Radiology Case Reports 18 (2023) 2960–2965

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journal homepage: www.elsevier.com/locate/radcr

Case Report

Radiology findings in neurocysticercosis: A case report ☆

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ARTICLE INFO

Article history:
Received 15 February 2023
Revised 23 May 2023
Accepted 25 May 2023
Available online 20 June 2023

Keywords:
Neurocysticercosis
CT scan
Magnetic resonance imaging
Magnetic resonance spectroscopy
Neuroradiology

ABSTRACT

Neurocysticercosis is a serious underreported tropical disease caused by the ingestion of *Taenia solium* eggs through fecal-oral contact. The infection can affect any organ but frequently affects the central nervous system, eyes, and muscles, and is able to remain dormant for years in the brain. Medical imaging is crucial in making the diagnosis of neurocysticercosis as there are no identifiable clinical symptoms of the condition. In this case, we present a 71-year-old man with neurocysticercosis diagnosed by CT scan, MRI and MR spectroscopy. Calcified nodules were found with surrounding vasogenic edema on CT scan. Magnetic Resonance Imaging (MRI) showed multiple lesions that were hypointense on T1-weighted image, hyperintense rim on T2-weighted image, with ring enhancement on post-contrast scanning characterizing granular nodular stage and multiple lesions that were hypointense on T1-weighted image and no signal on T2-weighted image characterizing nodular calcified stage of the disease. MR Spectroscopy showed decreased levels of choline, creatine, NAA, NAA/Cr and Cho/Cr ratio with increased levels of lactate and lipid.

Introduction

Neurocysticercosis is a serious underreported tropical disease caused by the ingestion of the eggs from pig tapeworm *Taenia solium* [1]. It is endemic in most low-income countries where pigs are raised can remains one of the most important causes of seizures in the world, contributes to 30% cases of epilepsy in endemic areas [2,3]. This disease is acquired through consumption of food contaminated with feces of a *T. solium* tapeworm carrier through fecal-oral contract. In poor hygiene, eggs of tapeworm are shed in stool and contaminate food. Ingestion of eggs followed by exposure of eggs to gastric acid in human stomach turn eggs to larval cysts (oncospheres). Even though infection can affect any organ, it most frequently affects the central nervous system, the eyes, and the muscles. Once in the brain, the larval cysts initially only cause a minor immune reaction and can survive there for years. After the parasite dies, the host typically experiences an inflammatory response that leads to diverse manifestations of symptomatic neurocysticercosis with low sensitivity and specificity for serologic tests. Therefore, medical imaging is crucial in

☆ Competing Interests: The authors have declared that no competing interests exist.

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https://doi.org/10.1016/j.radcr.2023.05.054

making the diagnosis of neurocysticercosis because there are no identifiable clinical symptoms of the condition [3–5]. In this report, we present an adult man with neurocysticercosis presented with intermittent headaches, loss of vision, weakness of left extremities and no seizures, together with CT scan, MRI and MR spectroscopy findings.

Case presentation

A 71-year-old man presented with intermittent headaches in the past 4 months improved with analgesics. The patient also had abrupt loss of vision in the past 4 months and was referred to Cicendo Eye Center for examination. Weakness of extremities was felt since 1 year ago especially with left extremities. The patient went for therapy and was able to walk with assistance. The patient had no seizures, anosmia, hearing loss, or vomiting. Patient had good appetite and no weight loss. Patient had no history of tuberculosis, hypertension, diabetes, or malignancy in the family. Patient had history of stroke 1 year ago. Clinical examinations showed no abnormality. Routine laboratory and immunoserology examinations were within normal limits, especially tests for human immunodeficiency virus antibodies were negative. Computed tomography (CT), MRI, and MR spectroscopy were conducted to confirm the diagnosis.

Computed tomography (CT) of the brain was conducted and showed multiple calcified nodules showing nodular calcified stage at right subcortical parietal lobe, left thalamus, left basal ganglia and granular nodular stage at right cortical-subcortical occipital lobe (Fig. 1).

