

Effect of Albendazole Therapy in Parenchymal Neurocysticercosis Lesion Resolution, Lesion Load Reduction and Lesion Stage Evolution Observed on Serial Neuroimaging in Patients Presenting with Seizures

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ABSTRACT

Neurocysticercosis (NCC) is the commonest parasitic disease of nervous system in humans and single most common cause of acquired seizures in developing countries. In this study, we intended to evaluate the effect of Albendazole therapy in Parenchymal Neurocysticercosis lesion resolution, lesion load reduction and lesion stage evolution observed on serial neuroimaging in patients presenting with seizures. Albendazole was given for 1 month. Patients were followed up and neuroimaging was carried out after a 6-month interval. Temporal changes in lesion profile including lesion load reduction, lesion resolution, lesion stage evolution and seizure recurrences during the interval period were observed. After Albendazole therapy for 6 months, patients were followed up with serial neuroimaging. Lesion resolution was seen in 29.33%, lesion load reduction in 41.33% and calcified lesions in 52% cases.

Key words: Neurocysticercosis (NCC), Albendazole, Seizure, Lesion load reduction, Lesion resolution.

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INTRODUCTION

Neurocysticercosis is caused by the larval stage of the tapeworm *Taenia solium*, is the commonest parasitic disease of nervous system in humans and the single most common cause of acquired seizures in the developing world, where prevalence rates of active epilepsy are twice those in developed countries.¹ It is highly prevalent in low- and middle- income countries (LMICs).² 60-90% of infested patients have CNS involvement with cerebrum and cerebellum as common sites.³ NCC is common in communities with poor hygiene and sanitation where untreated human faeces are used in field/garden as fertilizer. Vegetarians might get infected by eating raw vegetables contaminated with ova or proglottids of *Taenia solium* whereas non- vegetarians may get infected after eating improperly cooked pork meat.⁴ The life cycle of *T. Solium* (adult tapeworm) involves two hosts, humans and pigs. Humans are definitive host and pigs are intermediate host and acquire intestinal infection by ingestion of undercooked pork infected with live encysted larvae or through feco-orally by consuming food or water contaminated by eggs either accidentally or via infected food handlers (carriers of tapeworm), or rarely by autoingestion.^{5,6}

The cysts pass through a sequence of four morphological stages: vesicular, colloidal, granular nodular, and nodular calcified stage.³ Neuroimaging of symptomatic cases of NCC usually shows colloidal, granular, nodular or vesicular stages whereas calcified stage represent dead and inactive cysts, these are not totally inert and may intermittently release parasite antigens that result in inflammatory edema and symptoms.^{7,8}

NCC can cause a variety of symptoms and signs depending on the number, size, stage, and location of the pathological changes, as well as the inflammatory host response, or it can also be clinically asymptomatic.^{9,10}

NCC can cause ependymitis, increased intracranial pressure, Arachnoiditis (especially in the basal cisterns, which can lead to communicating hydrocephalus, vasculitis and/or compression of cerebral vessels, can result from subarachnoid disease.¹¹ There is an absolute criterion which include: histological confirmation of parasites, evidence of subretinal cysts, and demonstration of the scolex within a cyst. Neuroimaging criteria are categorized as major (cystic lesions without scolex, enhancing lesions, multilobulated cysts, and calcifications), confirmative (resolution of

cysts after cysticidal drug therapy, spontaneous resolution of single enhancing lesions, and migrating ventricular cysts on sequential neuroimaging studies) and minor (hydrocephalus and leptomeningeal enhancement). Clinical/exposure criteria include detection of anticysticercal antibodies or cysticercal antigens by well-standardized tests, systemic cysticercosis, evidence of a household *Taenia* carrier, suggestive clinical manifestations, and residence in endemic areas. Besides patients having absolute criteria, definitive diagnosis can be made in those having two major neuroimaging criteria (or one major plus one confirmative criteria) plus exposure. For patients presenting with one major and one minor neuroimaging criteria plus exposure, definitive diagnosis of NCC requires the exclusion of confounding pathologies. Probable diagnosis is reserved for individuals presenting with one neuroimaging criteria plus strong evidence of exposure.¹²

Drug of choice of NCC is Albendazole (administered at doses of 15 mg/kg/d for 1 month).¹³ The use of antiepileptic drug (AED) often results in the control of seizures in patients with neurocysticercosis-related epilepsy.¹⁴ However, the optimal length of AED therapy in patients with neurocysticercosis has not been established.¹⁵

