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A role for statin therapy in Alzheimer's disease

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Background

Disruption to VLDL metabolism and removal of beta amyloid protein from neurons seems to play a pivotal role in the development of late- onset disease Alzheimer's disease (AD). Surveys suggest the apolipoprotein E4 allele is present in between 45-60% of late-onset AD and, together with the *SORL-1* gene, involved in the regulation of amyloid precursor protein (APP), probably accounts for a majority of late onset AD. Although the link of ApoE4 allele to AD is well documented, as yet there is no clinical consensus on whether genotyping has any value. Only ~ 30% of individuals with the Apo E4 allele will develop AD as co-related environmental factors, such as hypertension, cigarette smoking and dyslipidemia, are important contributors to outcome, in addition to epigenetic and epistaxis mechanisms yet to determined. This might suggest that individuals carrying the allele and identified to be 'at risk' might benefit from early intervention programs. Two studies published on the effect of statin therapy in AD prevention have shown no effect, though these studies might have been underpowered [1,2].

Case reports

Individuals with the ApoE4 allele consistently demonstrate thickened cIMT (Carotid Intima Media Thickness) readings in the absence of co-morbidity factors and clinical disease. We report on observational data in 10 patients who possess the ApoE4 allele demonstrating improved cIMT measurement when treated with statin therapy and lifestyle modification, despite otherwise normal lipid levels and co-morbidity factors. cIMT is a surrogate marker for atherosclerotic load and predictor for future cardiovascular events. Improvements in cIMT imply a reduction in future atherosclerotic events.

Conclusion

Observational data in a small number of patients suggests a marked benefit from statin therapy in individuals possessing the ApoE4 allele. ApoE4 is a risk factor for cardiovascular disease (CVD) as well as AD and statin therapy might have a role early in the pathogenesis of cardiovascular disease even where lipid levels are normal or low. Furthermore, as atherosclerosis is a co-morbidity factor in AD, it also implies that early statin therapy might delay the onset of AD.

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References

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