MR image was obtained and showed a small spherical lesion at right subcortical occipital lobe showing different intensities (Fig. 2). Lesion was hypointense on T1-weighted image, hyperintense rim on T2-weighted image. Ring enhancement was noted on the contrast enhanced T1 and blooming artefact on SWI. These findings suggesting granular nodular

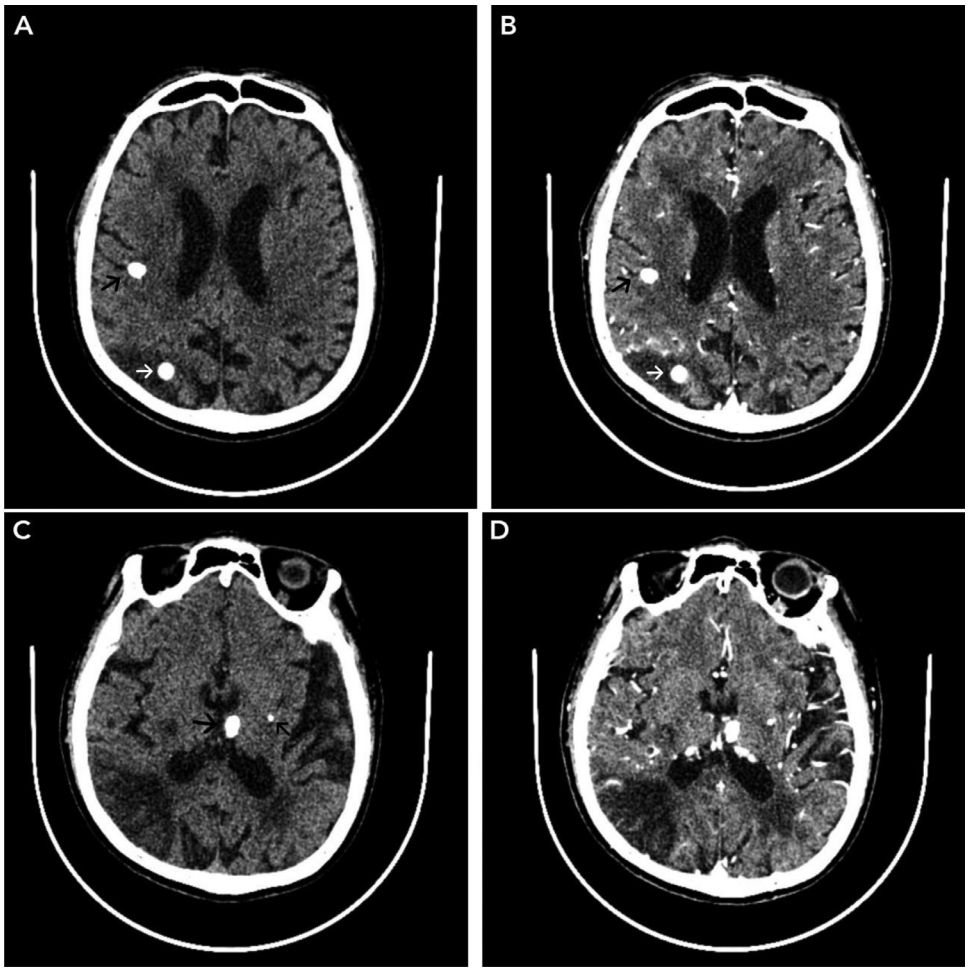

Fig 1 – Axial view CT scan without contrast (A, C) and with contrast (B, D) showing multiple calcified nodule with well-defined border at right subcortical parietal lobe, left thalamus, and left basal ganglia, with the largest diameter of 1.20 cm at left thalamus, characterizing nodular calcified stage (black arrow). There is also a calcified nodule with well-defined border, and diameter of 0.90 cm at right cortical-subcortical occipital lobe (white arrow). There was hypodense lesion surrounding the lesion that resulted in narrowing of surrounding sulci and gyri suggesting surrounding vasogenic edema characterizing granular nodular stage. No enhancement was seen at postcontrast scanning.

