Egyptian Pediatric Association Gazette

CASE REPORT Open Access

Paediatric parenchymal neurocysticercosis with pleomorphic clinico-radiological presentations: a case series

Sanjay Kumar[1], Radhamohan Rana[1], Mahesh Kumar[1*] and Aakash Yadav[1]

Abstract

Background Neurocysticercosis (NCC) is thought to be the most common helminthic infection of central nervous system in India. In children, it has pleomorphic clinical and radiological presentations depending on location and stage of lesion. Solitary cystic granuloma appearing as a single ring enhancing lesion is the most frequently encountered neuroimaging finding in patients with neurocysticercosis or tuberculoma in India. This series reports unusual clinico-radiological aspects of pediatric neurocysticercosis patients of Asian ethnicity.

Case presentation In the present case series, we have described socio-demographic and clinico-radiological profile of eight cases of neurocysticercosis with parenchymal lesions in varying stages of development. Among these two had single discrete ring enhancing lesion (SDREL), two had single conglomerated ring enhancing lesion (SCREL), and five cases reported to have multiple ring enhancing lesions (MREL). Two cases with recurrent neurocysticercosis have been reported which is quite rare. Magnetic resonance spectroscopy helps to differentiate between neurocysticercosis and tuberculoma and may avoid brain biopsies or unnecessary anti-tubercular treatment. Magnetic resonance spectroscopy (MRS) was done in three cases with findings of absence of lipid peak and choline/creatinine ratio less than 1.2.

Conclusion Despite the advances in neuroimaging, accurate diagnosis of NCC is still sometimes difficult, which is related to the pleomorphic nature of disease and significant overlapping features with tuberculoma. A combination of proper diagnostic criteria and neuroimaging findings are helpful in making the diagnosis without invasive and potentially harmful investigations in paediatric patients.

Keywords Paediatric seizure, Neurocysticercosis, Ring enhancing lesion, Magnetic resonance spectroscopy

Background

Neurocysticercosis (NCC) is the most common acquired infection of central nervous system caused by encysted larva of a cestode- *Taenia Solium.* Humans are considered definitive host and are infected when they consume raw or undercooked pork or food infected with live cysticerci. It may also spread via feco-oral route or consuming water contaminated with *Taenia Solium* eggs. World health organisation (WHO) considered cysticercosis as one of the "neglected tropical zoonotic disease" [1]. According to WHO data, 30% of epilepsy cases are in endemic countries and 3% of epilepsy cases globally may be due to NCC [2]. It is considered the most common preventable cause of epilepsy in developing countries. It is a major cause of epilepsy in tropics and the commonest cause of focal seizures in India [3]. NCC is the cause of seizures in about 37% of otherwise healthy children above 3 years of age in India [4]. NCC in children has pleomorphic manifestations depending on the location, number, viability of cyst and host response. Diagnosis of NCC is

*Correspondence:
Mahesh Kumar
drmahesh81@yahoo.com
[1] BPS GMC (W) Khanpur Kalan, Sonepat, Haryana, India

mainly neuro-radiologic. Sometimes it is challenging to differentiate NCC from tuberculoma, especially in regions where both diseases are endemic and coexist. Magnetic resonance spectroscopy (MRS) is very helpful in differentiating similar looking lesions like tuberculoma, toxoplasmosis, mycosis, small abscesses, brain tumors, and even vascular malformations [5]. The management of NCC mainly includes antihelminthics, antiepileptics and corticosteroids. Many aspects related to diagnosis and treatment of NCC in children remain poorly understood despite advancing knowledge. Through this case series we are describing the pleomorphic clinical and radiological presentations of NCC in pediatric age group and also the role of MRS in diagnosing those patients.

Case presentation

This case series describes eight children with NCC: five were males and three were females. All the identifiers for the cases were removed to maintain participant anonymity. Informed consents/assent were taken from the guardians/parents of the participants. The age of first presentation ranged from 5 to 12 years. All children were of Asian ethnicity and Hindu by religion except one who was Muslim (case 1). With regard to eating habits all of them reported that they washed the eatables (fruits and vegetables) prior to consuming. Four cases used ground water and rest used municipal water supply for drinking. All cases had garbage disposal except one who disposed waste directly into drain (case 8). All cases had toilet facilities at home. Case 1 reported to have pigs around his household. None of the cases reported to have slaughter house near their premises. The frequency of consuming non-vegetarian food varied from once a week to once a month among all the cases. None of the cases reported to consume pork as non-vegetarian food. Socio-demographic characteristics are shown in Table 1.

All cases had focal seizures as the presenting symptom. Other associated symptoms were headache, loss of consciousness (in 3 cases each) and one (case7) had vomiting. None of the cases in this series had visual disturbances, neurological deficit, midline shift or hydrocephalous. Six cases (1, 3, 5, 6, 7, and 8) received sodium valproate as first line antiseizure medication (ASM) and two received phenytoin. Case 2 developed phenytoin toxicity after receiving it for 2 years and presented in outpatient department (OPD) with headache and ataxia. Serum phenytoin levels were 42 mg/l and brain MRI done was normal. Phenytoin was stopped and the patient was managed conservatively. The patient showed gradual improvement in headache by day two while ataxia improved by day 7. Two cases (3 and 4) reported to have recurrent NCC. No case reported to have break through

