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"PATTERNS OF CONTRAST ENHANCEMENT ON MAGNETIC RESONANCE IMAGING (MRI) – USEFUL DIAGNOSTIC TOOL IN BRAIN AND MENINGEAL PATHOLOGIES"

Radio-Diagnosis

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ABSTRACT

Under normal conditions there will be no enhancement of the brain parenchyma in the central nervous system, because the brain is the only organ with a Blood -Brain barrier for proteins, inflammatory cells and also for contrast medium. The Blood-Brain Barrier can be damaged by various diseases, resulting in enhancement of the brain parenchyma and meninges. Understanding the pathophysiology and patterns of the different patterns of contrast enhancement is needed to narrow down the differential diagnosis and to arrive at the correct diagnosis3. This record based cross-sectional study was undertaken in the Department of Radio-Diagnosis, Gadag Institute of Medical Sciences, Gadag on 110 patients, showed peripheral rim/ring enhancement as a predominant pattern, intra axial heterogeneous enhancement as second, extra axial lesions showing solid homogeneous enhancement with focal dural enhancement being the third, diffuse pachymeningeal/ dural enhancement as fourth followed by leptomeningeal or sulcal enhancement and gyral/serpiginous enhancement pattern.

KEYWORDS

Contrast enhancement, MRI, Brain, Meninges.

INTRODUCTION

Magnetic resonance (MR) imaging has wide uses in the diagnosis, characterization, and planning of treatment for various brain pathologies. One particularly powerful feature of MR is the ability to exploit several different contrast mechanisms, thereby acquiring multiple views of the same tissue within a single examination[1].

The use of intravenous contrast on CT and MRI of the brain and spinal cord is an important diagnostic tool. The presence and pattern of contrast enhancement helps narrow and refine a differential diagnosis. Contrast enhancement results from 2 primary mechanisms: intravascular enhancement and interstitial enhancement. This study highlights the use of patterns of contrast enhancement in non-traumatic brain and meningeal pathologies on MRI brain.

MATERIALS AND METHODS

This was a record based cross sectional study conducted in the Department of Radio-Diagnosis, Gadag Institute of Medical Sciences, Gadag, on 110 patients referred to Department of Radio-Diagnosis, GIMS Gadag for Contrast Enhanced -Magnetic Resonance Imaging (CE-MRI) of the brain for various indications, from January 2022 to December 2023 for a period of 2 years. Any patient whose MRI Brain showed contrast enhancing lesion(s) and/or detected incidentally was included without any exclusion to specific age or gender. Any patient(s) with enhancing lesions in brain secondary to alleged history of trauma were excluded. 1.5 TESLA MRI scanner (Philips) was used to image the patients, in supine position using a head coil. Conventional routine brain MRI were performed followed by post contrast sequence. Post contrast axial T1 after manually injection of IV contrast medium (Gadolinium contrast) and dose of 0.1 mmol/kg of body weight. First, the conventional MR images were analyzed for extra axial spaces, ventricular system, basal cisterns, Cerebral parenchyma, Midline shift, Cerebellum, and Brain stem. Presence of enhancing lesions was documented in the form of number of lesions, predominant localization, morphology of lesions and intrinsic lesion architecture. Then, the lesions were grouped as per their patterns of contrast enhancement as elucidated by Smirniotopoulos et al in 2007[3] and Galina et al in 2023[2]: a) Linear pachymeningeal b) Leptomeningeal c) Gyral d) Nodular subcortical e) Ring enhancement (smooth thin or thick irregular or incomplete or open ring or Deep and periventricular) and f) Periventricular enhancement (Smooth and thin or Irregular and thick). Any enhancement pattern not following the above classification was grouped as unclassified. All the data collected were compiled as per patient and contrast enhancement pattern in Microsoft excel and descriptive statistics in the form of percentage, and mean were represented in the form of tables and charts. Data collected was also tabulated and distributed as per the aetiology. Various lesions demonstrating patterns of contrast enhancement were grouped accordingly. Final confirmatory diagnosis was affirmed as per Clinical / Histopathological features.

RESULTS

Total of 110 cases showed enhancement on MRI brain were studied. The study population included 59 males and 51females with the youngest patient in our study a 20 days old female child and the oldest patient 80 years old female.