Inflammation around the parasites is a hallmark of neurocysticercosis pathophysiology. Although mechanisms regulating this inflammation are poorly understood, anti-inflammatory drugs, particularly corticosteroids, have been long used alone or with anthelmintics to manage disease and limit neurological complications and perhaps damage to neural tissues.¹⁶ The only way to reduce the burden of disease due to NCC is to prevent it. Public education regarding proper hygiene and sanitation and enforcing strict animal husbandry and meat inspection procedures are warranted.^{17,18} No previous study has been done to compare the lesion load reduction in different lobes after anti-helminthic therapy. So we conducted this study check the effect of Albendazole Therapy in Parenchymal Neurocysticercosis lesion resolution, lesion load reduction and lesion stage evolution observed on serial neuroimaging in patients presenting with seizures. This prospective study was carried out on a total of 75 patients having neurocysticercosis presenting in OPD and emergency of the department of Paediatrics, Rajindra Hospital Patiala, over a period of 1.5 yr (Jan 2021 to July 2022). NCC patients with seizures aged 2-16 years were included in this study. Patients who refused to give consent, hemodynamically unstable, with any neurological deficit, progressive neurological disease, tubercular meningitis, severe claustrophobia and absolute procedural contraindication related to MRI as per se as those with ferromagnetic cardiac pacemakers or implants were excluded from study.

MATERIALS AND METHODS

In this present prospective study, we studied the effect of Albendazole therapy in Parenchymal Neurocysticercosis lesion resolution, lesion load reduction and lesion stage evolution observed on serial neuroimaging in patients presenting with seizures. Albendazole was given for 1 month. Patients were followed up and neuroimaging was carried out after a 6 months interval. Temporal changes in lesion profile including lesion load reduction, lesion resolution, lesion stage evolution and seizure recurrences during the interval period were observed.

Statistical Analysis

Data was compiled by using Microsoft excel version 2021. Data was tabulated and statistically analysed using microsoft excel version 2019 and Epi Info [CDC Atlanta] software version 7.2.50. For all statistical purposes p value <0.05 was considered statistically significant. Most of the results were expressed in percentage, lower upper confidence limits and at some places descriptive statistics which included mean, S.D., minimum, maximum and median.

RESULTS

Table I showed that mean age was 9.92 ± 3.89 with minimum age of patient being 3 years and maximum age being 16 years. 40%(n=30) were females and rest 60%(n=45) were males.69.33%(n=52) patients were from rural area while 30.67%(n=23) were from urban. In this study, 16%(n=12) belonged to lower middle class, 82.67%(n=62) belonged to upper lower class and 1.33%(n=1) belonged to upper middle class.

Table II showed 72%(n=54) had single episode of seizure, 25.33%(n=19) had 2 episodes of seizures while remaining 2.67%(n=2) had 3 episodes of seizures

In the present study, 58.67%(n=44) patients had GTCS type seizures while 41.33%(n=31) had focal type seizures. In our study, 88%(n=66) patients had no focal neurological deficit while 12%(n=9) had some focal neurological deficit.

In our study, seizure episode was treated with anti-epileptic drug Levetiracetam in 46.67%(n=35) while treated with Valproate in 53.33%(n=40).

It was observed that out of 75 patients, 80%(n=60) were compliant to ACT whereas, 20%(n=15) were non-compliant to ACT. 97.33%(n=73) patients were compliant to anthelmintic therapy whereas, 2.67%(n=2) were non-compliant.

In the present study, it was observed that out of 75 patients, steroids were administered in 93.33%(n=70) whereas, 6.67%(n=5) received no steroids.

In our study, on MRI (pretreatment) in table III, it was observed that most of patients 68%(n=51) had single lesion and 22.67%(n=17) had 2 lesions. Similarly, 4%(n=3) patients had 3 lesions and 2.67%(n=2) patients had 5 lesions. 6 number of lesions were seen in 2.67%(n=2) patients.

In the present study, it was observed that parietal region (41) was most commonly involved while occipital region (5) being least commonly involved. Out of 41 patients with parietal lobe involvement, 18 had only right parietal lobe involvement while 18 had only left parietal lobe involvement and 5 had bilateral parietal lobe involvement. Out of 19 patients who had frontal lobe involvement, 11 had right frontal while 8 had left frontal lobe involvement. Out of 10 patients with temporal lobe involvement, 6 had right temporal and 4 had left temporal lobe involvement. Out of 5 patients with occipital lobe involvement, 3 had right occipital and 2 had left occipital lobe involvement.