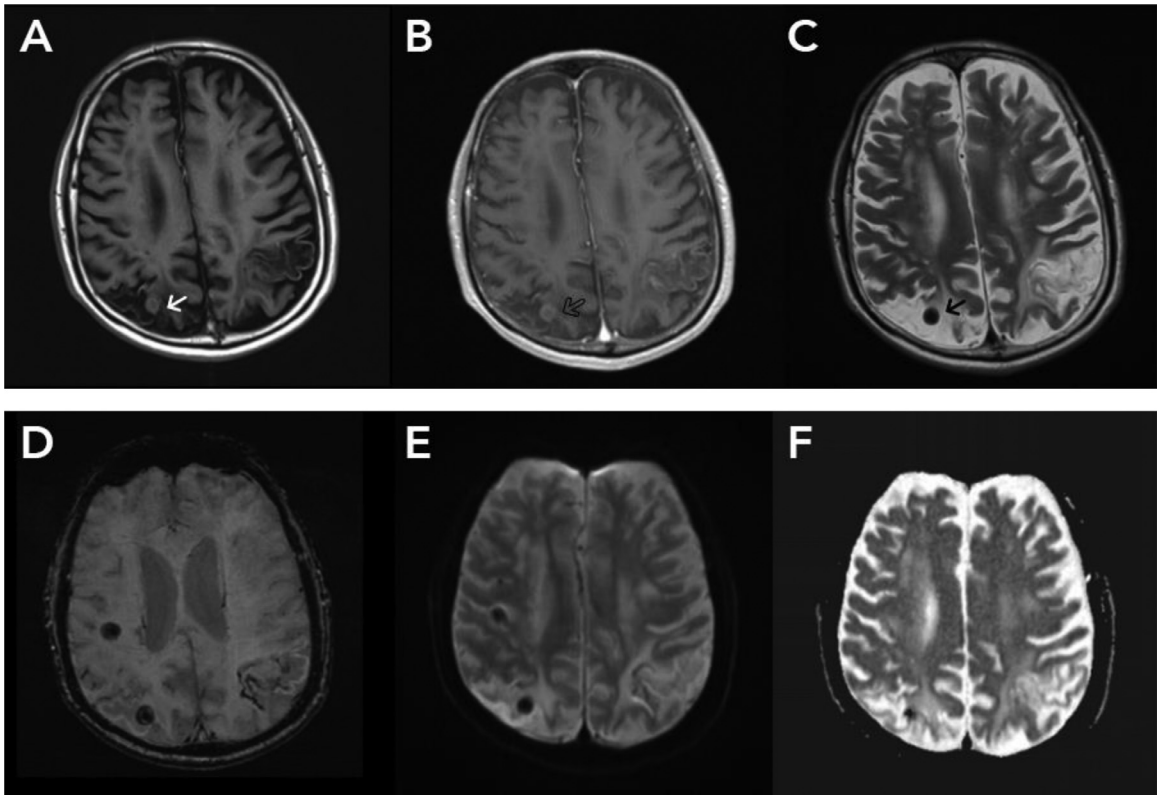

Fig. 2 – Granular nodular stage on axial view T1-weighted image (A), Postcontrast T1-weighted image (B), T2-weighted image (C), SWI (D), DWI (E), and ADC (F) showing single, spherical lesion with well-defined border, diameter of 0.90 cm at right subcortical occipital lobe with hypointense on T1-weighted image (A, white arrow), hyperintense rim on T2-weighted image (C, black arrow), ring enhancement (B, blank arrow), and blooming artefact (D). No restricted area was seen within the lesion on DWI and ADC. (E,F).

stage. Other multiple singular lesions were seen at left juxtacortical temporal lobe, right juxtacortical parietal lobe and left thalamus showing different intensities (Figs. 3, and 4). Lesion was also hypointense on T1-weighted image with no signal on T2-weighted image, with blooming artefact on SWI which suggesting nodular calcified stage. No restricted area was seen within the lesion on DWI-ADC on both stages.

MR Spectroscopy was conducted (Fig. 5). Minor shift scale (approximately -0.5 ppm) was made based on major resonances of N-acetylaspartate (NAA), creatine (Cr), and choline (Cho) in the spectroscopy control from the normal parenchyma. Increase in lactate and decrease in choline and N-acetylaspartate were seen which suggesting diagnosis of an inflammatory lesion. Levels of NAA/Cr ratio (0.38) and Cho/Cr ratio (1.21) were seen to be below 1.5.

