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Study on clinical and radiological follow up of ring enhancing lesions in brain

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Abstract
Rings with Eccentric Dots showing scolex however was found in 73.3% case of our NCC cases. Dense Rings with Irregular Margin are found mostly due to malignant causes. Target sign (ring enhancement with central calcification) pathogomic of tuberculomata of brain was present in 2.5% of cases in our series,

We observed that infective pathologies (71.8%) were the most common etiology in patients with ring enhancing lesions of the CNS. Neoplastic etiology (26.9%) was the next common etiology causing ring enhancing lesions of the CNS. NCC and TB were the most common infections causing ring enhancing lesions of the CNS. Among Neoplastic causes, Primary Brain tumor (predominantly GBM) most common infections causing ring enhancing lesions of the CNS.

In our study Surrounding edema (84.61%), is the most common perilesional parenchymal changes followed by midline shift in 51.2%, Meningeal enhancement in 34.61% and Hydrocephalus in 26.9%. Mean ADC Value from the cavity and wall of the lesions can easily distinguish from neoplastic and non-neoplastic ring lesions. Proton MR spectroscopy is useful for the differentiation of neoplastic and non-neoplastic brain lesions. Among infective etiologies 86.49% shows clinical improvement and 83.79% radiologically. Among infective etiologies 13.51% shows non improvement clinically and 16.21% shows non improvement radiologically. Among NCC at 6-7 months follow up on treatment, 17/21(80.95%) cases showed both clinical and radiological improvement.2 cases required earlier MRI due to clinical worsening.2 cases showed radiological worsening at 6-7 months follow up on treatment despite of clinical improvement.

Among Tuberculomas at 6-7 months follow up on treatment, 13/16(81.25%) cases showed both clinical and radiological improvement.3 cases required earlier MRI due to clinical worsening. 3 cases showed non resolution at 6-7 months follow up on treatment despite of clinical improvement. Overall among NCC and Tuberculomas at 6-7 months follow up on treatment 30/37 showed both clinical and radiological concordant improvement. There was a concordance between clinical worsening and lesion persistence in 2/37 caeses both of were in the Tuberculoma category. At 6-7 months follow up 5/37 fell in to the group of non-concodance between clinical and imaging data, the patients being improved clinically but imaging showing persistence of lesions.

Keywords: radiology, brain, tuberculomas, NCC

Introduction

Brain abscess can be medically managed by antibiotics or CT/MRI-guided drainage while neoplastic lesions usually require biopsy, surgical resection and metastatic workup. Progress in immunodiagnostic methods and stereotaxic technology has facilitated definitive diagnosis of the lesions [1-4] In developing countries often it is not possible to perform brain biopsies because of limited neurosurgical and neuropathological facilities [3] The Introduction of Magnetic Resonance Imaging (MRI) has created many important advances in the detection and characterization of brain lesions and is considered to be the state of the art technology in the evaluation of the brain. The detection rate of most types of brain lesions by MRI exceeds 90%, compared to 77% for CT - without the invasiveness or risk of iodinated intravenous contrast agents or the inherent problem of the radiation effect of X-rays. These safety features make MRI especially advantageous for the pediatric and elderly populations.

MR spectroscopy is a potential tool for differential diagnosis between brain abscesses and non-infectious lesions such as primary brain tumor, lymphoma, brain metastasis, and tuberculoma. Magnetic resonance spectroscopy (MRS) provides information about the

possible extent and nature of changes on a routine MRI scan by analyzing the presence and/or ratio of tissue metabolites such as NAA, creatine, choline, and lactate etc. [5] In this study, the distinguish between neoplastic and non-neoplastic peripheral enhancing ring like lesions on contrast MRI was carried out and MR spectroscopy in the evaluation of various ring enhancing lesions in the brain was also documented.

Materials and Methods

The present study was conducted in the Post-doctoral Department of Neurology at S.C.B. medical college & Hospital, Cuttack during the period of between August 2013-November 2015.

