

EDITORIAL

Poststroke Amnesia

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Studies investigating the time course of poststroke memory function are scarce specially because the stroke does normally not affect structures involved in memory [1]. However, amnesia has been repeatedly reported in patients following surgical repair of anterior communicating artery (ACoA) aneurysms [2].

Cerebral aneurysm is a relatively common disease with a prevalence of 5% in Western populations and whose incidence of rupture is estimated at 10-15 per 10,000 inhabitants per year [3]. The survival to surgical repair of cerebral aneurysms has, fortunately, increased considerably nowadays. However, it is common to find some type of behavioural and cognitive impairments because of these accidents.

Most aneurysms, approximately 90%, occur in the anterior part of the cerebral arterial supply, the junctions of the arteries in the circle of Willis are the site where these aneurysms are most likely to develop [4]. Anterior communicating artery (ACoA), located at the ventral portion of circle of Willis, connects the two anterior cerebral arteries to the optic chiasm and It seems to be very susceptible to neurobehavioral impairments and particularly to memory problems [5-7].

The ACoA has important arterial branches that perfuse basal forebrain sites, its aneurysm frequently resulted in a dense amnesia often described as “Korsakoff-like” which is known as the “ACoA syndrome” [8]. This syndrome is a neurobehavioural disorder comprising a severe multimodal anterograde and retrograde amnesia, often together with an impairment of frontal-executive functions and personality changes [9-11].

Despite the absence of diencephalic and mesial temporal lesions the literature tends to support the hypothesis that the amnesia observed in ACoA patients was a result of the basal forebrain lesions [12-14]. Damage in the basal forebrain area profoundly reduces the content of acetylcholine and choline acetyltransferase in the neocortex [15]. This may be the cause of the amnesic disorder since significant areas for memory function, as the hippocampal formation, are deprived from cholinergic innervation [16,17].

Memory dysfunction in ACoA patients may improve with cholinergic substitution therapy. To date we have only found one pilot study testing if ACoA patients with persistent memory impairment show a reduction of amnesic syndrome when given donepezil, a cholinesterase inhibitor.

Benke, *et al.* carried out an exploratory study in 11 patients with a memory impairment of more than a year duration because of a ruptured and repaired aneurysm, they have no other neurological or psychiatric disease [18]. At baseline, patients showed impairments of learning, recall and long-term retention of a shopping list. Recognition memory was preserved. The applied treatment was the following: 4 weeks of 5 mg donepezil daily, 8 weeks of 10 mg donepezil and 4 weeks after drug discontinuation.

The central finding of this research is that the patient group, improved their memory performance to a larger degree over time in comparison with matched neurologically normal controls. It is interesting to note that the improvement observed in memory tests scores was continued with an acute decrease in performance after drug discontinuation. Furthermore, donepezil had a greater influence on improving long-term memory functions.

This study confirms the hypothesis that loss of cholinergic innervation appears to be one of the most important causes of the memory impairment found in the ACoA syndrome and that cholinesterase inhibitors can be a good option for pharmacological treatment. Nevertheless, more research is necessary in this regard since we only have one single investigation, which has some limitations, such as a too small patient group, lacking placebo-control, blinding and randomization.

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