Letters to the Editor

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Previous Cardiac Abnormalities in Subarachnoid Hemorrhage May Also Have Background Genetic Polymorphisms

To the Editor:

The recent and very intriguing article on adrenoceptor polymorphisms (AR) and the increased risk of heart abnormalities after subarachnoid hemorrhage (SAH) by Zaroff and colleagues1 as published in the July issue of Stroke merits congratulations. They found that AR genotypes, in particular β1AR 389 CC, β2AR 27 CC or α2AR, increased the odds of the release of cardiac troponin I and/or reduction of left ventricular ejection fraction in a genetic study with 182 patients. This approach is to be very highly appreciated; however, some points need further consideration. The main warranty is why the authors had excluded the patients with previous cardiac abnormalities (eg, with prior myocardial infarction or congestive heart failure), thus possibly having introduced selection/exposure bias. At the same time, however, about 42% of their patients had history of hypertension and up to 10% history of coronary artery disease (see Table 2, lines 16 to 24). This point needs clarification and it would be worth further discussing the role of previously existing (pre-SAH) heart and hemodynamics pathologies as possibly interfering with such post-SAH cardiac sequelae within the revealed genetic background (in particular, a range of possibly relevant single-nucleotide polymorphisms) of post-SAH complications. For instance, early abnormal ECG changes (eg, even within 48 hours from SAH initiation2) are of relevance for management and prediction of eventual complications, including post-SAH cardiac pathologies of neurogenic origin. Such ECG changes at admission might be possible signs of previous, even not diagnosed earlier (ie, without prior tracing in the natural history) “silent” hypertension and/or heart disease in SAH patients; notably, repolarization, ischemic-like ECG findings and/or QT-interval prolongation had been seen in >70% of SAH patients, most often representing pre-existing ischemic heart disease.3 At the same time, β2AR polymorphisms/single-nucleotide polymorphisms are well known to be related to hemodynamic and cardiac pathologies (eg, hypertension, congestive heart failure, etc).4 Being of increasing concern, we have recently emphasized the issue of abnormal ECG findings as eventual indices of pre-SAH cardiac pathology, in the view of neurological deficit.5 Because SAH happens predominantly during the early adulthood and occurs suddenly and more than one-third of the patients may die within 2 weeks of admission, whether such patients may have had and/or have been considered to have any previous heart disease (PHD) is of extreme importance. We hypothesized that abnormal changes of ECG during the acute phase of SAH may be also an indication for unknown previous “silent” heart pathology. For instance, hypertension is a risk factor for SAH and, at the same time, especially when lasting for many years, may be associated with both ECG changes and/or pre-SAH cardiac pathology. Our data revealed that most frequent were repolarization abnormalities; ECG changes in patients without PHD were comparable to that in patients with PHD. The repolarization was more frequent in SAH patients with less severe neurological deficit (Hunt & Hess scale) whereas the rhythm and conductive abnormalities were more frequent in patients with more severe neurological deficit.6 Moreover, in their multivariable models on the relationships of the specific adrenoceptor polymorphisms with post-SAH cardiac outcomes,1 the authors had considered age, gender and race/ethnicity as possible covariates but neither history of hypertension nor history of coronary artery disease or neurological deficit were addressed as potential confounders and/or effect modifiers. Although presented with small prevalence (Table 2), no roles for risk factors of coronary artery disease such as diabetes or hyperlipidemia were mentioned, either. Though the latter interactions might have been analyzed, however, no description of such results could be found in the published article.1 Notably, such potential pre-SAH effects should be seen as even more important in the light of the recently presented evidence on the links of SAH with such novel vascular- or neurologically related polymorphisms as in UCP3, TNF, and PKD1-like genotypes (eg, Gly243Asp, etc) on one side, and of such common AR polymorphisms as α2Delt322–325 and α2Gln27 allelic states with vasospastic angina, on the other.6,7 The above points surely need further consideration/discussion, especially in the view of potential cardioprotective benefits of adrenergic blockade in SAH patients as suggested by the authors.1

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Disclosures

None.

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