

Clinical and Neuroimaging Profile of Ring Enhancing Lesions in Children Presenting to Department of Pediatrics, Government General Hospital, Kakinada

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Abstract

Original Research Article

Ring enhancing lesions are one of the most commonly encountered neuroimaging abnormalities in patients presenting with acute seizures in tropical countries. A variety of infective and noninfective processes display a pattern of ring enhancement on neuroimaging, which often prohibits a reliable diagnosis and clinical correlation is essential. Various causes of ring enhancing lesions are neurocysticercosis, tuberculoma, pyogenic abscess, toxoplasmosis, cavernous angioma, primary or metastatic tumours of brain. Aim is to study the various clinical presentations of ring enhancing lesion, etiology, distribution and size of ring enhancing lesions and their outcome. This is a prospective observational study done over 18 month's i.e, from January 2012 to June 2013, in the department of pediatrics, Government general hospital, Kakinada. A total of 74 children less than 14 years of age with ring enhancing lesion in neuroimaging are enrolled in the study. Neurocysticercosis was diagnosed based on Del Brutto diagnostic criteria. Most common cause of ring enhancing lesion was Neurocysticercosis (70.27%) followed by Tuberculoma (27.7%). Most common clinical manifestation was seizure (91%). Partial seizures were more commonly seen than generalised seizures. No gender difference was seen in the prevalence of Neurocysticercosis and Tuberculoma. Both Neurocysticercosis and Tuberculoma were common in children above 5 years of age (90%). Single ring enhancing lesion was common, both in Neurocysticercosis and Tuberculoma. Larger lesions were more commonly associated with Tuberculoma. Response to therapy was similar both in children who were given 8 day Albendazole therapy versus 28 day Albendazole therapy.

Keywords: Ring enhancing lesion, Neurocysticercosis, Tuberculoma, Clinical profile, Neuroimaging findings, Response to therapy, Albendazole.

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INTRODUCTION

Ring enhancing lesions (REL) are one of the most commonly encountered neuroimaging abnormalities in patients with acute seizures. A variety of infective and non infective processes display a pattern of ring enhancement on neuroimaging, the common causes being neurocysticercosis (NCC) and tuberculoma particularly in tropical countries. From the previous studies in various countries, the etiologies for REL were categorized into 2 main groups: the first group was considered as 'Dying cysticercus'[1], suggested to be the most common cause with a benign clinical course and usually resolve spontaneously after a brief period of few weeks to 3 months[2-4]. The second group was uncommon; needed more aggressive diagnostic approaches and included tuberculoma, pyogenic abscess, toxoplasmosis, cavernous angioma and primary or metastatic tumours of brain. The size,

site, number of lesions, pattern of contrast enhancement and associated findings like perilesional edema facilitate radiological diagnosis. Even though several diagnostic criteria are proposed to diagnose NCC and to differentiate it from neurotuberculoma, a reliable diagnosis is difficult and clinical correlation is essential and they often pose diagnostic and therapeutic challenges. Hence this study is taken up to evaluate the etiology, clinical presentation and outcome after treatment of RELs.

MATERIALS & METHODS

The present study is a prospective observational study done over 18 months i.e, from January 2012 to June 2013 in the department of pediatrics, Government General Hospital, Kakinada. All children <14 years with ring enhancing lesions in neuroimaging were included and children with other

neurological abnormalities like cerebral palsy and malformations were excluded from the study. Age, gender and clinical features of children with REL were recorded. The site, size and number of ring enhancing lesions were noted. Neurocysticercosis was diagnosed based on Del Brutto diagnostic criteria. Children with ring enhancing lesion after establishing a diagnosis are treated accordingly with albendazole, antiepileptics, steroids or antitubercular drugs and were regularly followed up for 1 year. Statistical analysis was done using SPSS software version 20. Institutional ethics committee approval was taken and informed consent was also taken from the parents of children.

