Comment to the paper: double neural tube defects: a case report and controversies around neural tube closure by Vashu Ravindran

Mehmet Selcuki

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Comments for publication

This paper is a case report of a midline fusion defect patient with parietal encephalocele and a thoracic myelomeningocele. Presented case is, indeed, a rare combination of neural tube closure defects. However, the combination of midline fusion defects does not necessarily point the existence of multiclosure sites as the authors wanted to endorse. It is well known that the defects of the neural tissue development can be multiple on the same patient, which is very interesting but not a surprise at all.

Neurulation in cranial and spinal regions are quite different processes as far as the molecular genetics and movements of the neural folds are concerned. While the neural folds at the second closure site at mouse embryo are convex at the beginning of the neurulation, they become concave because of the dorsolateral bending points and neurulate, finally. However, along the spinal level, the case is not closed neural plate and not closed bones, muscles, and surface ectoderm as well. In case of ancephalocele, surrounding skin and inner layers are relatively intact. Although the multisite closure theory was used for the explanation of the various localizations of encephaloceles, actin cytoskeleton deficiency, failure of emigration of neural crest, timing defect of apoptosis, and failure of cooperation between cell proliferation and

the onset of the neuronal differentiation may be the culprit of the subsequent nonclosure of cranial neural tube as well.

Previously accepted zipper theory of neural tube closure has been abandoned for a while ago (as the authors indicate in the paper), and multisite closure commencement has been discovered as a result of various investigations. Experiments on chick and mouse embryo models revealed that without having the cranial part of the embryo, cultured caudal piece could successfully neurulate. During closure of the neural plate, completed closure at the previous level is not a begin-to-close signal for the subsequent level. Actually, it is a matter of time. During the neurulation, two temporally different unexpected events may cause developmental defects at two different related levels while other levels continue to neurulate normally.

In conclusion, this case is a rare and an interesting case having two midline closure defects at different levels. Closure defects at different levels can be explained by temporally differing accidents while the cranial part of the neural tube begins to neurulate earlier than spinal part does. Regarding this particular case, it sounds more fruitful to discuss on various probable insults that may act on cranial and spinal parts of the neural tissue rather than coming to a conclusion based on multiclosure sites.

M. Selcuki (✉)
Department of Neurosurgery, School of Medicine,
University Hospital, Celal Bayar University,
45000 Manisa, Turkey
e-mail: mselcuki@yahoo.com