CASE REPORTS

Finding the Hidden-cerebellopontine Angle Neurocysticercosis

KK Pandita[1], Sushil Razdan[1]

Abstract

Neurocysticercosis (NCC) is the most common parasitic disease of the central nervous system worldwide. The lesions of NCC are considered to be readily visualized by MRI or CT scan. We present a patient, of new daily persistent headache (NDPH) with normal initial CT and MRI scan of head, who later was found to have cerebellopontine angle region NCC.

Introduction

Despite impressive gains in diagnosis and availability of antiparasitic treatments, large gaps in both basic and practical knowledge about the Cysticercus and host responses to it exist. Factors hindering further advances are the lack of clinically appropriate model infections, and inability to maintain the life cycle experimentally. Therefore, reliance on naturally infected humans is, at present, the most appropriate study of NCC.[1] We report a patient, who initially presented with new daily persistent headache of undetermined origin, and later turned out to have neurocysticercosis.

Case Report

A 30-year-old truck driver presented to us on 24th December, 2015 with complaint of headache, for the past about one month. Initially, the headache, which was frontotemporal in location, would occur daily at any time of the day and was moderate to severe in intensity, and would last few hours. For the previous 15 days he described his headache being located at frontal and occipital regions and would occur at waking hours. The headache had no relation to posture change, coughing and straining. There was no history of trauma or fall. He declined any history of substance abuse except drinking of about 120mL of alcohol every day for the past many years. Non-contrast CT scan of his head (Figure 1) done ten days prior to presentation was unremarkable. He had no history of fever, anorexia, vomiting, vision disturbance, nasal, respiratory or any other systemic symptom. There was no history of seizures. On examination, he was in good general health and he did not seem to be in distress. He was afebrile with a blood pressure of 110/70 mmHg. His general and systemic examination was normal. His neck was supple. Examination of the optic fundi didn't reveal any abnormality. His complete blood count, erythrocytic sedimentation rate, routine blood biochemistry was within normal limits. On his insistence non-contrast MRI of brain (Figure 2) was done on 25th December, 2015 that was unremarkable. He declined consent for lumbar puncture. In view of absence of red flag symptoms and signs, we prescribed oral prochlorperazine and propranolol along with analgesics and asked him to be under close follow up. At a follow up visit on 4th January, 2016, he reported complete resolution of his headache with no new symptoms. Three days later, that is on 7th January, 2016, he reported new symptoms of sudden onset double vision and right ear fullness. His otoscopic and other ear related clinical examination was within normal limits. Neurological examination was remarkable for

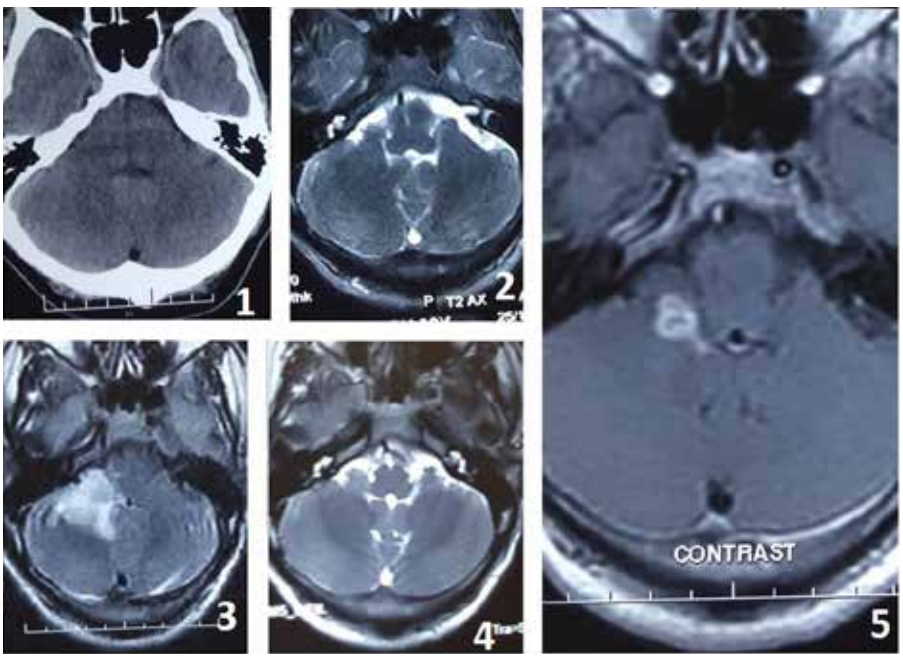

Fig. 1: Serial axial images of brain, at the level of lower brainstem, of a patient with neurocysticercosis. (1) CT scan done on 14th December, 2015 showing no abnormality; (2) MRI scan done on 25th December, 2015 showing no abnormality; (3) T2 weighted MRI scan done on 7th January,2016 showing a hyperintense lesion with perilesional oedema; (4) T2 weighted MRI done on 28th March, 2016 showing clearing of lesion; (5) MRI showing a Coalescing ring enhanced lesion in the right cerebello-pontine angle

[1]Visiting Consultant, Shri Mata Vaishno Devi Superspeciality Hospital, Kakryal, Jammu
Received: 03.06.2016; Revised: 23.01.2017; Accepted: 25.10.2017

Table 1: Time course of clinical events vis a vis imaging findings in a patient of Neurocysticercosis in the region of right cerebello-pontine angle