Revised diagnostic criteria of neurocysticercosis [5]

Absolute

- Histological demonstration of parasite
- CT or MRI showing cystic lesions with scolex
- Fundoscopic visualization of parasite

Major

- Lesions suggestive of neurocysticercosis on CT or MRI
- Positive serum EITB
- Resolution of cyst after therapy.
- Spontaneous resolution of single enhancing lesions

Minor

- Lesions compatible with neurocysticercosis on CT or MRI
- Suggestive clinical features.

- Positive CSF ELISA
- Cysticercosis outside CNS.

Epidemiologic

- Household contact with Taeniasolium infection.

Revised degrees of certainty for the diagnosis of neurocysticercosis

Diagnostic certainty

Definitive

- Presence of one absolute criterion or
- Presence of two major plus one minor and one epidemiologic criterion.

Probable

- Presence of one major two minor criteria or
- Presence of one major plus one minor and one epidemiologic criterion or
- Presence of three minor plus one epidemiologic criterion.

RESULTS

The most common cause of REL in the study population was neurocysticercosis seen in 70.27% of the children followed by tuberculoma, seen in 27.7% of the children. One child had cerebral abscess and one child had intra cerebral tumour. Details of etiology of REL are given in table 1.

Table-1: Etiology of ring enhancing lesions

Etiology	n = 74
NCC	52(70.27%)
Tuberculoma	20(27.7%)
Abscess	1(1.35%)
Tumors	1(1.35%)

Table-2: Clinical presentations of ring enhancing lesions

Etiology	NCC(n=52)	Tuberculoma(n=20)	Abscess(n=1)	Tumors(n=1)
Asymptomatic	1(1.92%)	0	0	0
Seizures	49(94.23%)	18(90%)	0	1
Headache	13(25%)	8(40%)	1	1
Vomiting	9(17.3%)	6(30%)	0	1
Fever	0	5(25%)	0	0
Papilledema	1(1.92%)	4(20%)	1	0
Hemiparesis/hemiplegia	2(3.84%)	1(5%)	0	0
Cranial nerve palsies	1(1.92%)	0	0	0
Falls	3(5.76%)	1(5%)	0	0
Comatose	1(1.92%)	1(5%)	0	0
Olfactory hallucinations	1(1.92%)	0	0	0
Ataxia	1(1.92%)	0	0	0
Torticollis	1(1.92%)	0	0	0
Loss of speech	2(3.84%)	0	0	0
Raised ICT	0	0	1	0
Death	0	1(5%)	1	0

Most common clinical manifestation was seizure seen in 94.23% of children with NCC and 90% of children with tuberculoma. Next common presentation was headache seen in 25% of children with NCC and 40% of children with tuberculoma. Third common presentation was vomiting (17.3% in NCC, 30% in tuberculoma). Papilledema was seen in 1.92% cases of NCC and 20% cases of tuberculoma. The details of various clinical presentations of REL are given in table no 2.

67.35% of children with NCC presented with partial seizures (simple or complex).66.67% of children with tuberculoma presented with partial seizures (simple or complex).8.3 % of children with REL presented with status epilepticus & 12.5 % presented with generalised tonic clonic seizures. Different types of seizures and their incidence in NCC and tuberculoma are given in table no 3.

Table-3: Type of seizure

Etiology	Status	Simple partial	Complex partial	Partial with secondary generalization	GTCS
NCC	3(5.76%)	26(50%)	7(13.46%)	7(13.46%)	6(11.53%)
Tuberculoma	2(8.3%)	7(35%)	5(25%)	0	4(12.5%)

Out of 52 children with NCC males constitute 57.69%.Out of 20 children with tuberculoma males constitute 42.3%..There was no statistically significant difference in gender distribution in NCC and tuberculoma.94.24% children with NCC and 90% of children with tuberculoma were > 5 years of age. This difference in age distribution is statistically significant (p value <0.05).67.3% of children with NCC and 70 % of children with tuberculoma had single lesion in

neuroimaging. Most common site of REL was frontal lobe (48.57% in NCC and 57.14% in tuberculoma). Parietal lobe was the next common site of ring enhancing lesion.76.92% of NCCs and 35% of tuberculomas were <10 mm in size, whereas, 23.07% of NCCs and 65% of tuberculomas were >10mm in size.The details regarding the similarities and differences between NCC and tuberculoma are given in the table 4.

