Correspondence

Letter by Hiemann et al Regarding Article, “Assessment of Microcirculatory Remodeling With Intracoronary Flow Velocity and Pressure Measurements: Validation With Endomyocardial Sampling in Cardiac Allografts”

To the Editor:

We read with interest the results reported by Escaned et al concerning vascular remodeling in endomyocardial biopsies and microvascular function in cardiac transplant recipients. Since we published our experience with microvasculopathy in 2007, we have appreciated the growing interest in that phenotype of vascular pathology after heart transplantation. However, to avoid the vague terminology concerning morphological and functional microvascular findings that has unfortunately developed for epicardial vasculopathy during the past 40 years, exact definitions should be used when assessing microvasculature. CD34, like CD31, is not a marker for capillaries but for endothelial cells in general. Thus, a pan-endothelial marker reflects total vascularization rather than capillary vascularization, which has to be calculated as the difference between CD34-positive and muscularized microvessels. The latter are easily identified with immunohistochemical techniques to detect α-actin, because “arterioles” have a diameter of less than 20 μm, whereas vessels sized <100 μm and >20 μm do not represent the terminal endings of the coronary artery tree (“microvessels”). We showed previously that capillary rarefaction is specific for transplant vasculopathy and that microvascular remodeling, including increased arteriolar vascularization and progressive capillary loss, undergoes a critical phase within the first 3 months after heart transplantation. Therefore, a mean time since cardiac transplantation of almost 2 years, ranging from 1 to 82 months, for sampling might have influenced the results. Further biases might be acute cellular rejection, which we showed to induce on-off phenomena in CD31-positive blood vessels, and the nontransplanted controls being more than 10 years older than the study population. In contrast to our findings, which are cited, hypertension was a risk factor for microvasculopathy only in monovariate regression analysis, and immunosuppression was not tested because the regimen was similar in 70% of patients. Despite the present methodological limitations, we appreciate the approach of hypothesis generation, which stimulates further study in the important field of microvasculopathy after heart transplantation.

Disclosures

None.

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References

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