Patients admitted in different specialities with single or multiple ring-enhancing lesions found on CNS neuroimaging were analyzed in view of their clinical presentation and investigative profile. All patients underwent routine haematological, biochemical, and serological tests including HIV, VDRL, Chest X-ray and a Mantoux test.

In all cases MRI of Brain with Gadolinium contrast (T1w, T2w, FLAIR, DWI, GRE) was done in our Radiology department. Conventional MR examinations were performed with a 1.5-T imager capable of echo-planar imaging (HDXT). Conventional MR images were obtained with transverse proton density– and T2-weighted sequences (5400/122/2 [TR/TE/excitations]; matrix, 192 x 256; field of view, 24 X 24 cm2; section thickness, 5 mm; section gap, 2.5 mm), transverse T1-weighted sequences (1800/17/ 2; matrix, 192 x 256; field of view, 24 X 24 cm2; section thickness, 5 mm; section gap, 2.5 mm), and transverse contrast-enhanced T1-weighted sequences (0.1 mmol/kg of gadopentetate dimeglumine [Magnevist; Schering, Berlin, Germany]). The contrast-enhanced images were obtained following DW imaging. Isotropic DW images were obtained in the transverse plane by using a single-shot echo-planar spin-echo pulse sequence (4650/78/1; field of view, 24 X 24 cm2; section thickness, 5 mm; section gap, 2.5 mm),with four b values (0, 333, 666, 1000 s/mm2). The total duration of the diffusion-sensitizing gradients was 80 m. To obtain isotropic diffusion weighting, a gradient scheme was used according to Heid and Weber. The maximum diffusion gradient was 26 mT/m. ADC map was obtained in selected patients. The ADC maps were calculated and the ADC value of the cystic or necrotic portion was measured in a region of interest of at least 1 cm2. On visual inspection, the signal intensities of the lesions on the DW images and on the ADC maps were interpreted relative to the contralateral brain parenchyma. GRE image was obtained with (720/20/2 [TR/TE/excitations]; section thickness, 5 mm; section gap, 2.5 mm).

MR Spectroscopy was done in selected cases. Metabolites which were detected in brain tissue include choline (Cho), creatine (Cr), N-acetylaspartate (NAA), lactate, mioinositol (MI), glutamine/glutamate, lipids and amino acids. Brain lesions contain abnormal quantities of these metabolites compared with normal brain tissue.

CSF analysis was done where feasible including cell count, cell type, Gram stain, AFB stain, culture and sensitivity, protein and sugar estimation were done. Special stains like India Ink were done for fungal detection and fungal cultures. CSF –RT PCR for Tuberculosis to be done in selected patients. Analysis for neurocysticercal antigens was done using Cysticheck kits (ELISA technique). A RMS 16 channel EEG machine was used to record EEG's.

Probable etiological diagnoses were based on the presence of supportive findings detected after clinical evaluation and above mentioned battery of investigations. The diagnosis confirmed in few cases whose biopsy and histopathology of the lesion was done and rest of the cases final diagnosis remained probable because the patients histopathological verification was not obtained. Patients were provided appropriate symptomatic treatment (corticosteroids, mannitol, antiepileptic drugs and/or analgesics if required). Appropriate specific treatment according to the suggested diagnosis was provided. Some of the above mentioned laboratory investigations were periodically repeated during hospitalization and on outdoor follow-up to monitor the disease activity on modifying the ongoing treatment.

The Data collected from cases were recorded in a Master chart. Data analysis was done using appropriate statistical tests and graphs.