Table 1 Socio-demographic characteristics of the cases

Socio-demographic variables	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8
Age at diagnosis/ gender	5 years/male	8 years/female	8 years/female	7 years/male	11 years/male	12 years/male	6 years/female	8 years/male
Source of water	Ground water	Municipal supply	Ground water	Ground water	Ground water	Municipal	Municipal supply	Municipal supply
Method of water storage	Tank	Tank	Pots	Pots	Tank	Tank	Tank	Pots
Method of water purification	None	None	None	None	None	None	Boiling	None
Distance of garbage dumping from water source	> 100 feet	> 100 feet	> 100 feet	> 100 feet	> 100feet	> 100feet	> 100feet	> 100 feet
Pigs in and around household	Yes	No	No	No	No	No	No	No
Presence of slaughter house in vicinity	No	No	No	No	No	No	No	No
Peel fruits before eating	No	No	No	No	No	No	No	No
Frequency of patient eating outside home	Once a week	Once a month	Once a month	Once a month	Once a month	Week	Once a week	Once a week
Vegetarian or non-vegetarian?	Non-vegetarian	Non-vegetarian	Vegetarian	Non-vegetarian	Non-veg	Non-veg	Vegetarian	Vegetarian
Which type of non-vegetarian food do you consume most often?	Chicken	Chicken	None	Chicken	Chicken	Chicken/mutton	Chicken	None
Non-vegetarian food cooked at home or outside?	Home	Outside	None	Outside/home	Home	Home	Home	None

Table 2 Clinical profile of cases

clinical variables	Case 1	Case 2	Case 3	Case 4	Case 5	Case6	Case 7	Case 8
First symptom at diagnosis	Focal seizure	Focal seizure	Focal seizure	Focal seizure	Focal seizure	Focal seizure	Focal seizure	Focal seizure
Other symptoms	Loss of consciousness, headache	Loss of consciousness	Headache	Headache	No	No	Vomiting	Headache
Recurrent NCC/reinfestation	No	No	Yes	Yes	No	No	No	No
First antiepileptic used	Sodium valproate	Phenytoin	Sodium valproate	Phenytoin	Sodium valproate	Sodium valproate	Sodium valproate	Sodium valproate
Total duration of treatment with AED	12 months	24 months	24 months	12 month	On therapy	On therapy	On therapy	On therapy
AED side effects	None	Yes (ataxia, headache)	None	None	None	No	No	No
Steroid used	Prednisolone	Dexamethasone	Dexamethasone	Dexamethasone	Prednisolone	Prednisolone	Prednisolone	Prednisolone
Duration of steroid use	14 days	5 days	5 days	5 days	14 days	5 days	5 days	5 days
Compliance to treatment and follow-up visits	Good	Good	Poor and lost to follow up	Poor	Good	Good	Good	Good
Use of traditional medicine for treatment	No	No	Yes	No	No	No	No	No

seizures. None of the cases required antiseizure polytherapy for seizure control. Overall compliance was good except case 3 (loss to follow-up). All the cases of this series received Albendazole (dose 15 mg/kg/day) as antihelminthic therapy for 28 days. All cases used steroid for treatment. The duration of corticosteroids used varied from 5 to 14 days. Only case 3 reported to have received some traditional medicine. Table 2 shows the details of the clinical profile of the cases.

Magnetic resonance imaging (MRI) was done for neuroimaging details for all the cases. Out of total eight cases, five cases as shown in Figs. 1, 2, 3, 4 and 6 had multiple lesions of NCC. Of these two had single conglomerated ring enhancing lesions (SCREL) as shown in Figs. 3 and 4. Three cases had single discrete ring enhancing legions (SDREL) as shown in Figs. 5, 7 and 8. The commonest location of NCC lesions in brain parenchyma was parietal and parieto-occipital region. Varying stages of NCC such as colloidal-vesicular, granulo-nodular, and calcified nodular with perilesional oedema were seen. No case was reported to have hydrocephalus, midline shift and lepto-meningeal enhancement on MRI brain. MRS was done for three cases shown in Figs. 6, 7 and 8 to differentiate NCC from tuberculoma with findings of absence of lipid peak and choline/creatinine ratio less than 1.2. Table 3 depicts details of investigations and neuroimaging findings of all the cases.

Cases 1 to 8 are shown in following description and their corresponding Figures are respectively shown below with their descriptions.

Discussion

Cysticercosis is caused by ingestion of Taenia solium. In the intestinal tract of human host its eggs release the encysted larvae (oncospheres). These oncospheres penetrate the intestinal wall, enters the blood stream then are transported and deposited to various tissues of the body including brain, eyes, skin, and muscle. In these tissues oncospheres differentiate into metacestodes which further develop into cysticerci. In NCC, the brain parenchyma is most commonly infested with high rates of deposition at grey-white matter junction. It may be due to accumulation of metacestodes in small terminal blood vessels [6]. Most lesions are single ring enhancing lesion (SREL) or multiple ring enhancing lesion (MREL) in different stages of evolution [7]. Cystecerci of Taenia solium undergo four stages of involution in brain parenchyma. First stage is vesicular stage, showing cyst with translucent walls and viable scolex followed by colloidal stage characterised by cyst with a thick wall. Third stage is granular stage showing cyst with thicker wall and degenerated scolex. Final stage is calcification stage in which cyst develops into calcified nodule [8].

Clinical features of NCC vary widely in children and continue to pose a challenge in clinical practice. Most common symptoms are focal seizures, generalised seizures, neurological deficit and signs of raised intracranial pressure. In this series, all cases had focal seizures as primary manifestation of the disease. A study of 500 children with NCC in India reported that 95% of cases had seizures, of these 84% cases had focal seizures, 30% children showed symptoms of increased intracranial pressure, and 4% showed neurological deficit [9]. In this series, none of the cases reported to have increased

