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In moderate communicating hydrocephalus of human fetuses, ependymal denudation is a common feature that may result in abnormal neurogenesis

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Background

Recent investigations carried out in natural and experimental mutant mice have provided strong evidence that a primary alteration of the ependymal cell lineage triggers a moderate foetal hydrocephalus [1,2]. In human cases of hydrocephalus, however, ependymal loss has been regarded as resulting from the ventricular dilatation due to the accumulation of cerebrospinal fluid [3].

Materials and methods

The present investigation was carried out in 16–40 week old human foetuses with a communicating hydrocephalus and displaying a moderate dilatation of the ventricular cavities (n = 8), and foetuses of similar ages with no neuropathological alterations (n = 15). Paraffin sections throughout the walls of the cerebral aqueduct and lateral ventricles were processed for lectin binding and immunocytochemistry using ependyma, astroglia, neuroblasts and macrophague markers.

Results

Large areas of ependymal denudation were found in the aqueduct and lateral ventricles of all foetuses developing a communicating hydrocephalus. At variance, no ependymal detachment was observed in non-hydrocephalic foetuses. In the youngest foetuses with hydrocephalus, denuded areas were not covered by astrocytes or other organized cell elements, leaving the neuropile directly exposed to the ventricular lumen. The area devoid of ependyma increased as the foetus developed. In the oldest foetuses studied, the denuded areas of the lateral ventricles were lined by a dense plexus of astrocytes. Under the denuded surface the presence of ependymal rosettes was observed. In the denuded areas of the lateral ventricles of hydrocephalic foetuses it was found (i) a loss of the germinal ependymal zone, (ii) disorganization of the subventricular zone and, (iii) abnormal migration of neuroblasts into the ventricular cavity.

Conclusion

The early loss of ependyma in human hydrocephalic foetuses would be associated to both, the hydrocephalic process and an abnormal migration of neuroblasts.

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