Table 1: Clinical Follow Up After 6-7 Months

Etiology	No of cases	improvement	Detoriations	Death	Lost Follow Up
NCC (n=30)	21	18 (85.71%)	3	2	7
Tuberculoma (n=21)	16	14	2	4	1
Glioma (N=12)	5	1	4	7	
Metastasis (n=8)	1	0	1	7	
Abscess n=4	2	2		1	1
Medulloblastoma				1	
Toxo n=1				1	
Hematoma n=1	1	1			

Out of total 78 patients 23 patients died within 6months of follow up. Most of these cases were of Primary Brain tumours and metastasis. Two patients of NCC were died probably due to Cysticercal encephalopathy. Four Patients of CNS Tuberculomas were died, out of 2 cases because of Oppertunistic infections associated with HIV. Another two patients were died probably may had drug resistant Tuberculosis. Each of Seven cases of Primary CNS Malignancy and CNS metastasis were died within 6months of follow up. Out of Four CNS Pyogenic abscess patients 1patient had CCHD, TOF and was died because of CCF. Rest of 3 operated patients all shows clinical improvements. One case of CNS toxoplasmosis was died during hospital stay. He died probably because of Oppertunistic infections associated with HIV. One case of CVA, frontoparietal lobe hematoma patient shows clinical improvement. Seven cases of NCC, 1case of Tuberculoma, 1case of Brain abscess patients lost follow up after 6month. Rest of the cases who were followed up were given in the (Table 1).

Table 2: Radiological Follow UP after 6-7 Months

Etiology	No of cases followed up	Complete resolution	Partial resolution	Non resolution	Calcification of lesions
NCC (n=30)	21	2(9.5%)	15(71.4%)	3(14.28%)	1(4.7%)
Tuberculoma (n=21)	16	5(31.25%)	8(50%)	3(18.75%)	
Glioma (N=12)	5	1	4		
Metastasis (n=8)	1	0		1	
Abscess n=4	2	2			
Hematoma n=1	1	1			

Out of total 78 cases complete resolution of lesion was seen in 11 (14.10%), partial resolution (no new lesion and resolution of surrounding brain parenchymal changes) in 27(34.61%), unresolved (no change in lesion size, shape or surrounding) in 7(8.97%). Death in 23 cases (29.48%). Calcification was noted in inflammatory pathologies. Calcified scolex was seen in neurocysticercosis (NCC) in 4.7% of NCC (Table 2).

Table 3: Clinico radiological Corelations in radiologically improved patients with tuberculomas/ NCC

Improved on imaging	No. of Cases	Clinical improvement Yes	Clinical improvement No
YES	31/37	30 (96.7%)	1 (3.3%)
NO	6/37	2 (33.3%)	4(66.7%)

By Chi square test: p= 0.05

In this study 3.3% cases who were improved radiologically had no clinically improvement which was significantly less as compared to 66.7% with no radiological improvement (Table 3).

Table 4: Clinico Radiological corelations in clinically improved patients with tuberculomas/ NCC

Improved Clinically	No. Of Cases	Radiological Improvement Yes	Radiological Improvement No
YES	32/37	29 (90.6%)	3(9.4%)
NO	5/37	2 (40%)	3(60%)

In this study 90.6% improved clinically had radiological improvement as compared to 40% cases who were not improved clinically (Table 4).

Discussions

In our cases series multiple ring lesions predominates over solitary ring enhancing lesions. Solitary ring enhancing lesions found in 25(32.05%) cases where as 53 (67.9%) cases had multiple lesions.

However preminance of solitary ring enhancing lesions were found in studies by Wadia *et al*. [6] in 1987, and Manjari *et al*. [7] in 2011. Similarly Rudresh K *et al*. [8] in 2008 from Karnataka found majority of the lesions were single ring enhancing lesions comprising about 91% of total cases in his study population. Study by Krishna *et al* [9] in 2013 on Twenty-five patients of intracranial small ring (multiple / single) enhancing lesions noted single ring enhancing lesions in 76%, multiple in only 24%.