Discussion

Manifestations of neurocysticercosis are variable depending on the location, stage of cysts in the nervous system, and host immune response. Escobar and Nieto described four essential forms of neurocysticercosis according to the location which are meningeal, ventricular, parenchymatous, and mixed forms where most cysts are found in parenchymal part of brain, particularly at grey-white cortical junction [6]. This is similar to this case where cysts are found at right subcortical parietal lobe, left thalamus, left basal ganglia and right cortical-subcortical occipital lobe. Parenchymal cysts are usually small and rarely larger than 10 mm in diameter, while in this case, the size was relatively bigger (1.20 cm) [5]. Singhi et al. [6] reported seizures in 70%-90% of parenchymal neurocysticercosis cases (particularly in granular or calcified lesions), while in this case, patient was presented with intermittent headaches and no seizures. Oscar et al. [7] reported patients with calcified neurocysticercosis experience headaches about five times more frequently compared to matched controls without neurocysticercosis. Due to various manifestations of neurocysticercosis, diagnosis in mainly made by neuroimaging.

However, it remains a challenge to diagnose neurocysticercosis lesions when a patient has intracranial calcifications as the differential diagnoses includes physiologic/age-related, dystrophic, infectious, vascular, neoplastic, metabolic, inflammatory, and toxic diseases. Our patient was living in a developing country so infectious etiology was considered first. There are many possibilities in infectious diseases including CMV, Herpes, Toxoplasmosis, Rubella, Zika, HIV, Neurocysticercosis, mycobacterium tuberculosis, and cryptococcus neoformans