In our study, it was observed that parietal lobe involvement was maximum 54.67%(n=41) patients while occipital lobe involvement was minimum 6.67%(n=5) patients. Frontal lobe was involved in 25.33%(n=19) patients and temporal lobe was involved in 13.33%(n=10) patients. Peri-lesional edema was present in 93.33%(n=70) and absent in 6.67%(n=5).37.33%(n=28) patients had granular-nodular stage of lesion(maximum), 33.33%(n=25) patients had colloidal-vesicular stage of lesion, 14.67%(n=11)

patients had nodular calcified stage of lesion, 9.33%(n=7) patients had variable stage of lesion and 5.33%(n=4) patients had vesicular stage of lesion (minimum).

We observed that post treatment 46.67%(n=35) patients had single lesion, 29.33%(n=22) patients had zero lesion, 17.33%(n=13) had 2 lesions, 2.67%(n=2) patients had 3 lesions, 2.67%(n=2) patients had 6 lesions and 1.33%(n=1) patient had 5 lesions.

Post treatment MRI showed 29.33% (n=22) with complete disappearance of lesion. It was observed that 41.34%(n=31) had parietal lobe involvement, out of which 18.67%(n=14) had only left parietal lobe involvement and 16%(n=12) had right parietal lobe involvement. 6.67%(n=5) had bilateral parietal lobe involvement. It was observed that 18.67%(n=14) had frontal lobe involvement, out of which 12%(n=9) had right frontal lobe involvement whereas, 6.67%(n=5) had left frontal lobe involvement. Out of 8%(n=6) with temporal lobe involvement, 4%(n=3) had right temporal lobe involvement and 4%(n=3) had left temporal lobe involvement.

2.67%(n=2) had right occipital lobe involvement. Post treatment MRI showed involvement of parietal lobe in 41.33%(n=31), complete disappearance in 29.33%(n=22), frontal lobe involvement in 18.67%(n=14), temporal lobe involvement in 6.67% (n=5) and occipital lobe involvement in 4%(n=3). We

observed that in 10.67%(n=8) peri-lesional edema was present post treatment whereas, in 89.33%(n=67) peri-lesional edema was absent.

Table IV revealed that It was observed that out of 75 patients with seizures at admission, 10.7%(n=8) had seizure recurrence whereas, 89.3% (67) patients were seizure free post treatment with p value <0.0001 that was statistically significant. 10.7%(n=8) of the total patients had seizure recurrence, among them 1.7% were complaint to ACT and 89.3%(n=67) did not have any seizure recurrence, among them 93.8% were complaint to ACT with p value of < 0.0001 that was statistically significant.

In our study, it was observed (post treatment) that 29.3%(n=22) had complete lesion resolution while 70.7% (53) had persistent lesions that was statistically significant (p value <0.0001). It was observed (pretreatment) peri-lesional edema was present in 94.7%(n=71) patients whereas, post treatment peri-lesional edema was present only in 10.3%(n=8) patients with p value <0.0001, which means results are statistically significant. During pretreatment phase 14.7%(n=11) had calcified lesions and 85.3%(n=64) did not have any calcified lesions. Whereas, in the same 75 patients during post treatment phase, 52%(n=39) had calcified lesions and 48%(n=36) did not have any calcified lesion with p value 0.00001 implies that was statistically significant.

Table I: Distribution of NCC Cases According To Demographic Factors

| Age Groups | Frequency | Percent | Exact 95% LCL | Exact 95% UCL |
|----------------------------|-----------|---------|---------------|---------------|
| 10-13 years | 22 | 29.33% | 19.38% | 40.98% |
| 14-16 years | 20 | 26.67% | 17.11% | 38.14% |
| 2- 5 years | 13 | 17.33% | 9.57% | 27.81% |
| 6-9 years | 20 | 26.67% | 17.11% | 38.14% |
| Gender | | | | |
| Female | 30 | 40.00% | 28.85% | 51.96% |
| Male | 45 | 60.00% | 48.04% | 71.15% |
| Residence | | | | |
| Rural | 52 | 69.33% | 57.62% | 79.47% |
| Urban | 23 | 30.67% | 20.53% | 42.38% |
| Socioeconomic class | | | | |
| Lower middle | 12 | 16.00% | 8.55% | 26.28% |
| Upper lower | 62 | 82.67% | 72.19% | 90.43% |
| Upper middle | 1 | 1.33% | 0.03% | 7.21% |