This discordant may be explained due to a small population of cases were studied. However this is in concordance with previous studies done at R K Desai, Garg *et al* [10] showed Multiple enhancing computed tomography (CT) brain lesions are common as compaired to isolated ring enhancing lesions. *Mahato et al.* [11] studided 40 patients of CNS ring enhancing lesions, out of them 30 cases (75%) had multiple ring enhancing lesions. This is in concordance to our study.

Sites of lesions in MRI of brain and spine

The most common site of involvement both in multiple as well as solitary ring enhancing lesions was frontal lobe in 56 cases (71.79%) followed by parietal lobe in 41 cases (52.56%) and temporal lobe in 21 cases(26.9%).

This was in concordance with a study done by B. Murali *et al*. [12] which shows the most common site of involvement was frontal lobe. Another study by Krishna *et al*. [9] in 2013 also shows commonest site of involvement was frontal lobe encountered in 36% followed by parietal lobe in 12% of cases.

However in contrary Rudresh K *et al*. [8] in 2008 found majority of the lesions with the parietal lobe (45.2%) being the commonest site of occurrence followed by Frontal lobe lesions constituted in 32.9%, and occipital lobe lesions in 15%. Temporal lobe lesions were the least forming 6.9% of cases.

Sizes of lesions in MRI of brain and spine

In our series the small lesions of sizes less than 2 cm were the most common 43 cases (55.14%), followed by large size lesions in 24 cases (30.76%). Small lesions mostly are of infective pathologies like NCC and tuberculomas. Large lesions are due to combined infective and non infective pathologies like Abscess and Tumours. Mixed lesions are mostly due to CNS metastasis.

Study done by M. L. Ravindranath [13] in 2007 small lesions of sizes less than 2 cm were the most common 169 cases (61%) in his series followed by Medium size lesions 2 cms to 3 cms in 82 cases (30%) and Large size lesions > 3 cms in 25 (9%). Similarly study by Tae Kyoung Kim *et al*. [14] found predominance of rings of size less than 2cm in their case series. Another study by Rudresh K *et al*. [8] in 2008 also found majority of the lesions were less than 20 mm in size which was in concordance to our study.

Types of ring enhancement in MRI of brain and spine

Thin rim enhancements are the commonest 62.8% in our case series. This is attributable by mostly NCC, tuberculomas and brain abscess. Study done by *M. L. Ravindranath.* [13] in 2007 noted thin Rim Enhancement in 109 (43%) followed by Dense Ring Enhancement in 94 (37%) of their case series.

Coalescence of the ring lesions were noted in 5% of cases in our study. However McCormick *et al*. [15] noted Coalescence of the ring lesions in 55% of their patients in a study done in 1982, where as Bhargava S. Tandon P.N. *et al.* [16] reported 8% of such cases in their study. Further Bharghava S *et al* quoted that coalescent lesions were present in mature tuberculomas and they were a conglomeration of multiple rings or discs resulting in the irregular contour. Thus is can be inferred that our series contained a lesser number of

mature tuberculoma patient. Target sign (ring enhancement with central calcification) was present in 2.5% of cases in our series. Vandyke and Welchman [17] postulated Target sign was pathgnomic of tuberculoma of brain. Our study is in concordance to a previous study done by *M. L. Ravindranath* [13] in 2007 who found Coalescence of the ring lesions and Target sign were noted in 4 % and 2% of their case series respectively. Rings with eccentric dots showing scolex which was pathognomonic of NCC, however was found in 73.3% case of our NCC cases.

Conclusions

Our study establishes the role of NCC and TB as the leading cause of ring-enhancing lesions in the Indian setup as compared to tumors in the Western world. It might serve a basis for early recognition and intervention in these patients. A large number of infectious and non-infectious diseases can cause multiple ring-enhancing lesions of the brain. We reviewed the most common diseases in our setup. The most common etiologies were that of TB, parasitic (especially neurocysticercosis), primary brain tumors and Gliomas and metastases, cerebral abscess. Other rare causes include cerebrovascular stroke.

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