14th December, 2015	25th December, 2015	7th January, 2016	28th March, 2016
CT scan of brain: No abnormality detected (Figure 1)	MRI scan of brain: No abnormality detected (Figure 2)	MRI scan of brain: Coalescing ring enhancing lesions in right cerebello-pontine angle region (Figures 3 and 5)	MRI scan of brain: resolution of right cerebello-pontine angle region lesion (Figure 4)
Last week of November, 2015 to last week of December, 2015		First, second and third week of January, 2016	March, 2016.
New daily persistent headache (NDPH)		Diplopia, right ear fullness, and nystagmus	Asymptomatic

nystagmus in right horizontal gaze. Rest of his general and systemic examination was unremarkable. His complete blood count erythrocytic sedimentation rate, routine blood biochemistry, X-ray chest, tests for antinuclear antibody and human immunodeficiency virus were within normal limits. Fresh Contrast enhanced MRI scan of his brain revealed two coalescing ring enhancing lesions in right cerebellar pontine angle region. (Figures 3 and 5). We made presumptive diagnosis of neurocysticercosis (NCC) and administered oral deflazacort and albendazole for a period of three weeks. At a follow up visit on 23rd March, 2016, he reported total resolution of his symptoms. We repeated his MRI brain (Figure 4) which revealed that the lesion had disappeared completely.

Discussion

Review of the published literature reveals three cases of cerebellopontine angle region NCC, all having been managed with surgery.[2-4]

Differential diagnoses of new daily persistent headache (NDPH) include migrainous-type headache, tension-type headache, subarachnoid hemorrhage, low cerebrospinal fluid (CSF) volume headache, raised CSF pressure headache, posttraumatic headache and chronic meningitis.[5] Our patient's clinical profile did not suggest any of these headaches, although tension-type headache could not be ruled out. Thinking retrospectively, it is obvious that NDPH, which our patient presented with initially, must have been because of the neurocysticercosis (NCC).

After ingestion of embryonated eggs of adult tapeworm from the contaminated environment, the hatched oncospheres are carried by the blood stream to various organs and lodged in the small blood vessels where they may or may not develop into viable cysts. Viable cysts form after 2-3 months. Cysticerci in the brain parenchyma initially suppress the host inflammatory response. After an incubation period, estimated to be several years, the cysticerci lose the ability to suppress the host inflammatory response, leading to parenchymal inflammation. The cysticerci induce a granulomatous response, which gradually degrades the parasites. In some cases the lesions resolve. However, in others, degradation leads to formation of calcified granulomas, which become intermittently inflamed.[1,6] The lesions of NCC are considered to be readily visualised by MRI or CT scans.[7] In addition, the process of cyst degeneration is best depicted by MRI examinations. Viable cysts appear clear to gross examination (same density as CSF), hypointense on T1 and FLAIR examinations. The first indication of degeneration is the presence of enhancement, which indicates dysfunction of the blood brain barrier, most likely due to pericyst inflammation around a still viable cyst. When the cyst becomes grossly opaque, the MRI appearance changes from no signal to a bright signal on T1 and FLAIR sequences accompanied by dense enhancement.[1,6] In the case of our patient CT and MRI scan has not been able to detect the viable cyst stage and early granulomatous stage of cysticercus (Table 1). Despite their high sensitivity, conventional MR sequences can fail to detect the cysticercus cysts within the cerebrospinal fluid spaces. However, obtaining three-dimensional spoiled gradient recalled echo imaging sequences can help in improving the detection of intraventricular NCC.[7] At the initial presentation these special sequences were not done as there was no suspicion of neurocysticercosis, clinically.

Cysticercus can produce headache by two mechanisms. One, by obstructing the flow of cerebrospinal fluid and producing hydrocephalus. Two, when pain producing structures are irritated by the inflammation[5] that occurs when viable cyst transforms into granulomatous stage. Imaging studies did not reveal any hydrocephalus, so it must have been the early inflammation of transition phase between viable cyst and granulomatous stage irritating some pain producing structure that was responsible for the initial NDPH. So in regions of world where cysticercosis is endemic, patients presenting with NDPH of undetermined origin may be imaged by special MR sequences,[7] like three dimensional constructive interference in steady state(3D-CISS), or followed up closely and reimaged for seeking the hidden cysticercus in the brain.

References

1. Nash TE, Garcia HH. Diagnosis and Treatment of Neurocysticercosis. *Nat Rev Neurol* 7:584–594.
2. Singh S, Gibikote SV, Bannur U, Chacko G, Korah IP, Rajshekhar V. Cysticercosis of the cerebellopontine angle cistern mimicking epidermoid inclusion cyst. *Acta Neurol Scand* 1999; 99:260-3.
3. Celis MA, Mourier KL, Polivka M, Boissonnet H, Kato T, Lot G, George B, Cophignon J. Cisternal cysticercosis of the cerebellopontine angle. A surgically-treated case and review of the literature. *Neurochirurgie* 1992; 38:108-12.
4. Revuelta R, Soto-Hernández JL, Vales LO, González RH. Cerebellopontine angle cysticercus and concurrent vascular compression in a case of trigeminal neuralgia. *Clin Neurol Neurosurg* 2003; 106:19-22.
5. Goadsby PJ, Raskin NH. Headache. Pp. 107-111. In: Harrison's principles of internal medicine. Volume 2, 19th edition. Edited by Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J. New York: Mc Graw Hill, 2015.
6. White AC, Jr., Brunetti E. Cestodes. Pp. 2052-7. In: Goldman's cecil medicine. 24th edition. Edited by Goldman L, Schafer AI. Philadelphia: Elsevier, 2012.
7. Robbani I, Razdan S, Pandita KK. Diagnosis of intraventricular cysticercosis by magnetic resonance imaging: Improved detection with three-dimensional spoiled gradient recalled echo sequences. *Australasian Radiology* 2004; 48:237–239.