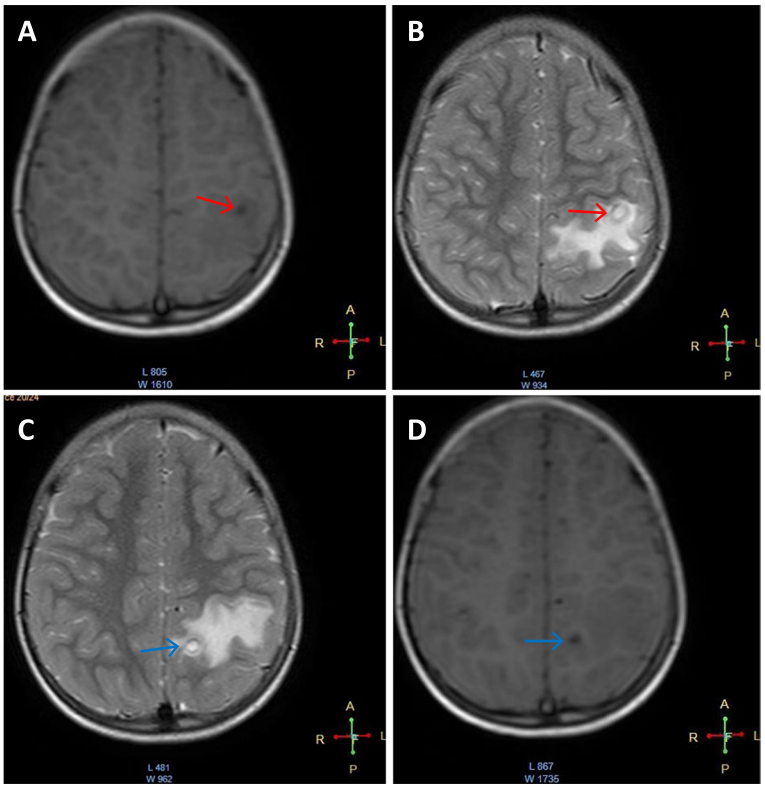

Fig. 1 T1 (**A**, **D**) and T2 (**B**, **C**) showing multiple cysts in colloid-vesicular stage cyst with extensive perilesional oedema in left parieto-occipital lobe

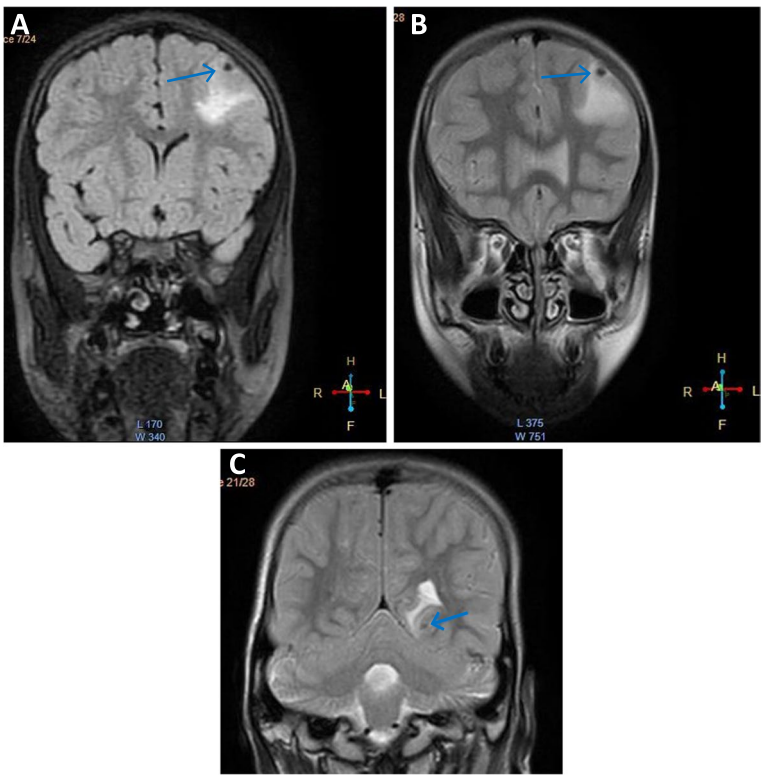

Fig. 2 FLAIR (**A**), T2 (**B**, **C**) shows multiple hypo intense cysts of NCC in granular-nodular stage with some perilesional oedema

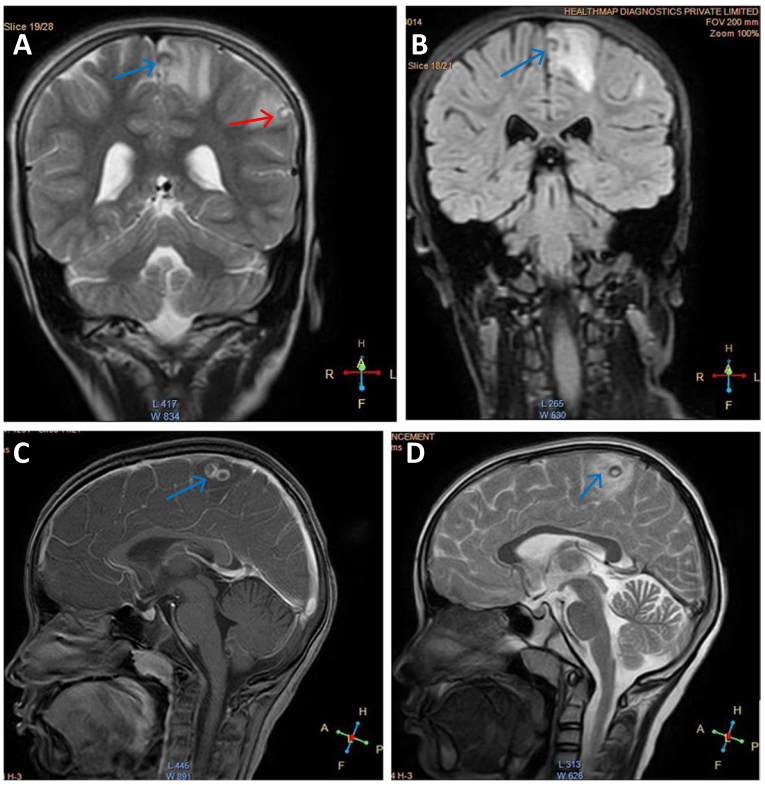

Fig. 3 T2 (**A**, **D**) and FLAIR (**B**) images showing two cysts in colloid-vesicular stage in parietal lobe with perilesional oedema. CISS (constructive interference in steady state) sequence (**C**) show conglomerated lesions with perilesional oedema