Table 2: Distribution of NCC Cases According To CNS Manifestations And Anthelmintic Therapy

| Number of Seizure Episodes | Frequency | Percent | Exact 95% LCL | Exact 95% UCL |
|--|-----------|---------|---------------|---------------|
| 1 | 54 | 72.00% | 60.44% | 81.76% |
| 2 | 19 | 25.33% | 15.99% | 36.70% |
| 3 | 2 | 2.67% | 0.32% | 9.30% |
| Type of Seizure | | | | |
| Focal | 31 | 41.33% | 30.08% | 53.30% |
| GTCS | 44 | 58.67% | 46.70% | 69.92% |
| Focal Neurological Deficit | | | | |
| Absent | 66 | 88.00% | 78.44% | 94.36% |
| Present | 9 | 12.00% | 5.64% | 21.56% |
| Anti-Convulsant Drug | | | | |
| Levetiracetam | 35 | 46.67% | 35.05% | 58.55% |
| Valproate | 40 | 53.33% | 41.45% | 64.95% |
| Anti-Convulsant Drug Compliance | | | | |
| Yes | 60 | 80.00% | 69.17% | 88.35% |
| No | 15 | 20.00% | 11.65% | 30.83% |
| Compliance to albendazole | | | | |
| Yes | 73 | 97.33% | 90.70% | 99.68% |
| No | 2 | 2.67% | 0.32% | 9.30% |
| Steroids | | | | |
| Yes | 70 | 93.33% | 85.12% | 97.80% |
| No | 5 | 6.67% | 2.20% | 14.88% |

Table 3: Distribution of NCC Cases According To Pre And PostTreatment MRI Findings

| No. Of Lesions pre-treatment | Frequency | Percent | Exact 95% LCL | Exact 95% UCL |
|---|-----------|---------|------------------|------------------|
| 1 | 51 | 68.00% | 56.22% | 78.31% |
| 2 | 17 | 22.67% | 13.79% | 33.79% |
| 3 | 3 | 4.00% | 0.83% | 11.25% |
| 5 | 2 | 2.67% | 0.32% | 9.30% |
| 6 | 2 | 2.67% | 0.32% | 9.30% |
| Site involved | | | | |
| Bilateral Parietal | 5 | 6.67% | 2.20% | 14.88% |
| Left Frontal | 8 | 10.67% | 4.72% | 19.94% |
| Left Occipital | 2 | 2.67% | 0.32% | 9.30% |
| Left Parietal | 18 | 24.00% | 14.89% | 35.25% |
| Left Temporal | 4 | 5.33% | 1.47% | 13.10% |
| Right Frontal | 11 | 14.67% | 7.56% | 24.73% |
| Right Occipital | 3 | 4.00% | 0.83% | 11.25% |
| Right Parietal | 18 | 24.00% | 14.89% | 35.25% |
| Right Temporal | 6 | 8.00% | 2.99% | 16.60% |
| Lobes involved Pre treatment | | | | |
| Parietal Lobe | 41 | 54.67% | 42.75% | 66.21% |
| Frontal Lobe | 19 | 25.33% | 15.99% | 36.70% |
| Temporal Lobe | 10 | 13.33% | 6.58% | 23.16% |
| Occipital Lobe | 5 | 6.67% | 2.20% | 14.88% |
| MRI Peri-Lesional Edema Pre-Treatment | | | | |
| Present | 71 | 94.67% | 86.90% | 98.53% |
| Absent | 4 | 5.33% | 1.47% | 13.10% |
| MRI Stage of Lesion | | | | |
| Colloidal-Vesicular | 25 | 33.33% | 22.86% | 45.17% |
| Granular-Nodular | 28 | 37.33% | 26.43% | 49.27% |
| Nodular -Calcified | 11 | 14.67% | 7.56% | 24.73% |
| Variable | 7 | 9.33% | 3.84% | 18.29% |
| Vesicular | 4 | 5.33% | 1.47% | 13.10% |
| No. of Lesions post treatment | | | | |
| 0 | 22 | 29.33% | 19.38% | 40.98% |
| 1 | 35 | 46.67% | 35.05% | 58.55% |
| 2 | 13 | 17.33% | 9.57% | 27.81% |
| 3 | 2 | 2.67% | 0.32% | 9.30% |
| 5 | 1 | 1.33% | 0.03% | 7.21% |
| 6 | 2 | 2.67% | 0.32% | 9.30% |
| MRI Location Post Treatment | | | | |
| Left Parietal | 14 | 18.67% | 10.60% | 29.33% |
| Right Parietal | 12 | 16.00% | 8.55% | 26.28% |
| Bilateral parietal | 5 | 6.67% | 2.20% | 14.88% |
| Left Frontal | 5 | 6.67% | 2.20% | 14.88% |
| Left temporal | 3 | 4.00% | 0.83% | 11.25% |
| Right Frontal | 9 | 12.00% | 5.64% | 21.56% |
| Right Occipital | 2 | 2.67% | 0.32% | 9.30% |
| Right Temporal | 3 | 4.00% | 0.83% | 11.25% |
| Complete Disappearance | 22 | 29.33% | 19.38% | 40.98% |
| Lobes involved Post Treatment | | | | |
| Complete Disappearance | 22 | 29.33% | 19.38% | 40.98% |
| Parietal Lobe | 31 | 41.33% | 30.08% | 53.30% |
| Frontal Lobe | 14 | 18.67% | 10.60% | 29.33% |
| Occipital Lobe | 3 | 4.00% | 0.83% | 11.25% |
| Temporal Lobe | 5 | 6.67% | 2.20% | 14.88% |
| MRI Peri-Lesional Edema Post Treatment | | | | |
| Present | 8 | 10.67% | 4.72% | 19.94% |
| Absent | 67 | 89.33% | 80.06% | 95.28% |
| MRI Load Resolution | | | | |
| Complete Resolution | 22 | 29.33% | 19.38% | 40.98% |
| Partial Resolution | 49 | 65.33% | 53.46% | 75.96% |
| No Resolution | 4 | 5.33% | 1.47% | 13.10% |
| MRI Load Reduction | | | | |
| Yes | 31 | 41.33% | 30.08% | 53.30% |
| No | 44 | 58.67% | 46.70% | 69.92% |