Table-4: Characteristics of NCC & Tuberculoma

Characteristics	NCC (n=52)	Tuberculoma (n=20)
1.Gender distribution	p value – 0.2637	p value- 0.1797
Male	30 (57.69%)	7 (35%)
Female	22 (42.3%)	13 (65%)
2. Age wise distribution (yrs)	p value – 0.0001	p value- 0.0078
1-5	3 (5.76%)	2 (10%)
6-10	24 (46.15%)	5 (25%)
11-14	25 (48.07%)	13 (65%)
3. Number of lesions		
Single	35 (67.3%)	14 (70%)
Multiple	17 (32.7%)	6 (30%)
4. Site of lesion		
Frontal	25 (48.57%)	12 (57.14%)
Parietal	20 (37.14%)	7 (35.71%)
Temporal	1 (2.85%)	0
Occipital	3(5.71%)	0
Other	3 (5.71%)	1 (7.14%)
5. Size of lesion	p value – 0.001	p value – 0.001
< 10 mm	40 (76.92%)	8 (40%)
>10 mm	12 (23.07%)	12 (60%)

At the end of 1 year 51.9% of the lesions in NCC either decreased in size or disappeared.34.61% lesions in NCC underwent calcification.85% of the

lesions in tuberculoma, disappeared or decreased in size at the end of one year. The details regarding the fate of the lesion at the end of one year is given in the table 5.

Table-5: Fate of the lesion at the end of 1 yr

Etiology	Disappeared	Decreased	Increased	Calcified	Lost to follow up
NCC	12 (23.07%)	15 (28.84%)	1 (1.92%)	18 (34.61%)	6 (11.53%)
Tuberculoma	8 (40%)	9(45%)	0	0	3 (15%)

15 children with NCC received 8 day albendazole treatment and 37 children received 28 day

albendazole treatment.60 % children in the 8 day treatment group had disappearance or decrease in the

size of lesion at the end of 1 year. 62.16% of children in 28 day treatment group had disappearance or decrease in the size of lesion at the end of one year. 33.33% children in 8 day treatment group had calcified lesions where as 24.32% of children who received 28 day

albendazole treatment had calcification of lesions at the end of one year. The details of fate of lesion with 2 different schedule of albendazole therapy are given in the table 6.

Table-6: Fate of lesion with Albendazole therapy

Albendazole	Disappeared	Decreased	Calcified	Lost to follow up
8 days (n=15)	4 (26.6%)	5 (33.3%)	5 (33.3%)	1 (6.66%)
28 days (n=37)	10 (27.02%)	13 (35.13%)	9 (24.32%)	5 (13.51%)
	p value -0.856	p value -0.631	p value -0.607	

DISCUSSION

In the present study out of 74 children with REL, 70% had NCC and 27 % had tuberculoma. One child had pyogenic brain abscess and another child had intracerebral tumour. The etiology of REL is different in Mahato *et al.* study [6] which showed tuberculomas and tumours as the common causes of REL (study done in adults).

There was no significant gender difference in the occurrence of NCC and tuberculoma, which is similar to study by prathiba singhi *et al.* [7]. In the present study mean age of study population was 10 years. In prathiba singhi *et al.* study [7] mean age of study population was 8.02 years. 95% of children with NCC and 90% of children with tuberculoma were above 5 years of age. Both NCC and tuberculomas were uncommon in children under 5 years of age.