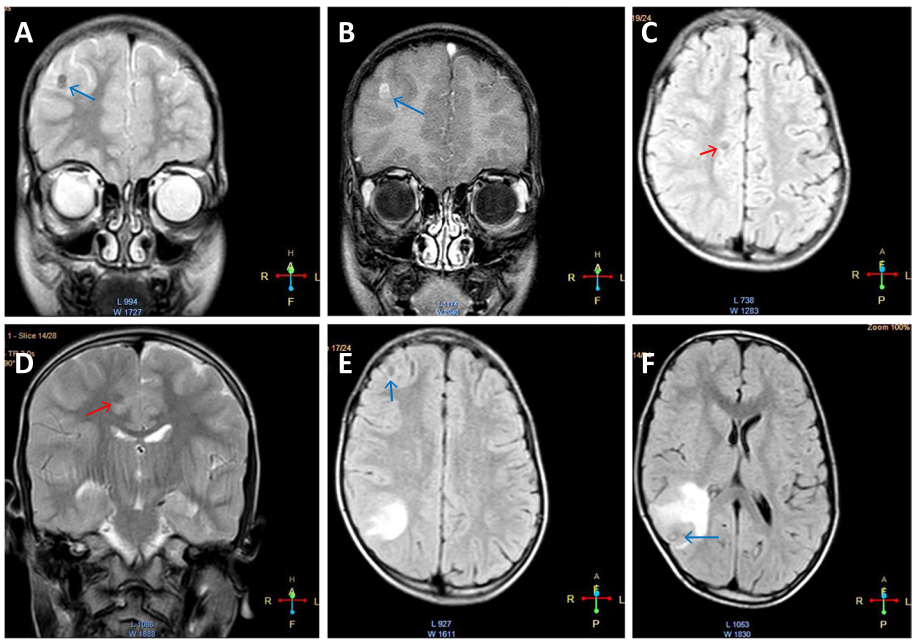

Fig. 4 Conglomerated cyst seen on T2 (**A**) and CISS (**B**) images (blue arrow). Two calcified nodular stage cyst of NCC seen as hypointense cyst on FLAIR (**C**, **E**) and T2 (**D**) in right frontal and parietal lobe. Single cyst in colloid vesicular stage NCC seen with perilesional oedema on T2 (**F**) in right parietal lobe

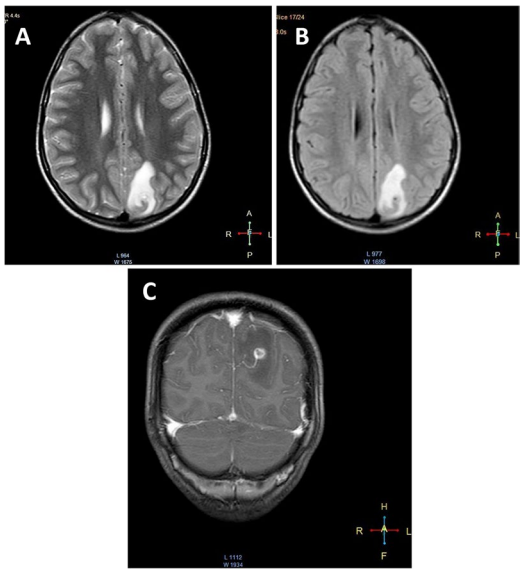

Fig. 5 T2 (**A**), FLAIR (**B**) and CISS sequence (**C**) shows solitary cysticercal lesion in occipital lobe in vesicular nodular stage with perilesional oedema

but were commonly reported as neurotuberculosis [10]. In the present series two cases were identified to have single conglomerated ring enhancing lesion (SCREL). Conglomerated ring enhancing lesions are best seen on MRI due to its better soft tissue resolution and multiplanar capabilities. Rajshekhar et al. [11] described atypical" type B" lesions having two confluent discs or rings or a combination in 4 out of 25 and 5 out of 43 biopsy proven cases of NCC. Recently Kumar et al. [12] reported "atypical" lesion having bilobed, septate or disc configurations in 28.8% of cases on MRI. These atypical lesions often persist unresolved for a longer period and may even be associated with a higher risk of seizure recurrence because of persistence of calcified focus.

Out of eight NCC cases, three had SDREL with perilesional oedema. The commonest neuroimaging finding of NCC is "single small ring enhancing lesion"- a single lesion, < 20 mm with perilesional oedema. These solitary lesions usually resolve during treatment between few weeks to one year whereas conglomerated lesions takes longer time to resolve [13]. Reappearance of NCC at the same site after documented resolution

