Dilated cardiomyopathy and coronary flow reserve

We read with interest the article by Rigo et al.1 about the prognostic impact of coronary flow reserve (CFR) by Doppler echocardiography in dilated cardiomyopathy. The authors conclude that in patients with idiopathic dilated cardiomyopathy, the prognostic role of impaired microvascular CFR has been shown to be unfavourable. In our opinion, some points of this work are not completely clear.

First of all, it is not correct to diagnose an idiopathic dilated cardiomyopathy only upon exclusion of ischaemic heart disease after coronary angiography. Authors excluded patients with mycarditis. How can they exclude myocarditis without performing endomyocardial biopsies? Myocarditis is only diagnosed by established histopathological, histochemical, or molecular criteria on endomyocardial biopsy.2 Clinical suspicion may be raised by global left ventricular dysfunction, acute congestive heart failure, or cardiogenic shock associated with left ventricular dilatation and/or segmental wall motion abnormalities. When myocarditis is suspected clinically, an endomyocardial biopsy may resolve an otherwise ambiguous situation by virtue of diagnostic inflammatory infiltrate and necrosis (i.e. the Dallas criteria). The diagnostic yield of myocardial biopsies is enhanced substantially by molecular analysis with DNA-RNA extraction and polymerase chain reaction amplification of the viral genome. Moreover, it is recognized that patients with biopsy-proven inflammatory infiltrates have a diminished CFR due to reduced coronary vasodilator capacity.3 Experimental data also showed that CFR measured by transcranial Doppler echocardiography is reduced in coxsackievirus myocarditis in mice.4 Low CFR is associated with progressive heart failure indicating that dysfunction of coronary circulation is a determinant of poor outcome in viral myocarditis.5 Therefore, we think that endomyocardial biopsy should be performed in order to exclude myocarditis. The event-free survival in the group of patients with a CFR >2 is nearly 100% in the first 3 years; this could be due to high incidence of myocarditis in this patients group and higher proportion of subjects with a spontaneous recovery. It would have been very interesting to have a second measurement of CFR in the follow-up period. Did the authors see a CFR improvement in this patients group? Secondly, the CFR cutoffpoint of >2 is arbitrary; in fact, it is takes on loan from ischaemic heart disease, in which it has the best accuracy as a predictor of significant LAD stenosis.6 To the best of our knowledge, there are no data dealing with the optimal CFR cutoff in identifying worse prognosis in patients with idiopathic dilated cardiomyopathy. To test the predictive discrimination of patients with and without events, ROC curve analysis should have been generated. Moreover, a high proportion of patients suffered from hypertension and diabetes, two conditions that must be excluded to diagnose an idiopathic dilated cardiomyopathy2 and that are known to influence CFR.6,7

Finally, the CFR impairment in dilated cardiomyopathy and heart failure could be due to many haemodynamic features;8 it is highly speculative to highlight a microvascular origin for CFR impairment. A multivariate analysis for CFR determinants in this patient should be performed, including also histopathological evidence of microvasculature damage.

In conclusion, we think that the article by Rigo et al. is very interesting for its prognostic impact, but contains some limitations that do not allow to provide comprehensive evidence for CFR as non-invasive prognostic tool in dilated cardiomyopathy. Further longitudinal studies, in larger patient cohorts with better characterized idiopathic dilated cardiomyopathy, are warranted.

References