アルツハイマー病動物モデルを用いた痴呆の定量化と抗痴呆療法の研究

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Research Abstract

A unilateral lesion was made in the nucleus basalis magnocellularis (NBM) of the Alzheimer rat model and after one week autotransplantation of the vagal nodosal ganglion (X) was carried out. Four weeks later serial measurements of Acetylcholine (Ach) markers (choline acetyltransferase, CAT ; acetylcholineesterase, AChE ; ^3H-QNB binding activity to muscarine receptor, mAChR) and latency of the P300 (event-related potentials) were carried out for the evaluation of its therapeutic effects. Histological studies by the AChE staining (Tago's method) showed the NBM lesions made by microinjection of the ibotenic acid. Accepted and proliferated viable X tissue was documented in the brain even 4 weeks after X autoplantation. The latency of P300 was 356(〕SY.+-.〔)21.3 msec in normal rats. It was continuously delayd over 400 msec until 4 weeks in the NBM-lesioned rats. Whereas it was found less than 351.0-360.0 msec after 2 weeks in the autotransplanted rats ; indicating significant improvement. The changes of ACh markers was as follows : 1) One weeks after making lesion, CAT activity was reduced to approximately 65% in the cerebral cortex (CC) and 75% in the subcortical structure (SS) on the lesion side compared to that on the contralateral side. From 2 weeks, CAT activity was increased in CC but not in SS.After 3 weeks, AChE activity was reduced to the lowest level and on the contrary mAChR was increased to reach its peak. 2) CAT activity was increased in the CC and SS from 2 weeks after autotransplantation. AChE activity was also increased in SS from 2 weeks and in CC from 3 weeks. However, mAChR was reduced continuously from 2 weeks in the whole brain. From results mentioned above, the conclusions is as follows : 1) In response to the rapidly decreased ACh production caused by

cholinergic deprivation, a kind of compensatory mechanism, such as the increased receptor-binding activity combined with the reduced AChE activity, might take place. 2) Autotransplantation of