In the present study seizure was the most common clinical presentation of REL (91.89%). This is similar to prathiba singhi *et al.* study. Most common type of seizure noted was partial seizure in both NCC and tuberculoma in contrast to adult study by Del brutto *et al.* where GTCS was the commonest seizure type. 12 children who presented with 1st episode of GTCS also had REL. This shows the importance of evaluating all children with 1st episode of partial as well as GTCS for REL. Second common manifestation was headache (31.08%), followed by vomiting (21.6%) similar to prathiba singhi *et al.* study. So, the possibility of ring enhancing lesion should be kept in mind while evaluating a child with recurrent or persisting headache. Hemiparesis and papilledema were the other manifestations of REL. One out of 52 children with NCC and 4 out of 20 children with tuberculoma had papilledema showing higher incidence of raised intracranial pressure in patients with tuberculoma. This can be explained by larger size of lesion in tuberculoma. Majority of children with NCC (76.93%) had lesions of <10mm size, whereas majority of children with tuberculoma (65%) had lesions of >10mm size. Thus smaller lesions were common in NCC and larger lesions were common in tuberculoma.

Majority of children with NCC and tuberculoma showed single ring enhancing lesion. Frontal lobe was the commonest site involved both in

NCC (48.57%) and tuberculoma (57.14%), followed by parietal lobe. This is in contrast to pratibasinghi *et al.* study, where, parietal lobe was the most common site of lesion.

40% of tuberculomas disappeared at one year followup, whereas only 23 % of NCCs disappeared at one year followup. Thus a greater decrease/disappearance of lesions at end of one year follow up was seen in tuberculoma than NCC. Nearly one third of lesions in NCC calcified at the end of one year. Very few lesions of NCC and tuberculoma showed change in size at the end of 3 months follow up neuroimaging. Majority of lesions showed change between 3-6 months of treatment. Hence in a resource limited setting, first follow up scan may be planned at 6 months.

In children with NCC, there was no significant difference in the response (with respect to decrease in number and size of lesions) with 8 day versus 28 day albendazole therapy.

CONCLUSION

In the present study, neurocysticercosis and tuberculoma were the major causes of ring enhancing lesion in children. Recurrent or persistent headache was present in 31% of children with ring enhancing lesion. So, the possibility of ring enhancing lesion should be kept in mind while evaluating a child with recurrent or persistent headache. Commonest type of seizure in children with ring enhancing lesion was partial seizures. But 12% of children with ring enhancing lesion presented with generalised tonic clonic seizures. Thus, even children with first episode of generalised tonic clonic seizure, should be evaluated for ring enhancing lesion. Very few children showed disappearance of lesions in size / number during follow up at 3 months. Thus in a resource limited setting like ours, follow up scan for persistence of ring enhancing lesion can be done at 6 months. No significant difference was seen in the resolution of lesions with 8 day versus 28 day course of albendazole in children with NCC.

Abbreviations

NCC – Neurocysticercosis

REL – Ring enhancing lesion

GTCS—Generalised tonic clonic seizures

REFERENCES

1. Yodnopaklow P, Mahuntussanapong A. Single small enhancing CT lesion in Thai patients with acute symptomatic seizures: a clinico-radiological study. *Tropical Medicine & International Health*. 2000 Apr;5(4):250-5.
2. Murthy JM, Reddy YS. Prognosis of epilepsy associated with single CT enhancing lesion: a long term follow up study. *Journal of the neurological sciences*. 1998 Aug 14;159(2):151-5.
3. Sethi PK, Kumar BR, Madan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. *Journal of Neurology, Neurosurgery & Psychiatry*. 1985 Sep 1;48(9):866-9.
4. Ahuja GK, Behari M, Prasad K, Goulatia RK, Jaikhani BL. Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the cause?. *Journal of neurology, neurosurgery, and psychiatry*. 1989 Jul;52(7):915.
5. Del Brutto OH. *Patho of Global Health 2012*. Department of Neurological Sciences Hospital-Clinics Kennedy, Guayaquil, Ecuador. 2012 Sep;106(5) 299-304.
6. Mahato PS, Dabhi AS, Thorat PB. Clinical and investigative profile of ring-enhancing lesions on neuroimaging. *Indian J Clin Pract*. 2012;22(10):512-8.
7. Singhi P, Ray M, Singhi S, Khandelwal N. Clinical spectrum of 500 children with neurocysticercosis and response to albendazole therapy. *Journal of Child Neurology*. 2000 Apr;15(4):207-13.