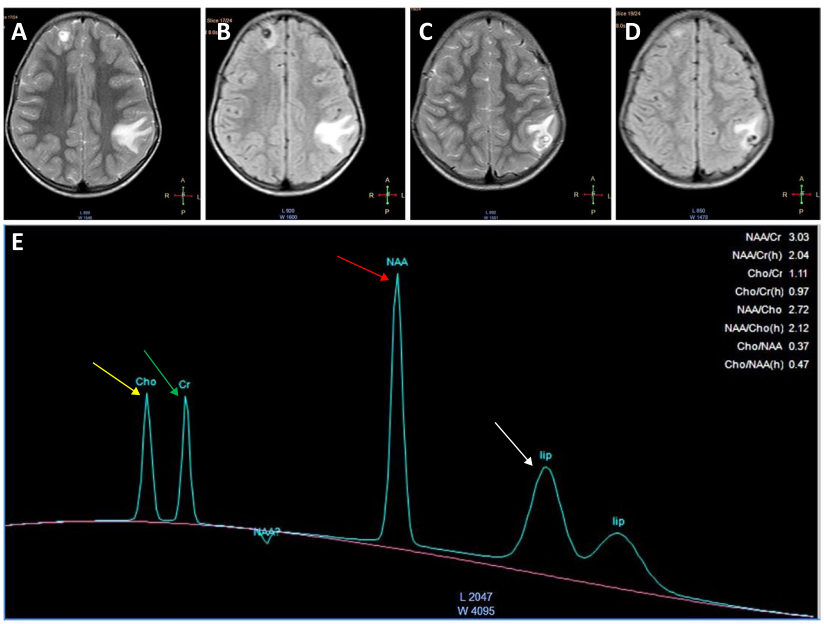

Fig. 6 T2 (**A**, **C**) and FLAIR (**C**, **D**) shows two lesions in frontal and parieto-occipital lobes. Scolex clearly visible in T2 (**A**) and FLAIR (**B**) suggestive of NCC. Magnetic resonance spectroscopy (**E**) shows NAA peak (red arrow) and Cho:Cr (choline:creatine) = 1.1. Cr (green arrow) and Cho (yellow arrow) peaks are also seen

intracranial pressure, neurological deficit or visual disturbances.

Neuroimaging showed five cases in colloidal-vesicular stage, two cases in granular-nodular stage, and one in calcified nodular stage in brain parenchyma. Conglomerated lesions were thought to be unusual in NCC has been sparsely described. We have reported two cases with recurrent NCC both having recurrent lesion at different locations after 2.5 and 2 years interval. Singh et al. [14] reported four cases of reappearing CT lesions; two at the same and two at different locations. It was postulated that recurrent lesions at the same

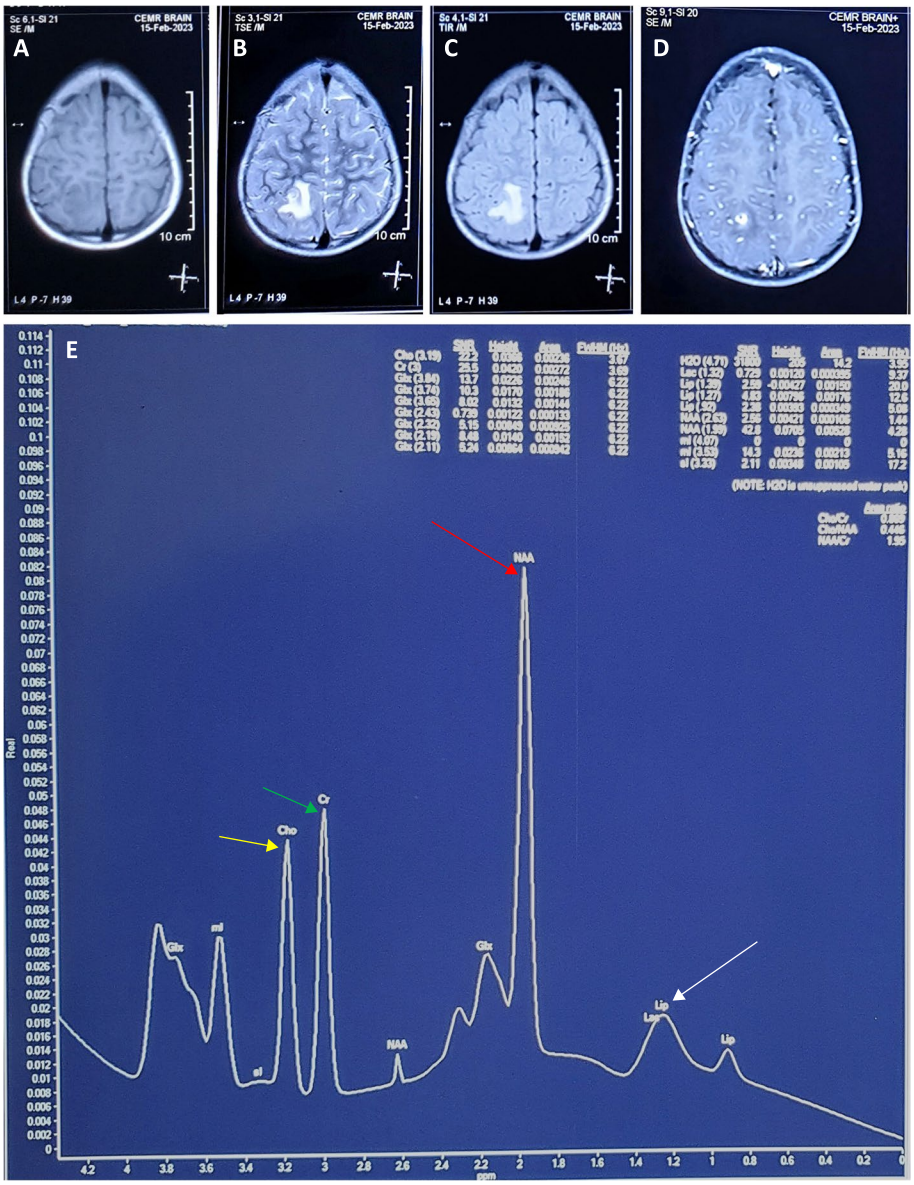

Fig. 7 T2 (**A**), T2 (**B**), FLAIR (**C**) and contrast images (**D**) show single parieto-occipital cyst with central hypointense scolex suggestive of lesion in colloid vesicular stage of NCC with surrounding oedema. Magnetic resonance spectroscopy (**E**) shows an absence of lipid peak (white arrow) and Cho:Cr (choline: creatine) = 0.89. NAA (red arrow), Cr (green arrow), and Cho (yellow arrow) peaks are also seen