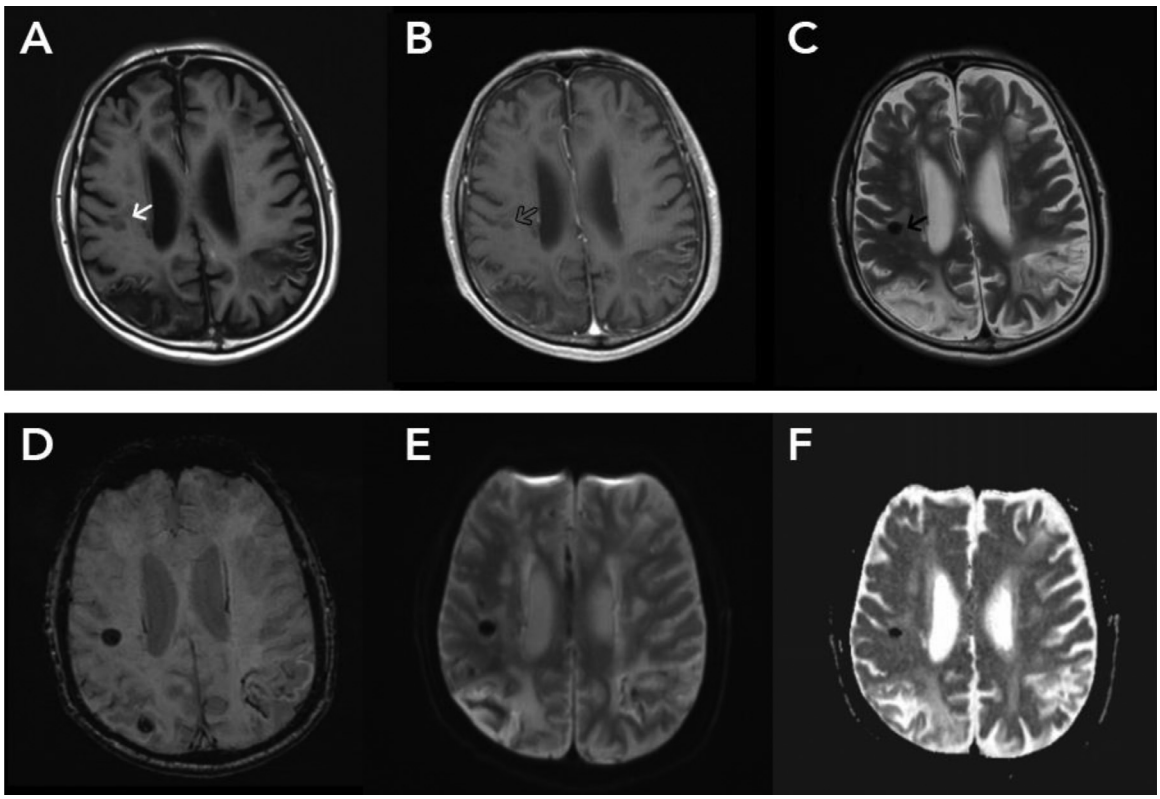

Fig. 3 – Nodular calcified stage at right subcortical parietal lobe seen on axial view T1-weighted image (A), Postcontrast T1-weighted image (B), T2-weighted image (C), SWI (D), DWI (E), and ADC (F) showing spherical lesions with well-defined border at right subcortical parietal lobe, diameter of 0.90 cm with hypointense on T1-weighted image (A, white arrow), no signal on T2-weighted image (C, black arrow), and blooming artefact on SWI (D). No restricted area was seen within the lesion on DWI and ADC (E, F).

[8]. Patient did not have a fever; no restriction area was found in diffusion-weighted image and ADC so pyogenic brain abscess was not considered. Appearance of ring enhancement is helpful in narrowing differential diagnosis, where 2 important causes of ring-enhancing lesions are neurocysticercosis and tuberculomas. In endemic areas, cysticercal cyst was diagnosed with a small (<20 mm), peripheral situated solitary ring-enhancing lesion whereas presence of raised intracranial pressure, progressive focal neuro deficit, size of CT lesion > 20 mm with lobulated irregular shape and marked edema causing midline shift may indicate tuberculoma. CT lesions are found to be 0.38-1.20 cm in diameter without marked edema which leads to the diagnosis of neurocysticercosis [6,9].

Neurocysticercosis has different neuroimaging findings depending on the stage of lesion. Cyst evolves through four different stages in the brain parenchyma which can be classified into 4 stages: vesicular stage, where cyst is filled with clear fluid, has a thin semitransparent wall and an eccentric opaque 4-5 mm scolex, and usually is asymptomatic; colloidal stage, where cyst starts to degenerate and elicits inflammatory response which results in replacement of clear cyst fluid with gelatinous material due to hyaline degeneration of larva; granular nodular stage, where cyst contracts and walls are replaced by lymphoid nodules and necrosis, with transformation of scolex into mineralized granules. CT depicts isoattenuating cyst with peripheral edema and enhancement after contrast administration; nodular calcified stage, where granulation tissue is replaced by collagen and calcification with no edema. CT shows calcified nodule without contrast enhancement [6]. In this case, CT shows multiple calcified nodules with well-defined border which suggesting nodular calcified stage and a calcified nodule, with well-defined border and hypodense lesion surrounding the lesion suggesting surrounding vasogenic edema which suggesting granular nodular stage.

Hayama et al. described MR images of granular nodular cysts as cysts with hyperintense on T1 and T2 weighted images with thick ring enhancement, whereas lesion is hypointense in T1-weighted image and hyperintense in T2-weighted image with ring enhancement. Variations may be due to ongoing mineralization of the cyst. Nodular calcified stage cysts are described as hypointense lesions on all MR imaging sequences, similar to this patient with hypointense lesion on T1-weighted image and no signal on T2-weighted image. Blooming artefacts were seen in various number of compounds, including calcification in neurocysticercosis [10,11].

MR Spectroscopy helps in differentiating between infectious and noninfectious lesions. The presence and/or ratio of various tissue metabolites can be analyzed by MR Spectroscopy to provide information about the possible extent and

