Labyrinthitis ossificans after meningitis: Superiority of high-resolution magnetic resonance imaging in demonstration of disease extent compared to high-resolution computed tomography

Sir,

Labyrinthitis ossificans after meningitis is a cause of sensorineural hearing loss in children. We discuss a case of an adolescent male with sensorineural hearing loss and magnetic resonance imaging (MRI) demonstrated the extent of the labyrinthitis ossificans much better than high-resolution computed tomography (HRCT). A 14-year-male presented with a history of profound bilateral sensorineural deafness. He had pyogenic meningitis 6 months earlier with Streptococcus pneumoniae as causative agent. MRI done at that time revealed leptomeningeal enhancement in basal cisterns and cerebellopontine angle region. After discharge, he started experiencing reduced hearing in both ears (left > right) which gradually progressed over a period of 2–3 months into profound deafness. His pure tone audiometry and auditory brainstem response were suggestive of bilateral profound sensorineural hearing loss. MRI [Figure 1] and HRCT temporal bones [Figures 2 and 3] of the patient revealed a diagnosis of bilateral labyrinthitis ossificans.

Bacterial meningitis is the most common cause of acquired sensorineural hearing loss in children seen in up to 5–7% of cases. Window of the cochlea, window of vestibule, or both are shown to be route for infection dissemination to the inner ear. In case of bacterial meningitis, ossification is more extensive and route of entry to the inner ear is through subarachnoid space, cochlear aqueduct, and through inner acoustic meatus.

Irrespective of the etiology, labyrinthitis ossificans pathogenesis involves an acute early stage where there is the presence of bacteria and leukocytes forming an inflammatory response in the perilymphatic spaces.
which is followed by proliferation of fibroblasts and finally, formation of osteoid matrix.

HRCT is traditionally the imaging study of choice for preoperative assessment of cochlear implant. Drawbacks of HRCT include the inability to detect early fibrosis, partial volume averaging, and suboptimal assessment of the cochlear region compartments. MRI allows earlier diagnosis of labyrinthine ossificans as a result of its ability to demonstrate low T2 signal in the fibrous phase of labyrinthitis ossificans.

In our patient, there was a loss of normal T2 hyperintensity seen in the right vestibule and semicircular canals which, however, did not show any ossification on computed tomography likely due to fibrotic phase of labyrinthitis ossificans. On the right side, there was ossification in the cochlea that also showed loss of normal signal on the T2-weighted image. There was a loss of normal signal intensity involving the superior and lateral canals appearing normal on HRCT which again likely represent fibrotic area. Thus, high-resolution T2 three-dimensional sequences provide higher sensitivity to detect the extent of disease as in our case.

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Conflicts of interest
There are no conflicts of interest.

Sameer Vyas, Vikas Bhatia, N. K. Panda, Paramjeet Singh, Niranjan Khandelwal

Departments of Radiodiagnosis and Imaging and Otolaryngology, Post Graduate Institute of Medical Education and Research, Chandigarh, India

Address for correspondence:
Dr. Sameer Vyas,
Department of Radiodiagnosis and Imaging, Post Graduate Institute of Medical Education and Research, Chandigarh, India.
E-mail: sameer574@yahoo.co.in

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Estimation of cerebrospinal fluid cortisol level in tuberculous meningitis

Sir,

Mahale et al. addressed in their interesting study that the mean cerebrospinal fluid (CSF) cortisol level in tuberculous meningitis (TBM) patients was significantly higher as compared to aseptic meningitis patients and control subjects (P<0.0001).

Accordingly, the authors suggested that CSF cortisol level estimation could be considered as a rapid, relatively inexpensive diagnostic marker in the early identification of TBM along with CSF findings of elevated proteins, hypoglycorrhachia, and lymphocytic pleocytosis.

I presume that the clinical implication of that suggestion is questionable. This is based on the following two points.

First, the cut-off values of CSF cortisol were not established to be practically implementable in the Indian clinical setting.

Second, the use of biological markers, including adenosine deaminase (ADA) has been suggested to enhance the accuracy of the initial diagnosis of various infections, including meningitis. As a better alternative to CSF cortisol, I presume that CSF-ADA measurement could be considered as a simple, useful, and rapid diagnostic tool for the early recognition of TBM and evaluating anti-TB therapy in TBM patients in India. This is based on the following three points. (1) The accuracy of CSF-AD has been recently studied in Indian TBM and non-TBM patients. The results indicated that CSF-ADA of 10 U/L as a cut-off value had 87.5% sensitivity and 83.3% specificity whereas the positive predictive value of the test was 87.5% and 83.3% negative predictive value. The study concluded that CSF-ADA estimation is not only simple, inexpensive, and rapid but also a fairly specific method for making a diagnosis of TBM, especially when there is a dilemma of differentiating tuberculous etiology from non-tuberculous ones.

(2) Comparing ADA levels and polymerase chain reaction (PCR) in CSF has revealed that CSF-ADA is a more sensitive indicator than PCR for the diagnosis of TBM in an Indian cohort with suspected TBM. Using a cut-off level of >10 U/L, CSF-ADA had the sensitivity of 92.5% and specificity of 97% for the diagnosis of TBM whereas PCR for TBM had a sensitivity of 44.5% and specificity 92% in the most likely TBM cases.

(3) Most recently, it has been found that even in low TB endemic areas, CSF-ADA measurement can be still used to early diagnose TBM. The best ADA cut-off in low TB endemic areas has been estimated to be 11.5 IU/L with 91% sensitivity and 77.7% specificity.

If CSF-ADA (>11.5 IU/L) estimation is combined with CSF glucose level (<65 mg/dL) and leukocytes (≥13.5 cell/mm³), the sensitivity and specificity will skip to 91% and 88%, respectively.

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Mahmood Dhahir Al-Mendalawi
Department of Paediatrics, Al-Kindy College of Medicine, Baghdad University, Baghdad, Iraq