location are secondary to co-localisation of a multiple stream of cysts or viable meta-cestode larvae in a particular brain region which appear and become active at different times, whereas recurrent lesions in different locations are secondary to recurrent auto infections in taeniid carriers. Kumar et al. [12] described three cases of recurrent symptomatic solitary NCC at a location different from initial site after 2.5, 4, and 7.5 years interval. The reason given was that all new granulomas resulted from reinfection rather than persistent of initial infection. VDe Souza et al. [15] described natural history of NCC in 81 patients on serial MRI over 24 months and did not report a single recurrence of NCC. In an MR-based prospective study, Kumar N et al. [16] evaluated natural course of 59 cases of single conglomerated granuloma (SCG) over 3 years without any antihelminthics or steroid treatment and did not report a single case of recurrent granuloma.

All cases in this series received antihelminthic therapy as Albendazole (15 mg/kg/day) for 28 days and corticosteroids for 5–14 days. Singhi et al. [9] reported new lesions in around 4% of NCC patients during long term

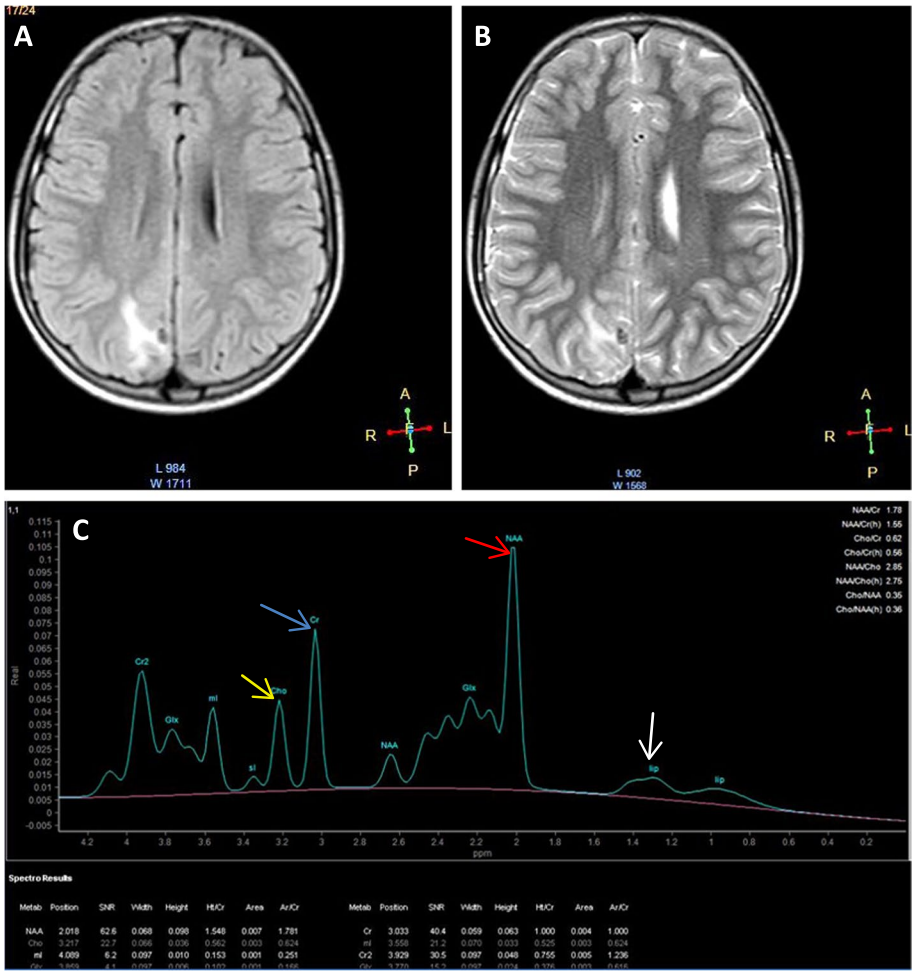

Fig. 8 FLAIR (**A**) and T2 (**B**) images show single lesion in granular nodular stage of NCC in the occipital lobe. Magnetic resonance spectroscopy (**C**) shows an absence of Lipid peak (white arrow) and Cho:Cr = 0.62. NAA (red arrow), Cr (green arrow), and Cho (yellow arrow) peaks are also seen

clinical and radiological follow up in 500 children with parenchymal NCC.

There are no clear guidelines for management and diagnosis of paediatric NCC. A recent meta-analysis involving 16 trials and another involving 15 trials have concluded that Albendazole was effective in early resolution and radiological clearance of lesions and also preventing seizures [17, 18]. Corticosteroids are administered to reduce host inflammatory reaction and increase level of Albendazole sulfoxide in plasma [19]. Antiseizure medication (ASM) used to treat NCC are similar to those used for other seizure disorders. In our series six cases received sodium valproate and two received phenytoin.

Case 2 in this series presented with ataxia and headache as manifestations of acute phenytoin toxicity at serum phenytoin levels of 42 mg/L. Acute phenytoin toxicity manifests as nystagmus (95%), ataxia (88%), lethargy and seizures. Ataxia usually precedes nystagmus in children. Phenytoin levels > 30 mg/L may cause side effects as ataxia, dysarthria, and seizure. Somnolence is often seen at serum phenytoin levels > 40 mg/L [20].

