Commentary

I read with a great interest of the report of the 3rd cranial palsy secondary to neurocysticercosis in the current issue of the *Journal of Neurosciences in Rural Practice*.[1] The pork tapeworm *Taenia solium* causes two types of human disease, namely taeniasis and cysticercosis.[2] It is important to differentiate the two infections because a frequent misconception is that cysticercosis is acquired by eating pork. It is taeniasis, a localized intestinal tapeworm infestation, which is secondary to human ingestion of pork meat containing larval cysts of *T. solium*. On the other hand, cysticercosis is a systemic invasive tissue infection of the larval stage (cysticercus) of the *T. solium*; it is transmitted through ingestion of food contaminated with *T. solium* eggs (not the larval cysts), usually via fecal-oral transmission from a tapeworm carrier (often an asymptomatic household member who handles food). Neurocysticercosis is a cysticercotic infection of the central nervous system and is the most severe form of cysticercosis.

Neurocysticercosis represents the most common cause of acquired epilepsy in the developing countries,[2,3] and it is estimated that there are more than 50,000 deaths per year from neurocysticercosis.[2] However, little attention has been paid to this disease entity until the World Health Organization (WHO) listed neurocysticercosis as one of the neglected tropical diseases and set targets for eradicating it by 2020.[4] Neurocysticercosis is highly prevalent in endemic regions of *T. solium*, such as Latin American countries, sub-Saharan Africa, Indian subcontinent, Southeast Asia, Korea, and China.[3] Even in the countries where swine breeding and pork meat consumption are prohibited, there have been increasingly reported cases of neurocysticercosis in local people who acquired from ingestion of contaminated foods prepared by domestic workers and housekeepers migrated from *T. solium* endemic countries.[5] Long-term (>1 month) international travelers to *T. solium* disease-endemic areas are considered to be a high likelihood of contracting neurocysticercosis from a local taenia carrier.[6] It has no longer been regarded as a rare disease in developed countries, but practicing physicians in North America and Europe have not been yet familiar with the disease.[7,8]

Clinical manifestations of neurocysticercosis depend on the number, location, stages of cysticercal cysts, and the degree of the host immune reaction.[2] Virtually, any parts of the central nervous system could be affected. Hence, a wide spectrum of neurologic manifestations secondary to neurocysticercosis has been described in the literature. Seizures, nausea, vomiting, headache, and intracranial hypertension are the most common clinical symptoms and signs.[2] Other reported clinical presentations of neurocysticercosis are facial myokymia, various forms of nystagmus, extrapyramidal signs, Kluver-Bucy syndrome, and Weber's syndrome.[9,10] Ocular neurocysticercosis is an uncommon disease and usually present with a myriad of striking neuro-ophthalmic manifestations.[11-14] Making a diagnosis of neurocysticercosis in patients presenting with ophthalmic signs is exceptionally challenging and daunting in non-endemic regions because it is still a forgotten disease in developed countries. In addition, the complexity of cranial nerve pathways and functions further poses a diagnostic challenge for the first responders who are usually non-neurologists or non-neuroophthalmologists. An early diagnosis requires a high index of clinical suspicion, and neurocysticercosis should be considered in differential diagnosis in an appropriate clinical context. Inquiry of of a detailed social history of demographic or epidemiological risk factors and a comprehensive travel history plays a significant role in supporting the initial suspicion. It would then be confirmed by neuroimaging studies and/or serologic assays to meet the diagnostic criteria of neurocysticercosis.

Neurocysticercosis should no longer be considered as a disease only of developing countries given advances and ease of intercontinental travel. In any parts of world including industrialized countries, there have been increasing numbers of reported cases of neurocysticercosis in returned travelers, immigrants from endemic regions, or local people living with domestic helpers from disease-endemic countries. Practicing clinicians require a heightened clinic index of suspicion of neurocysticercosis that could present with various neuro-ophthalmic manifestations. Also, interpreting radiologists should be familiar with and be able to recognize characteristic radiological appearance of neurocysticercosis to assist in diagnosis. Interdisciplinary and multidisciplinary consultation and collaboration plays a pivotal role in early recognition and diagnosis of the disease.

Zaw Min

Division of Infectious Diseases, Allegheny General Hospital, Allegheny Health Network, Pittsburgh, Philadelphia, USA

Address for correspondence:
Asst Prof. Zaw Min,
Division of Infectious Diseases,
Temple University School of Medicine,
Core Faculty, Allegheny General Hospital,
Allegheny Health Network, 420 East North Avenue,
East Wing, Suite 407, Pittsburgh, PA 15212, USA.
E-mail: zmin@wpahs.org

References

1. Chauhan S, Damani G, Gupta RK, Chaudhary KP. Isolated nuclear III nerve paresis secondary to neurocysticercosis showing spontaneous clinical improvement. J Neurosci Rural Pract 2014;5:S100-2.
2. King CH, Fairley JK. Tapesworms (Cestodes). In: Bennett J, Dolin R, Blaser MJ, editors. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 8th ed. Philadelphia, PA: Saunders; 2015. p. 3231-3.
3. Coyle CM, Mahanty S, Zunt JR, Wallin MT, Cantey PT, White AC Jr, *et al*. Neurocysticercosis: Neglected but not forgotten. PLoS Negl Trop Dis 2012;6:e1500.
4. Savioli LS, Daumerie D. Sustaining the Drive to Overcome the Global Impact of Neglected Tropical Diseases: Second WHO Report on Neglected Diseases. Geneva: World Health Organization; 2013. p. 1-153.
5. Del Brutto OH. Neurocysticercosis on the Arabian Peninsula, 2003-2011. Emerg Infect Dis 2013;19:172-4.
6. Del Brutto OH. Neurocysticercosis among international travelers to disease-endemic areas. J Travel Med 2012;19:112-7.
7. Venna N, Coyle CM, González RG, Hedley-Whyte ET. Case records of the Massachusetts General Hospital. Case 15-2012. A 48-year-old woman with diplopia, headaches, and papilledema. N Engl J Med 2012;366:1924-34.
8. Naddaf E, Seeger SK, Stafstrom CE. Neurocysticercosis in Wisconsin: 3 cases and a review of the literature. WMJ 2014;113:74-9.
9. Bhatia R, Desai S, Garg A, Padma MV, Prasad K, Tripathi M. Isolated facial myokymia as a presenting feature of pontine neurocysticercosis. Mov Disord 2008;23:135-7.
10. Patel R, Jha S, Yadav RK. Pleomorphism of the clinical manifestations of neurocysticercosis. Trans R Soc Trop Med Hyg 2006;100:134-41.
11. Keane JR. Cysticercosis: Unusual neuro-ophthalmologic signs. J Clin Neuroophthalmol 1993;13:194-9.
12. Ranjith MP, Divya R, Sahni A. Isolated one and a half syndrome: An atypical presentation of neurocysticercosis. Indian J Med Sci 2009;63:119-20.
13. Naphade PU, Singh MK, Garg RK, Rai D. Bilateral ptosis: An atypical presentation of neurocysticercosis. BMJ Case Rep 2012;2012. pii: bcr2012006195.
14. Khurana N, Sharma P, Shukla R, Singh D, Vidhate M, Naphade PU. Midbrain neurocysticercosis presenting as isolated pupil sparing third cranial nerve palsy. J Neurol Sci 2012;312:36-8.

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