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Vitamin B6 prevents cognitive impairment in experimental pneumococcal meningitis.

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Abstract

Streptococcus pneumoniae is the relevant cause of bacterial meningitis, with a high-mortality rate and long-term neurological sequelae, affecting up to 50% of survivors. Pneumococcal compounds are pro-inflammatory mediators that induce an innate immune response and tryptophan degradation through the kynurenine pathway. Vitamin B6 acts as a cofactor at the active sites of enzymes that catalyze a great number of reactions involved in the metabolism of tryptophan, preventing the accumulation of neurotoxic intermediates. In the present study, we evaluated the effects of vitamin B6 on memory and on brain-derived neurotrophic factor (BDNF) expression in the brain of adult Wistar rats subjected to pneumococcal meningitis. The animals received either 10 µL of artificial cerebral spinal fluid (CSF) or an equivalent volume of S. pneumoniae suspension. The animals were divided into four groups: control, control treated with vitamin B6, meningitis, and meningitis treated with vitamin B6. Ten days after induction, the animals were subjected to behavioral tests: open-field task and step-down inhibitory avoidance task. In the open-field task, there was a significant reduction in both crossing and rearing in the control group, control/B6 group, and meningitis/B6 group compared with the training session, demonstrating habituation memory. However, the meningitis group showed no difference in motor and exploratory activity between training and test sessions, demonstrating memory impairment. In the step-down inhibitory avoidance task, there was a difference between training and test sessions in the control group, control/B6 group, and meningitis/B6 group, demonstrating aversive memory. In the meningitis group, there was no difference between training and test sessions, demonstrating impairment of aversive memory. In the hippocampus, BDNF expression decreased in the meningitis group when compared to the control group; however, adjuvant treatment with vitamin B6 increased BDNF expression in the meningitis group. Thus, vitamin B6 attenuated the memory impairment in animals subjected to pneumococcal meningitis.

KEYWORDS: BDNF; Pneumococcal meningitis; memory; vitamin B6

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