CEREBROVASCULAR SYMPTOMS AND MRI FINDINGS ASSOCIATED WITH ELEVATED LIPOPROTEIN (A) LEVELS – SINGLE-INSTITUTE PRELIMINARY EXPERIENCE

K. Sas[1], E. Pósfai[2], I. Marton[2], R. Ónody[3], K. Jost[3], A. Csomor[4], L. Vécsei[1,5]
[1]*Department of Neurology, Medical Faculty, Albert Szent-Györgyi Clinical Center, University of Szeged, Hungary*
[2] *2nd Department of Medicine and Cardiology Center, Medical Faculty, Albert Szent-Györgyi Clinical Center, University of Szeged, Hungary*
[3]*Department of Laboratory Medicine, Medical Faculty, Albert Szent-Györgyi Clinical Center, University of Szeged, Hungary*
[4]*Department of Radiology, Medical Faculty, Albert Szent-Györgyi Clinical Center, University of Szeged, Hungary*
[5]*Neuroscience Research Group of the Hungarian Academy of Sciences and the University of Szeged, Hungary*
sask@freemail.hu

Background: Elevated serum lipoprotein (a) [Lp(a)] levels have been demonstrated to correlate with cardiovascular disease. Our aim was to assess the characteristics of cerebrovascular alterations in patients with elevated Lp(a) levels.
Methods: This study involved a retrospective analysis of the data on 25 consecutive patients with enhanced serum Lp(a) levels who presented with neurological symptoms at our stroke unit between 2010 and 2014.
Measurement of Lp(a): Lp(a) was determined by agarose gel electrophoresis. Serum Lp(a) levels above a cutoff value of 30 mg/dl were regarded as positive, and the patients were recruited into the study.
Results: To summarize the MRI findings, our results indicated that the most frequent cerebrovascular manifestations in patients with an elevated Lp(a) level were atherosclerosis, chronic periventricular white matter ischemic lesions, posterior type ischemic lesions and cerebral sinus thrombosis. Infarcts in the basal ganglia and the truncus cerebri were not characteristic. Coexisting conventional risk factors or thrombophilias which could modify the clinical or morphological picture were frequent.
Conclusions: In the event of the occurence of neurological signs or symptoms, cerebral venous thrombosis or premature atherosclerosis where the cause is not apparent, measurement of the serum level of Lp(a) is advisable. We suggest that such patients should be tested first by means of of a simple, semiquantitative method; then, in the case of positivity (a Lp(a) level above the cutoff of 30 mg/dl), precise determination of the serum concentration would then be worthwhile in order to to optimize their therapeutic management.