Gene Expression Microarray Studies of Intracranial Aneurysm Walls Lead to Similar Results

To the Editor:

We congratulate the authors Shi et al for their recent study of comparing 3 ruptured and 3 unruptured aneurysm walls with paired tissue of the superficial temporal artery.1 Back in May 2008 we published our gene expression microarray study results of 4 unruptured and 6 ruptured aneurysms, as well as 4 arteriovenous malformation feeders which were obtained in a similar way.2 We used the same pathway analysis program “Ingenuity Pathway Analysis”; the microarray platform used was an Agilent Human Oligo Microarray which carries 18 716 transcripts of human gene expressions. Interestingly, we obtained similar results: our top-scoring networks displayed high-level functions in immune response, cell to cell signaling and interaction, and immune and lymphatic system development and function. Shi et al report to have found 6 functional annotation groups to have the most relevance in terms of aneurysm development and rupture: collagens, cell communication, angiogenesis, inflammation, apoptosis and cytoskeleton negative regulation.1 In a previous serial analysis of gene expression comparing tissue of a single intracranial aneurysm compared with a matched superficial temporal artery, strong immune and inflammatory response was also detected.3

A drawback of our study and rightful criticism of the reviewers at the time of submission was the use of arteriovenous malformation feeder tissue as a control. It is not certain whether the feeder tissue is completely “normal” tissue or if there are gene expression changes as compared to other cerebral vessels. An alternative would have been to use postmortem tissue of intracranial vessels, but that may be subject to excessive RNA degradation. As it is difficult to obtain normal intracranial vessel wall tissue (matched or from a different person) the question of which tissue to compare the aneurysm wall tissue to remains a challenge.

The use of matched superficial temporal artery tissue could also lead to distorted results because its histological structure is different from intracranial vessels: intracranial vessels often lack the external elastic lamina and have a less prominent adventitial layer as compared to extracranial vessels.

Hopefully, larger studies including more samples and controls and increasingly sophisticated computer programs will be able to extract yet further pathways and discover the interactions during intracranial aneurysm wall formation.

Disclosures

None.

Boris Krischek, MD
Department of Neurosurgery
University of Tübingen
Tübingen, Germany

Hidetoshi Kasuya, MD
Division of Neurosurgery
Medical Center East
Tokyo Women’s Medical University
Tokyo, Japan

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Boris Krischek and Hidetoshi Kasuya

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