Abstract

Some clinical features of rabies and experimental evidence from cell culture and laboratory animals suggest impairment of gabaergic neurotransmission. Several types of gabaergic neurons occur in the cerebral cortex. They can be identified by three neuronal markers: the calcium binding proteins (CaBPs) parvalbumin (PV), calbindin (CB) and calretinin (CR). Rabies virus spreads throughout the cerebral cortex; however, rabies cytopathic effects on gabaergic neurons are unknown. The expression of calcium-binding proteins (CaBPs) parvalbumin (PV), calbindin (CB) and calretinin (CR) was studied in the frontal cortex of mice. The effect of gabaergic neurons was evaluated immunohistochemically. The distribution patterns of CaBPs in normal mice and in mice infected with fixed or street rabies virus were compared. PV was found in multipolar neurons located in all cortical layers except layer I, and in pericellular clusters of terminal knobs surrounding the soma of pyramidal neurons. CB-immunoreactivity was distributed in two cortical bands. One was composed of round neurons enclosed by a heavily labeled neuropil; this band corresponds to supragranular layers II and III. The other was a weakly stained band of neuropil which contained scattered multipolar CB-ir neurons; this corresponds to infragranular layers V and VI. The CR-ir neurons were bipolar fusiform cells located in all layers of cortex, but concentrated in layers II and III. A feature common to samples infected with both types of viruses was a more intense immunoreactivity to PV in contrast to normal samples. The infection with street virus did not cause additional changes in the expression of CaBPs. However, the infection with fixed virus produced a remarkable reduction of CB-immunoreactivity demonstrated by the loss of CB-ir neurons and low neuropil stain in the frontal cortex. In addition, the size of CR-ir neurons in the cingulate cortex was decreased.

Keywords

Rabies, parvalbumin, calbindin, calretinin, GABA