Table 4: Comparison of NCC Cases According To Seizure Recurrence, Pre And Post Treatment MRI Findings

| | Seizures Present at diagnosis | | | P value |
|---|--|----------------|-----------|---------|
| Seizure Recurrence | No | Yes | | <0.0001 |
| No | 0 | 67 | 67(89.3%) | |
| Yes | 0 | 8 | 8(10.7%) | |
| | 0(0.0%) | 75(100%) | | |
| | Compliance to ACT | | | |
| Seizure Recurrence | No | Yes | | <0.0001 |
| No | 8 (53.3%) | 59 (98.3%) | 67(89.3%) | |
| Yes | 7 (46.7%) | 1 (1.7%) | 8(10.7%) | |
| | 15(20.0%) | 60(80%) | 75 | |
| | MRI Lesions pre-treatment | | | |
| MRI Lesions post-treatment | Absent | Present | | <0.0001 |
| Absent | 0 | 22(29.3%) | 22(29.3%) | |
| Present | 0 | 53(70.7%) | 53(70.7%) | |
| | 0(0.0%) | 75(100.0%) | 75 | |
| | MRI Peri lesional edema Pre-treatment | | | |
| MRI Peri lesional edema Post Treatment | Absent | Present | | <0.0001 |
| Absent | 4 | 63 | 67(89.3%) | |
| Present | 0 | 8 | 8(10.7%) | |
| | 4(5.33%) | 71(94.7%) | 75 | |
| | Calcified Lesion Pre-treatment | | | |
| Calcified Lesion Post treatment | No | Yes | | 0.00001 |
| No | 29 | 7 | 36(48.0%) | |
| Yes | 35 | 4 | 39(52.0%) | |
| | 64(85.3%) | 11(14.7%) | 75 | |