The categorization of the lesions was done based on the different patterns proposed by the Smirniotopoulos et al in 2007[3] and Galina et al in 2023[2]. Few of the lesions that were not following the above proposed patterns, were categorized as unclassified. The distribution of the different contrast enhancement pattern is illustrated in Figure 1.

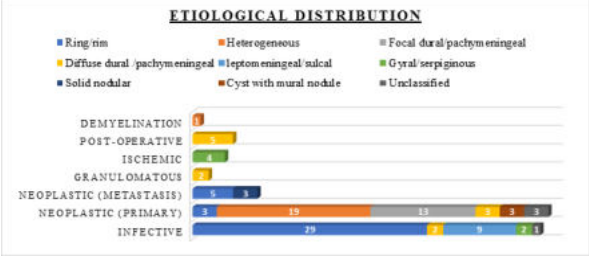

Figure I: Etiological distribution of different enhancement patterns

The predominant pattern recognized was peripheral rim/ring enhancement accounting for 33.6% (37 patients). In that, 32 patients showed smooth thin peripheral rim/ring enhancement, 3 patients showed thick, irregular peripheral ring enhancement, 2 showed target or targetoid enhancement patterns. Out of 32 patients, 21 showed multiple ring enhancing lesions, while 11 showed solitary lesions. The etiology as follows: 19 Tuberculoma (16 multiple; 3 solitary), 7 Abscess (all solitary), 5 Metastasis (4 multiple; 1 solitary), 1 NCC (multiple). Three thick peripherally enhancing lesions were GBM and multicystic GBM. Two target or targetoid lesions were toxoplasmosis and cyptococcosis.

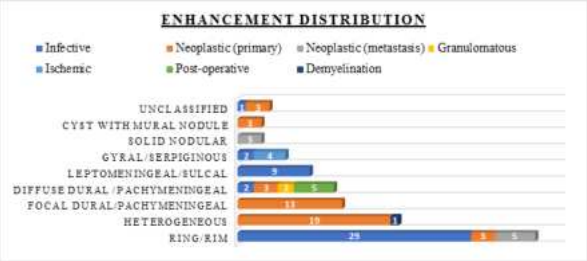

Figure II: Enhancement patterns distribution in different etiologies

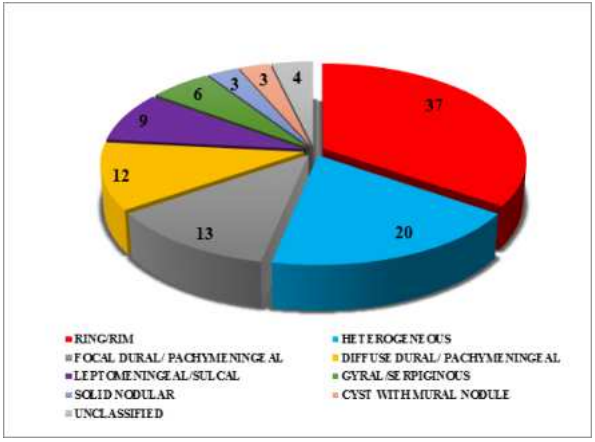

Figure III: Pie chart demonstrating distribution of the enhancement patterns

The second predominant pattern was intra axial heterogeneous enhancement amounting 18.18% (20 patients). Almost all the lesions were neoplastic lesions, most of them were malignant and recurrent lesions. The lesions were Glioblastoma multiforme, pituitary macroadenoma, medulloblastoma, malignant meningioma, acoustic neuroma, high grade gliomas, choroid plexus carcinomas, ATRT, DNET. One patient with Tumefactive demyelination also showed heterogenous enhancement.

Extra axial lesions showing solid homogeneous enhancement with focal dural enhancement occupied the third predominant pattern accounting 14.54% (16 patients). 13 out of 16 patients had meningiomas, 1 had pituitary macroadenoma, 1 had schwannoma and 1 had NF-2 (neurofibromatosis-2) comprising multiple meningiomas, schwannomas and spinal cord neurofibromas. Out of 13 meningiomas, one patient multiple meningiomatosis. All these meningiomas showed typical focal dural enhancement i.e, "Dural tail sign".

Diffuse pachymeningeal/ dural enhancement pattern came as fourth predominant pattern in our study – 12 patients (10.9%). In these, 5 patients were post operative cases who showed diffuse pachymeningeal enhancement at the surgical site. 2 were En-plaque type of meningiomas, 3 were tuberculous meningitis and 2 were granulomatous lesions. Out of 3, 2 tuberculous meningitis showed nodular pachymeningeal enhancement and one case of Neurosarcoidosis showed nodular pachymeningeal enhancement.