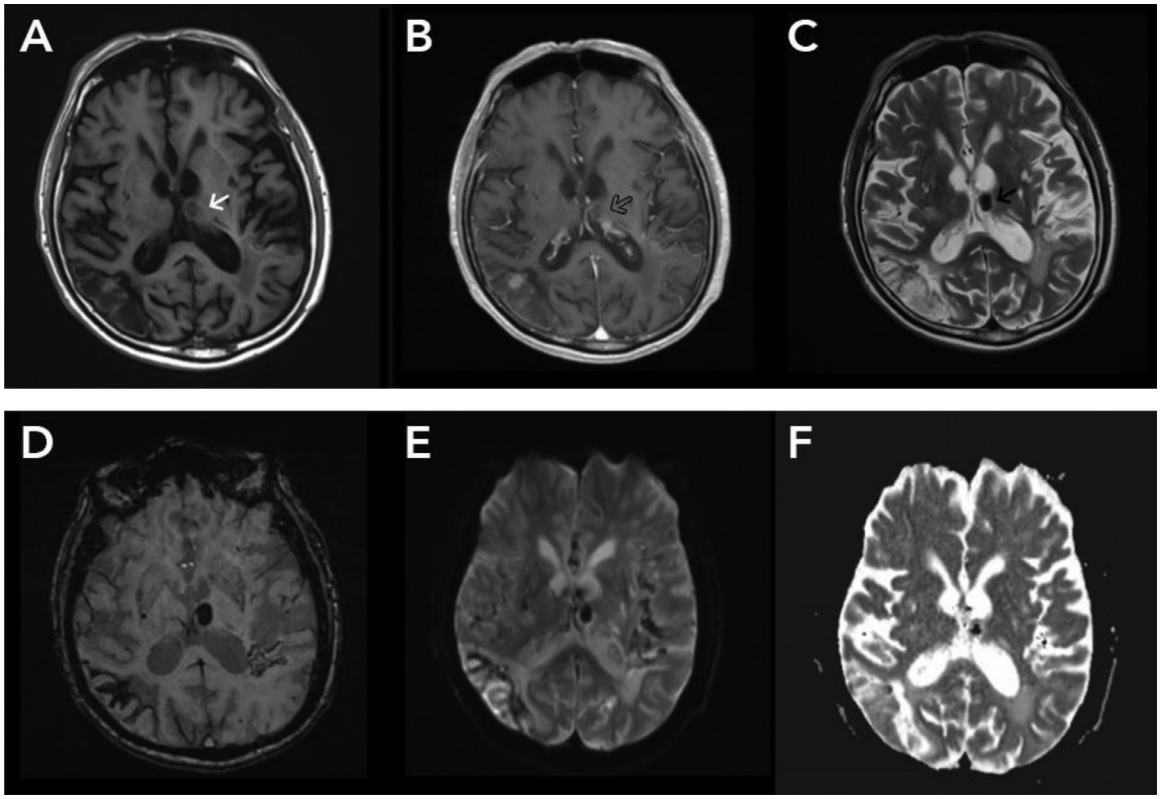

Fig. 4 – Nodular calcified stage at left thalamus seen on axial view T1-weighted image (A), Postcontrast T1-weighted image (B), T2-weighted image (C), SWI (D), DWI (E), and ADC (F) showing spherical lesion with well-defined border, diameter of 1.20 cm with hypointense on T1-weighted image (A, white arrow), no signal on T2-weighted image (C, black arrow), and blooming artefact on SWI (D). No restricted area was seen within the lesion on DWI and ADC (E, F).