DISCUSSION

This prospective study was conducted on 75 patients of neurocysticercosis in Department of Paediatrics, Rajindra Hospital Patiala from January 2021 to July 2022. Neurocysticercosis is the commonest parasitic disease of nervous system in humans caused by *Taenia Solium* (tapeworm) and is single most common cause of acquired seizures in children particularly in developing countries. We found that the mean age in our study was 9.9 ± 3.89 years. Minimum age was 3 years while maximum age was 16 years. This was comparable to many studies, in Bandana Shrestha et al¹⁹ mean age was 10.6 years, in Modak et al²⁰ mean age was 10.8 years. Similarly, Suthar et al¹⁴ observed mean age was 9.9 years. In this study, males (60%) were more as compared to females (40%). Adhikari S et al²¹ found that there were 61.3% males and 38.7% females. Almost similar results were seen in Sahu PS et al²² in which 69.5% were males and 30.4% were females. Male preponderance in our study might be possible because majority of population presenting to our hospital was from rural and less literate areas where there is preferential treatment of male children at the best medical facility with negligence towards female health. In the present study, we observed 82.67%(n=62) patients were from lower socio-economic class. Almost similar results were observed by Bhattacharjee S et al²³ and Saurabh K et al²⁴ where they found patients from lower socio-economic class 94.7% and 81.66% respectively. Similarly, Suthar et al¹⁴ observed 76.3% population was from low socio-economic class. Most of the patients in our study were from rural background. This possibly explains prevalence of NCC in lower socio-economic class where poor hand hygiene, poor sanitation and consumption of unsafe drinking water practices leads to occurrence of disease. In this study, generalized seizures 58.67%(n=44) were more common than focal. GTCS was seen in cases. Similar results were in the study conducted by Gauchan E

et al²⁵ who found generalized seizure in 52% of cases. Prasad R et al²⁶ found generalized seizure in 65% of cases. Similarly, Saurabh K et al²⁷ also found GTCS in 60% cases. This is possibly due to secondary generalization of focal seizures. Our study revealed that 89.33%(n=63) cases were seizure free post treatment whereas 10.7%(n=8) patients had seizure recurrence. This was comparable to other studies. Gogia S et al²⁸, they found that 80% of the children were seizure-free. Baranwal AK et al²⁹ showed that after 4 weeks seizure recurrence was seen in 31.3% of placebo-treated children vs. 12.9% of albendazole-treated children. Anti helminthic treatment with Albendazole leads to significant decrease in number and frequency of seizures on follow up. It was observed that 80%(n=60) patients were compliant to ACT whereas, 20%(n=15) were non-compliant. Among compliant patients, 98.3%(n=59) did not have seizure recurrence. This implies the role of ACT in controlling seizure and preventing its recurrence in children with NCC. It was observed that parietal lobes (54.6%) were most commonly involved in patients with parenchymal NCC. Similar results were seen by Sahu PS et al²² and Modak et al²⁰ in their studies with parietal lobe involvement of 50.9% and 54.7% respectively. We observed single lesion in 68%(n=51) of cases. Similar results were seen by PL Prasad et al³⁰ and Arora BS et al³¹ in which single lesions were seen in 88.5% and 94% cases. We observed that peri-lesional edema after 6 month follow up was present only in 10.66%(n=8) cases while 89.33%(n=67) patients had no peri-lesional edema on follow up. Suthar et al¹⁴ observed 78% patients were having no peri-lesional edema on follow up. We found that on follow up MRI brain after 6 months complete resolution was seen in 29.33%(n=22), partial resolution was seen in 65.33%(n=49) while no resolution was seen in 5.33%(n=4). It was comparable to Suthar et al¹⁴ in which they observed complete resolution in 28% cases. Similarly in Bhattacharjee S et al³² complete resolution was seen in 31.6%

patients. This signifies that Albendazole has a significant effect on lesion resolution in patients with parenchymal NCC. It was observed that lesion load reduction was seen in 41.33%(n=31) of the cases. Carpio A et al³³ also found that load reduction was seen in only 41.2 % cases. We observed that calcified lesion on 6 month follow up was seen in 52%(n=39) of cases. Suthar R et al¹⁴ also found that calcified lesions in 32.5 % of cases at 1 year follow up. Less load reduction and difference in lesion stage evolution after 4 weeks of Albendazole therapy possibly due to less time period of study and small sample size.

CONCLUSION

NCC is an important cause of epilepsy and other neurological manifestations. In our study we have observed that there was no correlation of lesion load reduction with specific lobes. No previous study has been done to compare the lesion load reduction in different lobes after anti-helminthic therapy. Our study concluded that the patients who were compliant with the antihelminthic therapy were found to have excellent outcomes in terms of lesion load reduction, resolution, stage evolution and marked decline in seizure recurrence. We did this comparison to prove that albendazole therapy is the mainstay of treatment of NCC irrespective of lesion location. It is observed that albendazole therapy is equally effective in resolution of lesions irrespective of the location of lesion in the brain parenchyma. We observed that improved hygienic practices, sanitation and health education among people especially rural areas might help in disease prevention.