Fifth pattern was leptomeningeal or sulcal enhancement with 8.18% - 9 patients who were cases of meningitis and meningoencephalitis. 3 of them showed only leptomeningeal/ sulcal enhancement. The rest 6 showed combinations of leptomeningeal, pachymeningeal and gyral enhancement.

6 patients showed gyral or serpiginous enhancement pattern was the sixth predominant pattern. Out of those, 4 were infarcts (3 were hemorrhagic infarcts) and 2 were encephalitis.

Three patients showed multifocal subcortical disciform nodular enhancement who were the case of cerebral metastasis. Three showed cysts with solid enhancing mural nodule pattern, those were pilocytic astrocytomas and schwannomas. Four cases were categorized as unclassified, as these four did not follow any proposed patterns of enhancement[2,3]. Those were cases of encephalitis, low grade glioma and craniopharyngioma.

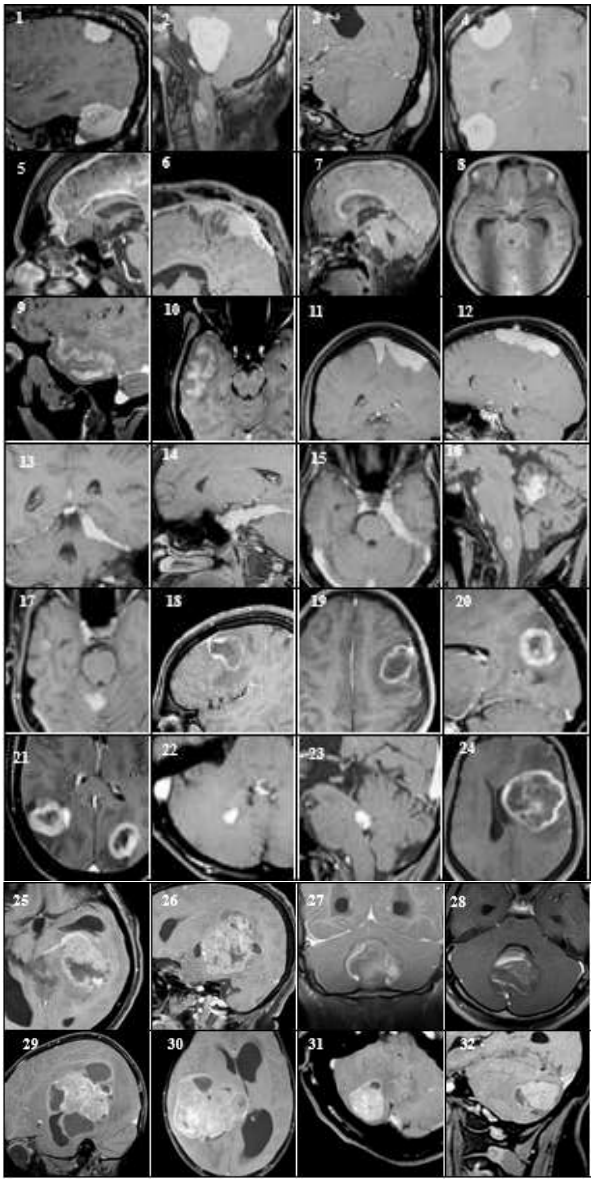

Figure-IV: 1-4: Case of Neurofibromatosis type II – Multiple extra axial, dural based meningiomas showing dural tail signs (Focal dural enhancement), Right CP angle schwannoma, Occipital scalp neurofibroma (3) and Cervical spinal neurofibroma (2). All lesions showing intense homogeneous enhancement. 5-6: Post operative case of meningioma showing diffuse pachy and leptomeningeal enhancement. 7-8: Meningitis – diffuse sulcal and cisternal (leptomeningeal) enhancement. 9-10: Hemorrhagic infarct – gyral or serpiginous enhancement. 11-12: En-plaque meningioma – diffuse pachymeningeal enhancement. 13-15: Neurosarcoidosis – nodular pachymeningeal enhancement. 16-17: Tuberculoma – multiple ring enhancing lesions(conglomerate). 18-19: Abscess – single peripheral ring enhancing lesion. 20-21: Toxoplasmosis (HIV positive) – Multiple target like ring enhancing lesions. 22-23: Metastasis - Solid nodular – disciform enhancement. 24: GBM – thick, irregular peripheral ring enhancement. 25-26: Choroid plexus carcinoma – heterogeneous enhancement. 27-28: Medulloblastoma - heterogeneous enhancement. 29-30: ATRT -heterogeneous enhancement. 31-32: Pilocytic astrocytoma - cyst with nodule.

