Extensive cerebellar necrosis

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DESCRIPTION

A developmentally normal, previously well 1-year-old girl presented with altered sensorium, 4 days after the onset of an episode of acute gastroenteritis. She had features of raised intracranial pressure and was in hypotensive shock at the time of admission. Investigations revealed mild thrombocytopenia; however, cerebrospinal fluid analysis, blood cultures and serologies for infections were unyielding. She required a ventriculoperitoneal shunt, following which gradual improvement in sensorium ensued. However, motor deficits persisted and the child was discharged after 3 weeks on a tracheostomy. Neuroimaging at the time of discharge (figure 1) revealed bilaterally symmetrical cerebellar involvement with no abnormal foci of contrast enhancement. At 1-year follow-up, MRI of the brain (figure 2), showed persistence of non-progressive, non-enhancing isolated cerebellar necrosis with the child showing mild improvement of residual motor deficits.

Diffuse cerebellar necrosis has been previously documented in a few case reports pertaining to conditions like postradiation necrosis,¹ posthypoxia² and systemic sclerosis,³ and is usually associated with brainstem involvement. Preferential necrosis of cerebellar tonsils due to coning is also documented in patients who underwent multiple lumbar punctures.⁴ Unlike the well-studied postinfective striatal necrosis

Figure 2 One-year follow-up MRI brain—axial T2 weighted (A), FLAIR (B), T1 weighted (C), susceptibility weighted (D), apparent diffusion coefficient (E) and contrast-enhanced T1 weighted (F) images showing, persisting non-progressive bilaterally symmetrical cerebellar T2 and FLAIR hyperintensities without restriction of diffusion and absence of contrast enhancement or susceptibility changes.

in children,⁵ the isolated extensive bilateral cerebellar necrosis of infective aetiology is hitherto unreported. Although, no precise neuroinfection could be established histopathologically or serologically in our case; clinico-radiological correlation points to the acute cerebellitis as a primary working diagnosis. Lack of clinical markers and presence of persistent bilaterally symmetrical cerebellar involvement rules out focal causes like dysplastic cerebellar gangliocytoma, crossed cerebellar diaschisis and cerebellar tumours. Relative sparing of cerebellar vermis and peduncles as well as brainstem may explain the relatively preserved clinical status in our case. Further follow-up of the patient with neurological examination and neuraxial imaging may open newer insights about cerebellar response to inflammation and natural history of its liquefactive necrosis.

Figure 1 Initial brain imaging (postventriculoperitoneal shunt): Non-contrast CT (A) showing bilaterally symmetrical cerebellar hypodensities, mild diffuse ventriculomegaly with pneumoventricle; MRI—axial T2 weighted (B), FLAIR (C), susceptibility weighted (D), diffusion (at $b=1000 \text{ s/mm}^2$) weighted (E) and apparent diffusion coefficient (F), showing bilaterally symmetrical non-restricting T2 hyperintensities with no significant susceptibility changes. Artefacts in relation to left cerebellar hemisphere (D–F) are due to ventriculoperitoneal shunt tube.

Patient's perspective

I was so scared when my baby acutely deteriorated last year after the bouts of loose motions. Although the doctors saved her life with the tube in her neck and other life-support systems, no exact cause was revealed in any of the lab tests. Thankfully, my baby has been significantly improving. I am told the abnormal patches in her hindbrain seen on MRI have also not worsened, but need to be followed up. I hope for her full recovery and near-normal development.

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Learning points

- Isolated non-progressive bilaterally symmetrical postinfective cerebellar necrosis in a child is an extremely rare clinical entity which needs a detailed workup to rule out probable causative factors.
- The topographical and chronological distribution of the involvement in the absence of abnormal contrast enhancement provide important imaging clues to the differential diagnoses.
- Clinikoradiological follow-up in such cases may provide newer insights into the management of inciting neurological insult.

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