Anticardiolipin Antibody and Stroke: Possible Relation of Valvular Heart Disease and Embolic Events

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Reading with great interest the editorial on 'Anticardiolipin Antibody and Stroke: Possible Relation of Valvular Heart Disease and Embolic Events' by Barbut et al. in Cardiology 1991;79:99-109 I wish to make some comments:

In the 'Method' section no other cardiovascular risk factors were reported. Furthermore, anticardiolipin antibodies (acL, correct abbreviation since the Workshop in Sirmioni, Italy) may also be observed in infectious diseases, usually as a transient phenomenon, although in patients with HIV or spirochetal infections acL may be persistent. Moreover, acL may be elevated (false positive) in patients with certain medications (pheno-thiazines, hydralazine, procainamide, dilantin). No reason was given why the lupus anticoagulant test was not performed, or is the reason that the patients were on anticoagulant therapy?

The level of antibody that is labeled as positive is also of major importance: Harris [1] uses 5 GPL-U/ml as the cut-off between negative and 'low positive'. 'Medium positive' is identified as between 5

and 100 GPL-U/ml, whereas 100 GPL-U/ml and above are identified as 'high positive'. The degree of positivity needs to be reported, because low positives are also frequently the result of laboratory errors. Unfortunately, no IgM-acL was determined.

When discussing the findings, I could not find an explanation how the authors come to the conclusion that all cerebral events were embolic, when they reported that all their patients had carotid stenoses. I certainly accept the results that IgG-acL is elevated in patients with cerebrovascular artery disease, which our group could also confirm in subjects even without autoimmune diseases [2]. Finally, the statement that acL represents a risk factor is not supported by the data of this study which is based only on a small number of patients without any time correlation of the event or control acL levels in a follow-up. Thus, the authors failed to answer the topical question whether these antibodies are the cause or only an epiphenomenon of thrombotic events in patients with or without autoimmune disorders [3].

References

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