REFERENCES

- García HH, Evans CA, Nash TE, Takayanagui OM, White AC, Botero D et al. Current consensus guidelines for treatment of neurocysticercosis. *Clinical Microbiology Reviews*. 2002 Oct 1;15(4):747-56.
- V. Rajshekhar. Neurocysticercosis: Diagnostic problems & current therapeutic strategies. *Indian Journal of Medical Research*.2016;144(3);319–26.
- S. K. Handique, R. R. Das, B. Saharia, P. Das, R. Buragohain, and P. Saikia, "Coinfection of Japanese encephalitis with neurocysticercosis: An imaging study," *American Journal of Neuroradiology*:2008;29(1): 170–5.
- Singhi P, Saini AG. Pediatric neurocysticercosis: current challenges and future prospects. *Pediatric Health Med Ther*. 2016;7:5-16.
- Ito A, Putra MI, Subahar R, Sato MO, Okamoto M, Sako Y et al. Dogs as alternative intermediate hosts of *Taenia solium* in Papua (Irian Jaya), Indonesia confirmed by highly specific ELISA and immunoblot using native and recombinant antigens and mitochondrial DNA analysis. *J Helminthol*. 2002 Dec;76(4):311-4
- Schantz PM, Moore AC, Muñoz JL, Hartman BJ, Schaefer JA, Aron AM, Persaud D, Sarti E, Wilson M, Flisser A. Neurocysticercosis in an Orthodox Jewish community in New York City. *N Engl J Med*. 1992 Sep 3;327(10):692-5
- Nash TE, Pretell EJ, Lescano AG, Bustos JA, Gilman RH, Gonzalez AE, Garcia HH, Cysticercosis Working Group in Peru. Perilesional brain oedema and seizure activity in patients with calcified neurocysticercosis: a prospective cohort and nested case–control study. *The Lancet Neurology*. 2008 Dec 1;7(12):1099-105.
- Govindappa SS, Narayanan JP, Krishnamoorthy VM, Shastry CH, Balasubramaniam A, Krishna SS. Improved detection of intraventricular cysticercal cysts with the use of three-dimensional constructive interference in steady state MR sequences. *American journal of neuroradiology*. 2000 Apr 1;21(4):679-84.
- Carabin H, Ndimubanzi PC, Budke CM, Nguyen H, Qian Y, Cowan LD, Stoner JA, Rainwater E, Dickey M. Clinical manifestations associated with neurocysticercosis: a systematic review. *PLoS Negl Trop Dis*. 2011 May;5(5):e1152
- Bern C, Garcia HH, Evans C, Gonzalez AE, Verastegui M, Tsang VC, Gilman RH. Magnitude of the disease burden from neurocysticercosis in a developing country. *Clin Infect Dis*. 1999 Nov;29(5):1203-9.
- Takayanagui OM, Odashima NS. Clinical aspects of neurocysticercosis. *Parasitol Int*. 2006;55 Suppl:S111-5.
- Del Brutto OH, Nash TE, White AC Jr, Rajshekhar V, Wilkins PP, Singh G, Vasquez CM, Salgado P, Gilman RH, Garcia HH. Revised diagnostic criteria for neurocysticercosis. *J Neurol Sci*. 2017 Jan 15;372:202-10.
- Escobedo F, Penagos P, Rodriguez J. Albendazole therapy for neurocysticercosis. *Arch Intern Med*. 1987;147(4): 738-41.
- Suthar R, Sahu JK, Ahuja CK, Khandelwal N, Sehgal R, Singhi P. A prospective cohort study to assess the frequency and risk factors for calcification in single lesion parenchymal neurocysticercosis. *Seizure*. 2020 Dec;83:132-138.
- Sinha S, Sharma BS. Intraventricular neurocysticercosis: a review of current status and management issues. *Br J Neurosurg*. 2012 Jun;26(3):305-9
- Nash TE, Mahanty S, Garcia HH; Cysticercosis Group in Peru. Corticosteroid use in neurocysticercosis. *Expert Rev Neurother*. 2011;11(8):1175-83.
- Allan JC, Velasquez-Tohom M, Fletes C, Torres-Alvarez R, Lopez- Virula G, Yurrita P, Soto de Alfaro H, Rivera A, Garcia- Noval J. Mass chemotherapy for intestinal *Taenia solium* infection: effect on prevalence in humans and pigs. *Trans R Soc Trop Med Hyg*. 1997 Sep-Oct;91(5):595-8
- Sarti E, Schantz PM, Avila G, Ambrosio J, Medina-Santillán R, Flisser A. Mass treatment against human taeniasis for the control of cysticercosis: a population-based intervention study. *Trans R Soc Trop Med Hyg*. 2000 Jan-Feb;94(1):85-9
- Shrestha B, Mainali P, Sayami S, Shrestha OK. Clinico-radiological aspects of neurocysticercosis in pediatric population in a tertiary hospital. *Journal of the Nepal Medical Association*. 2013 Jul 1;52(191).
- Modak A, Suthar R, Sharawat IK, Sankhyan N, Sahu JK, Malhi P, Khandelwal N. An ambispective cohort study to assess seizure recurrences in children with calcified parenchymal neurocysticercosis. *The American journal of tropical medicine and hygiene*. 2019 Oct;101(4):812.
- Adhikari S, Sathian B, Koirala DP, Rao KS. Profile of children admitted with seizures in a tertiary care hospital of Western Nepal. *BMC Pediatr*. 2013;13:43-7
- Sahu PS, Seepana J, Padela S, Sahu AK, Subbarayudu S, Barua A. Neurocysticercosis in children presenting with afebrile seizure: clinical profile, imaging and serodiagnosis. *Revista do Instituto de Medicina Tropical de São Paulo*. 2014 May;56:253-8.
- Bhattacharjee S, Biswas P, Mondal T. Clinical profile and follow-up of 51 pediatric neurocysticercosis cases: A study from Eastern India. *Ann Indian Acad Neurol*. 2013;16(4):549-55

