Metastases often are surrounded by vasogenic edema, which is high signal on T2 and low on T1-weighted images. Edema can often be quite extensive relative to the size of the underlying tumor. Edema typically is confined to the white matter, sparing the overlying cortex. Since metastases lack the normal blood–brain barrier, they usually avidly enhance on postcontrast T1 images. The enhancement may be solid, or peripheral. A peripheral enhancement pattern is more likely as metastases grow in size and outstrip their available blood supply, leading to central necrosis. A close differential diagnosis of metastatic lesions is radiation necrosis.



Image 4: Metastasis in left frontal lobe from lung cancer. Axial T2WI reveals heterogenous hypointense lesion in left frontal lobe with perilesional edema. Right occipital lobe shows encephalomalacia from post radiation necrosis for a prior solitary brain metastasis.

RESULTS

One thousand five hundred and sixty-eight cases met the criteria for inclusion in the study. Sixty (3.83%) of these 1,568 patients had ring enhancing lesions in the MRI of the brain.

The majority of patients were between 20 to 40 years ($n = 36$, 60%) (Table1). Out of the analysis group, 43 patients were males and the rest were females as shown in Table 2.

Table 1: Distribution of patients with ring enhancing lesions in brain according to age.

Age group (years)	Percentage (%)
11-20(n=4)	6.6
21-30(n=20)	33.3
31-40(n=16)	26.6
41-50(n=8)	13.3
51-60(n=12)	20

Table 2: Distribution of patients with ring enhancing lesions in brain according to gender.

Gender	Percentage (%)
Male(n=43)	71.6
Female(n=17)	28.3

The diagnoses of ring enhancing lesions (Table 3) included neurocysticercosis(n=27), brain metastasis(n=16), tuberculoma(n=12), brain abscess(n=4) and subependymal cyst(n=1)

Table 3: Distribution of various diagnosis in patients with ring enhancing lesions in brain.

Diagnosis (n)	Percentage (%)
Tuberculoma (12)	20
Neurocysticercosis (27)	45
Brain abscess (4)	6
Brain metastasis (16)	26
Subependymal cyst (1)	1

The commonest site was the parietal lobe where 67% of the lesions occurred and the left side (n= 32, 53%) was more commonly involved.

In our study tuberculomas typically appeared as solitary ring enhancing lesions on gadolinium-enhanced T1-weighted images predominantly involving the gray-white matter interface (n=12, 20%). Neurocysticercosis appeared as subcortical ring enhancing lesions with perilesional oedema and showed susceptibility weighted artefacts within suggestive of scolices (n=27, 45%).

Metastatic lesions in our study involved bilateral brain parenchyma and showed heterogenous peripheral enhancement (n=16,26%).

DISCUSSION

Ring-enhancing lesions are often variable in size and have variable amounts of vasogenic edema around them. The ring-enhancing lesions are commonly found at the junction of grey and white matter, but they could also be found in the subcortical area or deep within the brain parenchyma or may be superficial. No single feature is pathognomonic. History taking, clinical examination and thorough review of prior reports is essential in evaluation of ring enhancing lesions and all this information along with patient demographics must be considered together to reach a conclusive diagnosis for ring enhancing lesions in the brain.

In our study we compared imaging characteristics of ring enhancing lesions on MRI. Neurocysticercosis was the most common etiology in this study.

Neurocysticercosis typically appeared at the subcortical region of the brain parenchyma. Ring enhancing lesions of NCC is typically shows vasogenic edema and is seen in colloidal vesicular and granular nodular stages with no restriction of diffusion on DWI. Our findings were comparable to a previous study done by Ravishankar et al.

In our study tuberculomas presented as solitary ring enhancing lesions at cortico- medullary region which shows restriction of diffusion on DWI with low ADC values. These findings were comparable to a previous study conducted by Verma R, et al.

Differentiation between tuberculoma and neurocysticercosis^[6,7] is a challenge. T2 hypointensity of the lesion with conglomerate lesions showing ring enhancement is a feature of tuberculomas, which was evident in one of our patients. Presence of ring enhancing lesions in the CSF spaces and basal cisterns is again a feature that favours tuberculomas. Presence of lesions in various stages of growth like vesicular, granular nodular and calcified stages can hint towards the ring enhancing lesion being neurocysticercosis. Also, T2 and GRE hypointense scolex favours neurocysticercosis as the diagnosis.

Brain abscess appeared as heterogenous signal intensity lesions on T1 and T2WI with thick shaggy ring enhancement and perilesional edema with mass effects. These patients would also give history of febrile episode with seizure and show raised counts on hematological evaluation.

The higher magnitude of metastatic lesions in our study may probably be due to the selection of the study sample from a tertiary care centre with good load of oncology cases. Patients

with brain metastasis may have a history of primary cancer, undergoing treatment and hence, history taking, clinical examination and thorough review of prior reports is essential in evaluation of ring enhancing lesions.

The single center-based nature of the study may be considered as a limitation that is balanced by the use of standardized uniform protocols for the study assessments. The sample size for the study is small but is a pragmatic reality considering the rarity of the diagnoses that were considered for the study.

Imaging characteristics of ring enhancing lesions in brain on other advanced modalities like MR spectroscopy^[8] or functional brain imaging can be considered for further studies

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