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AGENESIS OF CORPUS CALLOSUM: A CASE REPORT

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Abtsract

Patient with complete callosal agenesis, male, aged 7, displayed cerebral disconnection symptom as deficit in verbal identification of objects, palpated by the left hand. Compensatory mechanisms do not substitute efficiently for callosal pathway in connecting lefthemispheric language centers with somato-sensory regions of the right hemisphere. It is not clear, whether insomnia in this patient could be ascribed to the absence of callosal system, however, clinical, EEG and MRI examination did not reveal other visible reasons for sleep-wakefulness disorders.

Key words: corpus callosum, agenesis, hemispheres, callosal system, insomnia

Introduction

Agenesis of corpus callosum by its origin is related to the disorders of the formation of structures, deriving from the anterior telencephalic wall.

During the 5th week of embryo development the telencephalic vesicles (future hemispheres) arise as lateral evaginations of the prosencephalic cavity. Hemispheres are formed as a result of the backward growth of the lateral vesicles by the 7th – 8th weeks. Forebrain commissures and related structures originate from *lamina terminalis*, which develops from anterior telencephalic wall (originally arising from the wall of prosencephalic cavity) and extends from the optic chiasm up to the velum transversum. Its dorsal part is known as the *lamina reuniens* of Hiss.

The dorsal part of the *lamina reuniens* forms so-called *sulcus medianus telencephali medii* by infolding between the growing vesicles. *Massa commissuralis* – the fused tissue of the *sulcus medianus*, gives rise to corpus callosum, hippocampal commissure and fornix as well as to the hippocampus and archicortex. Callosal fibres appear at about the third month of embryo development and attain final configuration two months later. Ventral part of *lamina reuniens*, adjacent to *lamina terminalis* gives rise to anterior commissure, septal nucleus and rhinencephalon [5, 15, 21].

Thus, it is obvious that early injury to the anterior telencephalic wall may lead to commissural malformation, as well as to underdevelopment of olfactory structures and to holoprosencephaly. The localization and extent of injury determines which structures will be involved in the pathology. Agenesis of corpus callosum, anterior commissure and the hippocampal commissure may stem from the defect in the entire *lamina reuniens*, whereas in the case of a more dorsal injury, anterior commissure could be spared. Focal damage to the posterior midline at a later stage of morphogenesis may cause partial callosal agenesis, while in cases of more ventral injury corpus callosum is spared [5, 17, 24]. Corpus callosum is always absent in holo-

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