24. Saurabh K, Ranjan S. Fasciolopsiasis in children: Clinical, sociodemographic profile and outcome. *Indian Journal of Medical Microbiology*. 2017 Oct 1;35(4):551-4.
25. Gauchan E, Malla T, Basnet S, Rao KS. Variability of presentations and CT-scan findings in children with neurocysticercosis. *Kathmandu Univ Med J (KUMJ)*. 2011 Apr-Jun;9(34):17-21
26. Prasad R, Anil, Mishra OP, Mishra SP, Upadhyay RS, Singh TB. Oxidative stress in children with neurocysticercosis. *Pediatr Infect Dis J*. 2012 Oct;31(10):1012-5
27. Saurabh K, Ranjan S. Fasciolopsiasis in children: Clinical, sociodemographic profile and outcome. *Indian Journal of Medical Microbiology*. 2017 Oct 1;35(4):551-4.
28. Gogia S, Talukdar B, Choudhury V, Arora BS. Neurocysticercosis in children: clinical findings and response to albendazole therapy in a randomized, double-blind, placebo-controlled trial in newly diagnosed cases. *Trans R Soc Trop Med Hyg*. 2003 Jul-Aug;97(4):416-21
29. Baranwal AK, Singhi PD, Khandelwal N, Singhi SC. Albendazole therapy in children with focal seizures and single small enhancing computerized tomographic lesions: a randomized, placebo-controlled, double blind trial. *Pediatr Infect Dis J*. 1998 Aug;17(8):696-700.
30. Prasad PL, Dawra R, Chandra S. Clinico-Radiological Profile of Neurocysticercosis in Children. *Journal of Nepal Paediatric Society*. 2019 Apr 27;39(1):15-21.
31. Arora BS, Dhamija K, Veeramachaneni R, Indurkar PS. Neurocysticercosis: clinical presentations, serology and radiological findings: experience in a teaching institution. *Int J Res Med Sci*. 2016 Feb;4(2):519-23.
32. Bhattacharjee S, Biswas P, Mondal T. Clinical profile and follow-up of 51 pediatric neurocysticercosis cases: A study from Eastern India. *Ann Indian Acad Neurol*. 2013;16(4):549-55.
33. Carpio A, Santillán F, León P, Flores C, Hauser WA. Is the course of neurocysticercosis modified by treatment with antihelminthic agents? *Arch Intern Med*. 1995;155(18):1982-8.

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