MRS was done for three cases in this series to differentiate between NCC and tuberculoma. Case 6 shows N-acetyl aspartate (NAA) peak, Cho/Cr (choline: creatine) = 0.62 and absence of lipid peak. Case 7 had NAA peak and Cho/Cr = 0.89 with absence of lipid peak. In case 8 there was NAA peak and absence of lipid peak with choline/Cr = 1.1. The presence of a high lipid peak and an increased choline/creatine ratio were seen consistently with tuberculoma. Lipid peak may be attributed to the presence of large lipid fraction in Mycobacterium tuberculosis and the increase in choline/creatine ratio is probably due to damage to brain tissue, which is minimal in NCC [21]. MRI and MRS features differentiating NCC from tuberculoma are shown in Table 4 [22, 23].

Table 3 Investigations and radiological profile of cases

Investigative and radiological profile	Case 1 (Fig. 1)	Case 2(Fig. 2)	Case 3 (Fig. 3)	Case 4 (Fig. 4)	Case 5 (Fig. 5)	Case 6 (Fig. 6)	Case 7 (Fig. 7)	Case 8 (Fig. 8)
Number of lesions	Multiple	Multiple	Multiple	Multiple	Single	Multiple	Single	Single
Location of lesions	Parietal/occipital	Parietal	Parietal	Parietal/frontal	Occipital	Parieto-occipital/frontal	Parieto-occipital	Occipital
Type of lesion (SDREL/SCREL/MREL)	MREL	MREL	SCREL/SDREL	SCREL/SDREL	SDREL	MREL	SDREL	SDREL
Stage of lesion at diagnosis	Colloid vesicular	Granulo-nodular	Colloid vesicular	Calcified nodular/colloid vesicular	Vesicular nodular	Granulo-nodular	Colloidal vesicular	Colloidal vesicular
Perilesional oedema	Present	Present	Present	Present	Present	Present	Present	Present
Follow-up neuroimaging done after how many months of diagnosis	6 months	6 months	6 months	Not done	Not done	Not done	Not done	Not done
Finding on follow-up neuroimaging.(resolution with or without calcification; non-resolution with active lesion still persisting)	Resolution without calcification	Resolution with calcification	Resolution without calcification	Unknown	Imaging pending	Imaging pending	Imaging pending	Imaging pending
Persistence of lesion with surrounding oedema after completion of cysticidal treatment and requiring a second course of the same	No	No	No	Unknown	Pending	Pending	Pending	Pending
Intraretinal cysticercosis on orbital sonogram or fundoscopy	No	No	No	No	No	No	No	No
Evidence of cysticercosis outside CNS	No	No	No	No	No	No	No	No
Evidence of taeniasis on stool examination	No	No	No	No	No	No	No	No
Other test (MTX, CXR, HIV ELISA)	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA-negative	MTX-non-reactive/CXR-normal/HIV ELISA- negative	MTX-non-reactive/CXR-normal/HIV ELISA- negative
MRS	not done	not done	not done	not done	not done	Absence of lipid peak, choline/Cr = 0.62, NAA peak seen	Absence of lipid peak, choline/Cr = 0.89, NAA peak seen	Absence of lipid peak, choline/Cr = 1.1, NAA peak seen

MTX Mantoux test, *CXR* chest X-ray, *MRS* magnetic resonance spectroscopy, *NAA* N-Acetyl aspartate

Table 4 MRI and MRS features differentiating NCC from tuberculoma [22, 23]

	Neurocysticercosis	Tuberculoma
Margin	Regular	Irregular with conglomeration
Size	Smaller (< 20 mm)	Larger (> 20 mm)
Signal intensity	T1W hypointense, T2W hyperintense	T1W and T2W hypo- to iso-intense, often with hyperintense rim, rarely onion-peal appearance (alternating rings of hypo- and hyper-intense signals)
DWI	Variable	Variable, those with T2W hyperintense centers usually show restricted diffusion
Midline shift	No	Yes
MR spectroscopy	• Elevated succinate (related to aerobic metabolism of scolices), lactate, and alanine levels	• Elevated lipid (mycobacteria which mainly composed of lipid), lactate, and Cho levels (associated benign inflammatory reaction)
	• Low NAA and Cr levels	• Even lower NAA and Cr levels
	• Cho/Cr ratio < 1	• Cho/Cr ratio > 1
		• Prominent decrease in NAA/Cr, slight decrease in NAA/Cho

Cho Choline, *Cr* Creatine, *DW1 D*iffusion-weighted imaging, *T1W* T1-weighted, *T2W* T2-weighted

Conclusion

Despite the advances in neuroimaging, accurate diagnosis of NCC is still sometimes difficult, which is related to the pleomorphic nature of disease and significant overlapping features with tuberculoma. A combination of proper diagnostic criteria and neuroimaging (MRI/MRS) findings are helpful in making the diagnosis without invasive and potentially harmful investigations in paediatric patients.

Abbreviations

NCC	Neurocysticercosis
CNS	Central nervous system
SCG	Solitary cystic granuloma
SREL	Single ring enhancing lesion
SDREL	Single discrete ring enhancing lesion
SCREL	Single conglomerated ring enhancing lesion
MREL	Multiple ring enhancing lesion
MRI	Magnetic resonance imaging
MRS	Magnetic resonance spectroscopy
NAA	N-acetyl aspartate

Acknowledgements

Not applicable.

Authors' contributions

SK contributed to the conception/design of the work and helped in acquisition of data. RM contributed in design of the work, acquisition of data and editing of the manuscript. MK contributed in design of the work and was the major contributor in writing the manuscript and revised it critically for important intellectual content. AY helped in collection of the data. All authors have read and approved the final manuscript.

Funding

Nil.

Availability of data and materials

All data generated or analysed during this study are included in this published article [and its supplementary information files].

Declarations

Ethics approval and consent to participate

Written informed consent/assent was taken from parent/guardian of the patient.

Consent for publication

Written informed consent/assent for publication of their clinical details and/or clinical images was obtained from parent/guardian of the patient.