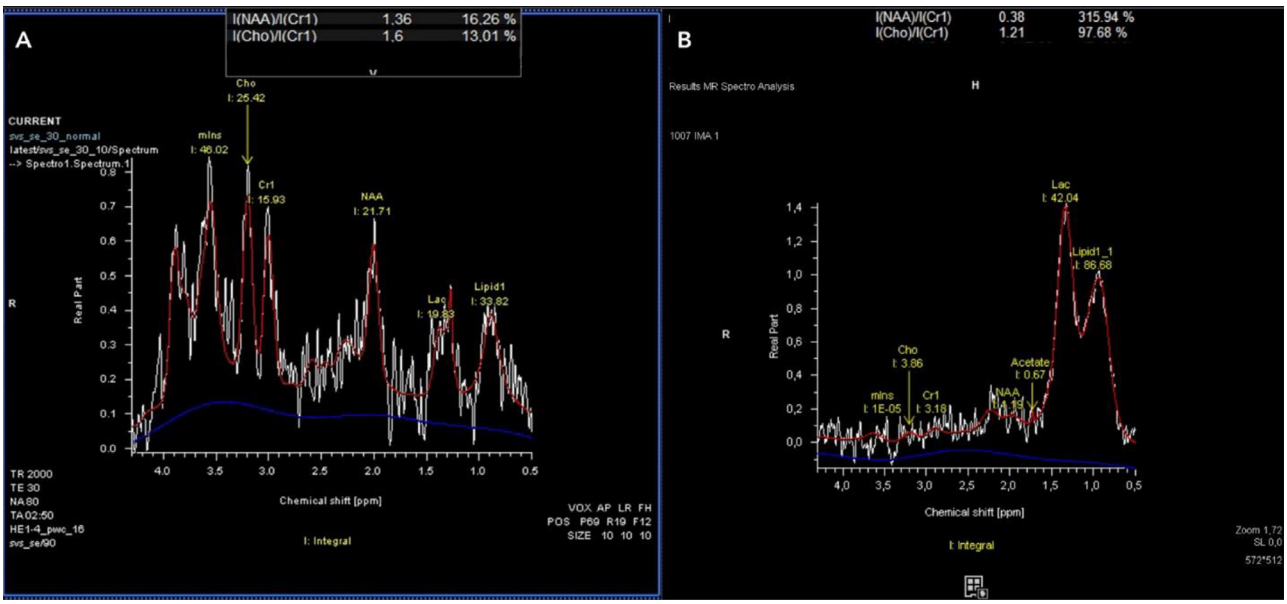

Fig. 5 – MR Spectroscopy shows peaks of metabolites on normal parenchyma (A) compared to intralesion (B). NAA, N-acetylaspartate; Cr1, creatine; Cho, choline. MRS sequence shows decrease in choline (from 25.42 to 3.18), decrease in creatine (from 15.93 to 3.18), decrease in N-acetylaspartate (from 21.71 to 1.19), increase in lactate (from 19.83 to 42.04), and increase in lipid (from 33.82 to 86.68), with decrease in NAA/Cr and Cho/Cr ratio (from 1.36 to 0.38 and from 1.6 to 1.21, respectively).

nature of changes. Reduced level of NAA was found in this case, where reduced level of NAA indicates the absence of neurons and axons. NAA is also reduced in most gliomas, meningiomas, and other brain lesions. Absence of lactate is normal in the brain except in small mounts found in the ventricles. Lactate usually accumulates in cysts, necrotic tissues and in other pathologic processes that undergo anaerobic metabolism, which was increased in this case. The presence of lipid, which is raised in cases of tuberculoma but decreased in this case, is a signal for distinguishing tuberculoma from neurocysticercosis, according to Ravishankar et al. On the other hand, presence of reduced Cr at location of 3.0 ppm reflects the absence of brain tissue within the lesion as Cr is a very accurate indicator of healthy brain metabolism [12]. The present case shows low NAA/Cr and Cho/Cr ratio (0.38 and 1,21, respectively) and these ratios are fairly correlating with a study done by Singh et al., showing ratios were less than 1.5 in cases of neurocysticercosis. On the other hand, presence of increased Cho/Cr ratio was consistently present in tuberculosis cases but not in neurocysticercosis [6,13]. With these results, this case can be diagnosed as neurocysticercosis.

Conclusion

Neurocysticercosis is variable depending on location, stage of cysts, and host immune response. Diagnosis is mainly made by neuroimaging, but it is difficult to diagnose when a patient has intracranial calcifications. We found 2 stages in this case which are granular nodular and nodular calcified stage. MR Spectroscopy can detect the presence and/or ratio of various tissue metabolites, which can be used to diagnose neurocysticercosis. We recommend that MR spectroscopy should be a part of the routine MR examination for cases that are suspected of tuberculoma and neurocysticercosis.

Patient consent

The patient has seen a version of the manuscript to be submitted/published and he gave his consent for his image or other information relating to him to be reported in the above named manuscript for consideration of publication in the *Radiology Case Reports (RCR)*.

The patient understands that protected health information such as identification number, billing information, address, will not be published and that efforts will be made to conceal his identity. However, diagnostic or medical imaging, may be published.

The patient understands that the material may be published in the Radiology Case Reports (RCR) Journal. As a result, he understands that the material may be seen by the general public. He understands that he may revoke consent at any time before publication, but once the information has been published revocation of the consent is no longer possible.

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