DISCUSSION

The analysis of results from our study shows that specific enhancement patterns can pinpoint a definitive diagnosis in most cases as different aetiologies have different patterns on contrast enhanced MRI[4]. Contrast enhancement results from 2 primary mechanisms: intravascular enhancement and interstitial enhancement. Interstitial enhancement depends on abnormal permeability of the blood-brain barrier (BBB) and is seen in a variety of neoplastic, inflammatory, and

infectious pathologies. This chapter features a diversity of cases that illustrate interstitial enhancement, organized by extra-axial and intra-axial enhancement[2].

Extraaxial pachymeningeal enhancement may arise from various benign or malignant processes, including transient postoperative changes, intracranial hypotension, neoplasms such as meningiomas, metastatic disease (from breast and prostate cancer), secondary CNS lymphoma, and granulomatous disease[3]. Lymphoma, leukemia can also be associated with dural enhancement[2].

Leptomeningeal enhancement is usually associated with meningitis, which may be bacterial, viral, or fungal. The primary mechanism of this enhancement is breakdown of the blood brain barrier without angiogenesis. Neoplasms may spread into the subarachnoid space and produce enhancement of the brain surface and subarachnoid space, a pathologic process that is often called "carcinomatous meningitis"[3].

Intra-axial enhancement can be further characterized by location—cortical, subcortical, periventricular, and ependymal—and pattern, such as partial or complete rim enhancement[2].

Gyral Enhancement - Superficial enhancement of the brain parenchyma is usually caused by vascular or inflammatory processes and is only rarely neoplastic. Vascular causes of serpentine (gyral) enhancement include vasodilatation after reperfusion of ischemic brain, the vasodilatation phase of migraine headache, posterior reversible encephalopathy syndrome (PRES), and vasodilatation with seizures[3]. Singular or multifocal nodular enhancing lesions in cortical and subcortical parenchyma may represent hematogenously spread metastases or thromboemboli[2].

Rim-enhancing lesions are more commonly subcortical than superficial. Deep white matter lesions, especially with mass effect or surrounding vasogenic edema, are most likely primary neoplasms or abscesses. Multiple cortical and subcortical smoothly rim-enhancing lesions are usually infectious in aetiology. The rim of reactive tissue is usually thin (2–7 mm), uniformly convex, and smooth on both the outer and inner aspects in cerebral abscess[3].Imaging features of a necrotic neoplasm include a thick irregular ring with a shaggy inner margin, multilocular and complex ring patterns, and a wall that is thicker than 10 mm (at least in some areas). Cerebritis, radiation necrosis, contusion, or subacute infarction can also show surrounding rim enhancement[2].

Fluid secreting neoplasms may demonstrate an incomplete ring of enhancement, because part of the margin surrounding the fluid is neoplastic and part is nonneoplastic (compressed or gliotic brain tissue). Occasionally, thin (2-mm) rim enhancement, representing gliosis and not neoplastic tissue, can be seen in a pilocytic astrocytoma[3].

Incomplete or "C-shaped" rim enhancement can be associated with active demyelination which is also the most common cause of periventricular enhancement. Neoplasms such as primary CNS lymphoma and primary glial tumors and infectious processes such as abscesses, ependymitis, or ventriculitis may also present with periventricular enhancement. Some enhancement patterns are highly suggestive of a specific diagnosis—for example, linear radial periventricular enhancement in GFAP astrocytopathy[2].

CONCLUSION

Under normal conditions there will be no enhancement of the brain parenchyma in the central nervous system, because the brain is the only organ with a Blood -Brain barrier for proteins, inflammatory cells and also for contrast medium. The Blood-Brain Barrier can be damaged by various diseases like: Inflammation or infection (abscess), Demyelination. Ischemia, Tumors like gliomas, lymphomas and metastases. Understanding the different patterns of enhancement in CNS disease will improve image assessment and correct radiological diagnosis, and ultimately the management of the patients.

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