The prenatal hindbrain herniation through the foramen magnum is a consequence of a tethered spinal cord and prolonged prenatal CSF leakage. This transforaminal herniation may result in 'degeneration' of cerebellar tissue, presumably due to distortion, marked compression of cerebellar blood vessels and mechanically induced ischaemia.

Boltshauser et al reported this entity in three children with asymmetric involvement of the cerebellar hemispheres and sparing of the vermis. This phenomenon was called 'vanishing cerebellum in Chiari II malformation'.

We demonstrate the natural history of this cerebellar disruption and its prenatal onset in two cases in which there were signs of echogenic detritus in the posterior fossa and within the lateral ventricles that disappear progressively.

Case 1: Detected at 25+5w. Fig. 1a: shows echogenic material filling partially the posterior fossa, and replacing the cerebellum. Fig.1b-c: Disappearance of the detritus and complete absence of cerebellum at 30w. GA (T2WI)

Case 2. Figs. 2a-b: Neurosonogram at 30w depicting echogenic detritus in posterior fossa, upper cervical channel and in the lateral ventricles. Figs 2c-d: Two weeks later fetal MRI (T2WI) shows absence of most of the cerebellum. In FFLAIR sequence hypointense intraventricular sediment is seen in posterior lateral ventricle.

Both cases presented compromise of the vermis and small narrowed brainstem without pontine prominence. As well as in cerebral agenesis these findings may have a severe impact in neurocognitive development.

**Conclusion**

The cases shows the natural history of vanishing cerebellum that complicate the evolution of myelomeningocele. Neurosonography and fetal MRI play an important role in the diagnosis of these adverse events, which alters the prognosis of Chiari II malformation in the fetus.