Commentary

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Cardiac allograft vasculopathy Physiology Assessment in Early after Heart Transplantation

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Cardiac allograft vasculopathy (CAV) is the leading cause of late morbidity and mortality after heart transplantation. Cardiac allograft vasculopathy is a panarterial disease with a progressive and diffuse process involving both the epicardial coronary artery and the microcirculation. Approximately 10% of patients have angiographic coronary artery disease at 1 year, 50% at 5 years, and 80% at 15 years, with long-term mortality increasing with angiographic severity. Cardiac allograft vasculopathy can also manifest as a microvasculopathy, which occurs more frequently than epicardial coronary artery stenosis at 1 year after transplantation and is associated with a higher risk of cardiac events, independent of epicardial coronary artery stenosis [1].

Clinical guidelines recommend annual or biannual coronary angiography to assess the development of CAV. Intravascular ultrasound (IVUS) is often used to more accurately detect progression of CAV that is not readily apparent with coronary angiography. However, anatomical evaluation is limited to assessing the physiological consequences of epicardial coronary artery disease and is not able to assess microvascular dysfunction. In addition, the presence of epicardial CAV does not necessarily indicate that microvascular dysfunction is present and vice versa.

Assessing coronary physiology using a pressure-temperature sensor-tipped guidewire has been well validated in non-transplant patients. The comprehensive physiological assessment of the epicardial coronary artery and microcirculation has helped to characterize the physiological phenotype of patients and to better predict their prognosis. Similarly, in transplant patients, fractional flow reserve (FFR) correlates with plaque volume assessed by IVUS, and the index of microcirculatory resistance (IMR) measured after transplantation has been shown to predict the development of CAV, poor graft function, and long-term mortality in single-centre studies. The prognostic value of invasively assessing coronary physiology early after heart transplantation has not been adequately validated in a large multicentre study [2].

This international multicentre registry enrolled heart transplant recipients who underwent a comprehensive intracoronary physiology assessment at baseline and 1 year after transplantation. We then characterized the coronary physiological abnormality into abnormal epicardial coronary physiology and/or microvascular dysfunction and evaluated their long-term prognostic value.

Patients were pooled from five prospective cohorts [three prospective randomized trials and two prospective observational studies conducted in four countries (USA, Norway, Sweden, and Korea)]. The study design, detailed entry criteria of each study, and the key features are summarized. For this analysis, only patients evaluated by comprehensive coronary physiological assessment including FFR, IMR, and coronary flow reserve (CFR) at baseline and/or at 1 year after transplantation were included [3].

All patients received standard immunosuppressive therapy according to the clinical protocol of each participating centre. Briefly, patients received induction therapy with antithymocyte globulin, daclizumab, or basiliximab. Maintenance immunosuppression was based on calcineurin inhibitors (cyclosporin or tacrolimus), antimetabolites (azathioprine or mycophenolate mofetil), and prednisone, which was tapered during the first year at some centres. Calcineurin inhibitors were partially or completely replaced with mammalian target of rapamycin inhibitors (everolimus or sirolimus) in selected patients according to the clinical status or protocol. Therapeutic levels of immunosuppressive agents and associated side effects were carefully monitored and titrated accordingly. Concomitant medications including statins and, in some cases, aspirin were initiated as soon as the patient was able to comply with oral intake. As part of standard clinical care, patients were monitored for the occurrence of acute cellular rejection by endomyocardial biopsies performed at the standard interval according to the clinical protocol of each participating centre and at the time of any suspected episode of rejection [4].

After performance of coronary angiography, FFR, IMR, and CFR were measured in the usual fashion with a pressure-temperature sensor-tipped guide wire (Abbott Vascular) placed in the distal two-third of the left anterior descending artery. Fractional flow reserve was defined as the mean distal coronary pressure divided by the mean aortic pressure at maximal hyperaemia. Index of microcirculatory resistance was calculated as the distal coronary pressure at maximal hyperaemia divided by the inverse of hyperaemic mean transit time. Coronary flow reserve was calculated as resting mean transit time divided by hyperaemic mean transit time. Resting and hyperaemic mean transit time was measured using standard thermo dilution techniques [5].

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