Impairment of coronary microvascular flow in hypertrophic cardiomyopathy

We read with great interest the reports by Kuhn et al.\(^1\),\(^2\), which describe patients with an obstructive form of hypertrophic cardiomyopathy (HCM) developing abrupt no-flow phenomenon resembling no-reflow phenomenon reported in some coronary patients after percutaneous coronary intervention\(^3\),\(^4\).

HCM patients have been shown to have abnormal small coronary resistance vessels\(^4\),\(^5\),\(^6\),\(^7\). Accordingly, intramural coronary arteries and subendocardial arterioles have thicknessed walls and narrowed lumens\(^4\),\(^5\),\(^6\),\(^7\). The intima layer involving endothelium is hypertrophied\(^8\),\(^9\) and endothelial cells are structurally abnormal\(^8\),\(^9\), which provides a morphological substrate for functional impairment of the endothelium. Accordingly, endothelial-dependent vasodilator dysfunction has been demonstrated using the cold pressor test both in symptomatic\(^10\) and asymptomatic\(^11\) HCM patients without left ventricular outflow tract gradient. Even in asymptomatic untreated HCM patients, the cold pressor test may induce paradoxical microvascular constriction manifested by decreased coronary blood flow velocity and increased coronary vascular resistance during this stress\(^11\). The degree of vasoconstriction was mild but all patients were without significant symptoms and without obstruction. In the report of Kuhn et al.\(^1\) the severe microvascular constriction manifested by abrupt no-flow after the injection of contrast agent was observed in selected HCM patients (i.e. symptomatic and with obstruction). It is possible that increased left ventricular systolic pressure (as an extravascular force) compressing microcirculation in the subendocardial layer may intensify microvascular spasm. This hypothesis may be supported by an observation\(^1\) that emergency transcoronary ablation of septal hypertrophy decreases the left ventricular outflow tract gradient and eliminates microvascular spasm.

During the cold pressor test, released catecholamines contribute (via alpha-adrenergic stimulation of vascular smooth muscles) to the vasoconstrictor response in patients with endothelial dysfunction\(^1\),\(^2\). Importantly, the vasoconstrictor effect of catecholamine is dose-dependent in the coronary arteries with loss of endothelium function\(^8\),\(^9\). This catecholamine-induced, dose-related vasoconstriction may be in line with the observation of Kuhn and co-authors\(^1\) that patients with an anxious, stressed personality are predisposed to an abrupt no-flow phenomenon during a stressing procedure such as heart catheterization. The catecholamine release is probably smaller under the cold pressor test than during more stressing heart catheterization.

Kuhn and co-workers\(^1\) studied invasively an impressively large group of obstructive and non-obstructive HCM patients. It is interesting how frequently the slow-flow phenomenon (milder pathology than abrupt no-flow phenomenon) occurred in their group as a manifestation of increased microvascular resistance without occlusive spasm.

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References