Competing interests

The authors declare that they have no competing interests.

Received: 16 March 2023 Accepted: 17 August 2023
Published online: 08 December 2023

References

1. World Health Organization, ICONZ - Integrated control of neglected zoonotic diseases & United Kingdom. Dept for International Development Research in Use. (2011). The control of neglected zoonotic diseases : community based interventions for NZDs prevention and control : report of the third conference organized with ICONZ, DFID-RiU, SOS, EU, TDR and FAO with the participation of ILRI and OIE : 23 - 24 November 2010, WHO Heaquarters, Geneva, Switzerland. World Health Organization. https://apps.who.int/iris/handle/10665/44746.
2. World Health Organization [Internet]. *Taeniasis/Cysticercosis;* 2022 Jan 11 [cited 2022 Feb 8]. Available from : http://www.who.int/mediacentre/factsheets/fs376/en/.
3. Singh G (1997) Neurocysticercosos in South-Central America and the Indian subcontinent. A comparative evaluation Arq Neuropsiquiatr 55(3A):349–356
4. Udani V (2005) Pediatric epilepsy - an Indian perspective. Indian J Pediatr 72(4):309–313
5. Mishra AM, Gupta RK, Jaggi RS, Reddy JS, Jha DK, Husain N, Prasad KN, Behari S, Husain M (2004) Role of diffusion-weighted imaging and in vivo proton magnetic resonance spectroscopy in the differential diagnosis of ring-enhancing intracranial cystic mass lesions. J Comput Assist Tomogr. 28(4):540–7
6. Lotz J, Hewlett R, Alheit B, Bowen R (1988) Neurocysticercosis: correlative pathomorphology and MR imaging. Neuroradiology 30(1):35–41
7. Ahuja GK, Behari M, Prasad K, Goulatia RK, Jailkhani BL (1989) Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the cause? J Neurol Neurosurg Psychiatry 52(7):915–916
8. Garcia HH, Del Brutto OH (2005) Cysticercosis Working Group in Peru. Neurocysticercosis: updated concepts about an old disease. Lancet Neurol. 4(10):653–61
9. Singhi P, Suthar R, Deo B, Malhi P, Khandelwal NK (2017) Long-term clinical and radiologic outcome in 500 children with parenchymal neurocysticercosis. Pediatr Infect Dis J 36(6):549–555
10. Rajshekhar V, Haran RP, Prakash GS, Chandy MJ (1993) Differentiating solitary small cysticercus granulomas and tuberculomas in patients with

epilepsy. Clinical and computerized tomographic criteria. J Neurosurg. 78(3):402–7
11. Rajshekhar V, Chacko G, Haran RP, Chandy MJ, Chandi SM (1995) Clinicoradiological and pathological correlations in patients with solitary cysticercus granuloma and epilepsy: focus on presence of the parasite and oedema formation. J Neurol Neurosurg Psychiatry 59(3):284–286
12. Sujit Kumar GS, Rajshekhar V (2004) New solitary cysticercus granulomas causing recurrent symptoms in patients with resolved solitary granulomas. Neurol India 52(2):265–267
13. Singh MK, Garg RK, Nath G, Verma DN, Misra S (2001) Single small enhancing computed tomographic (CT) lesions in Indian patients with new-onset seizures A prospective follow-up in 75 patients. Seizure. 10(8):573–8
14. Singh G, Bhatia RS, Khurana D, Khurana SB (1999) Reappearing CT lesions: 4 cases. Neurol India 47(1):47–50
15. de Souza A, Nalini A, Kovoor JM, Yeshraj G, Siddalingaiah HS, Thennarasu K (2010) Natural history of solitary cerebral cysticercosis on serial magnetic resonance imaging and the effect of albendazole therapy on its evolution. J Neurol Sci 288(1–2):135–141
16. Kumar N, Garg RK, Malhotra HS, Gupta RK, Verma R, Sharma PK (2016) Natural course of typical and atypical parenchymal solitary cysticercus granuloma of the brain: a 3-year prospective clinico-radiological study. Neuroradiol J 29(1):19–29
17. Monk EJM, Abba K, Ranganathan LN (2021) Anthelmintics for people with neurocysticercosis. Cochrane Database Syst Rev. 6(6):CD000215
18. Otte WM, Singla M, Sander JW, Singh G (2013) Drug therapy for solitary cysticercus granuloma: a systematic review and meta-analysis. Neurology 80(2):152–162
19. Singhi P, Saini AG (2019) Pediatric Neurocysticercosis. Indian J Pediatr 86(1):76–82
20. Shukla A, Sankar J, Verma A, Dubey N (2013) Acute phenytoin intoxication in a 4-year-old mimicking viral meningoencephalitis. BMJ Case Rep. 2013:bcr2013009492
21. Gupta RK, Pandey R, Khan EM, Mittal P, Gujral RB, Chhabra DK (1993) Intracranial tuberculomas: MRI signal intensity correlation with histopathology and localised proton spectroscopy. Magn Reson Imaging 11(3):443–449
22. Pretell EJ, Martinot C Jr, Garcia HH, Alvarado M, Bustos JA, Martinot C (2005) Cysticercosis Working Group in Peru. Differential diagnosis between cerebral tuberculosis and neurocysticercosis by magnetic resonance spectroscopy. J Comput Assist Tomogr. 29(1):112–4
23. Del Brutto OH, Rajshekhar V, White AC Jr, Tsang VC, Nash TE, Takayanagui OM, Schantz PM, Evans CA, Flisser A, Correa D, Botero D, Allan JC, Sarti E, Gonzalez AE, Gilman RH, García HH (2001) Proposed diagnostic criteria for neurocysticercosis. Neurology 57(2):177–183

Publisher's Note