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Migrainous Stroke and Antiphospholipid Antibodies: Are They Pathogenetically Linked?

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Gupta’s letter contains hard criticisms of our paper which reports an association between antiphospholipid antibodies (aPLs) and stroke in some patients [1]. According to the author of the letter, the major problem seems to be the creation of scientific myths from the description of an association. This, in turn, would be responsible for increasing ignorance about scientific problems not yet fully understood. I am quite surprised by this kind of criticism. In fact, in my opinion, ignorance can never arise from the description of a phenomenon or an association among phenomena, as in the case of our paper. More often it can be sustained by an excess of trust in one’s own opinion. I know for certain that even the truth or falsity of the statement ‘Smoking caused X’s lung cancer’ cannot be established, but it is also true that many discoveries have been made by chance. This has occurred even following the observation of an association between two or more phenomena. This kind of process probably is responsible for the concept that smoking can cause lung cancer. This does not mean that I am able to say with absolute certainty that Mr X’s cancer is related to his smoking habit, but it does not prevent me from affirming that smoking can cause lung cancer and that stopping smoking is useful for everyone because it probably reduces the possibility of developing lung cancer. I have read and re-read our article in an attempt to find statements strong enough to generate false myths, but I have not been able to find any. We affirm, or it might be better to say that we only suggest that our observations should encourage further study of the possibility that in a subgroup of patients with migrainous stroke, the association with aPLs could be relevant. This is not the same as stating that migraine, aPLs and stroke are pathogenetically linked. On the other hand, the growing number of reports of patients with stroke, migraine and aPLs has also raised the hypothesis that a number of migrainous strokes can be explained by the presence of antiphospholipid antibodies, a condition predisposing to coagulopathy [2].

The major objection of Gupta to the hypothesis that the presence of aPLs could predispose to ischemic stroke seems to be open to criticism. It is true in fact that thrombotic events have been observed in patients with aPLs during anticoagulation or antiplatelet agents [2], but stroke recurrence can also occur in patients without aPLs during prophylactic therapy. In fact, prophylaxis is expected to reduce but not eliminate the risk of an ischemic event. It would be wonderful if all patients with embolic cardiopathy could be prevented from having a stroke by simply prescribing an anticoagulant for them! In any case, other studies suggest the usefulness of antiplatelet or anticoagulant therapy in patients with aPLs for preventing ischemic events [3, 4]. For this reason I do not find the statement ‘such therapy in the aPLs syndrome is illogical’ reasonable. Presumably, a definitive judgment on the effect of antiplatelet and anticoagulant therapy will not be available before the completion of the only ongoing prospective, randomized trial comparing aspirin with warfarin for the prevention of stroke [5]. Moreover, it does not seem logical to criticize a pathogenetic hypothesis for a subgroup of migrainous strokes by upholding other possibilities (dehydration, platelet activation, etc.) that are not supported by stronger evidence.

In conclusion, I do not think that describing an association among different phenomena can be considered a danger for scientific progress. On the contrary, in some ways it may be a step toward knowledge. This is especially true when it is specified that the described data do not prove anything but only suggest the usefulness of further investigation. The confutation of these data should be based on research proving opposite results and not on theoretical assumptions. In other words, we must ask some questions: Does an association exist between aPLs and stroke? Can migraine be considered an early marker of this association? I suppose that the right answer at the moment is: we simply do not know. To solve the problem we need to be familiar with all the available data, without any prejudice. We reported data showing that in a population of 16 patients with migrainous stroke, aPLs were present in 37.5% of the cases. And that is all.

References

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