Pericardial and myocardial disease

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Diseases of the pericardium are particularly common in patients with infectious, inflammatory or rheumatic diseases, as well as in those with certain forms of cancer, and is a condition that is often not easy to diagnose, as it may mimic an acute coronary syndrome,1,2 or to treat.3–5 In contrast, post-infarction pericarditis has become quite rare with modern urgent primary percutaneous coronary procedures.

Frequently pericardial inflammation or infection also involves the myocardium, causing myocarditis, a common precursor of certain forms of non-ischaemic cardiomyopathies. Indeed, myocarditis may impair left ventricular function6 due to marked inflammation with secretion of cytokines,7 with sometimes a fulminant clinical course.8 Although myocardial biopsies may help in the diagnosis,9 effective treatment strategies are still missing.

In addition, dilated cardiomyopathies may develop due to genetic and unknown factors. Mutations in mitochondrial genes may cause a variety of clinical symptoms in the brain, eye, and peripheral and cardiac muscle.10–12 In the clinical research paper entitled ‘Long-term cardiac prognosis and risk stratification in 260 adults presenting with mitochondrial diseases’ by Karim Wahbi from the Cochin Hospital in Paris, France,13 the authors characterized the long-term cardiac prognosis of adults with mitochondrial diseases. They retrospectively included 260 consecutive patients with genetically proven mitochondrial diseases, including mitochondrial DNA (mtDNA) single large-scale deletions, the m.3243A>G mutation in MT-TL1, other mtDNA point mutations, and 36 with nuclear gene mutations. Cardiac involvement was present at baseline in around one-third of the patients over a follow-up of 7 years. A total of 10% of the patients experienced an adverse cardiac event, defined as sudden death, death due to heart failure, resuscitated cardiac arrest, third degree atrioventricular block, sinus node dysfunction, cardiac transplantation, or hospitalization for heart failure. Patients with single large-scale mtDNA deletions or the m.3243A>G mutations had the highest incidence of adverse cardiac events. Independent predictors of adverse cardiac events were intraventricular conduction block with a hazard ratio of 16.9, diabetes with a hazard ratio of 7.0, premature ventricular complexes with a hazard ratio of 3.6, and left ventricular hypertrophy with a hazard ratio of 2.5. In patients with 0, 1, and ≥2 risk factors, the incidence of adverse cardiac events was 2, 15, and 42%, respectively. Thus, patients with mitochondrial diseases are at high risk of adverse cardiac events. Independent predictors are a intraventricular conduction block, diabetes, ventricular prematurity, and left ventricular hypertrophy. The manuscript is accompanied by an excellent Editorial by Sabine Pankuweit from the Philipps-University Marburg in Germany.14

Cardiomyopathies are an important cause of heart failure,15 with diverse causes, particularly in younger patients.16 Some pre-clinical and a few clinical studies suggest that transplantation of autologous bone marrow mononuclear cells may improve pump function in dilated cardiomyopathies, although clinical studies have been disappointing so far.17 In the second clinical research paper ‘Multi-centre, randomized, double-blind trial of intracoronary autologous mononuclear bone marrow cell injection in non-ischaemic dilated cardiomyopathy (The Dilated Cardiomyopathy Arm of the MiHeart Study)’. Antonio Carlos Campos De Carvalho and colleagues from the Instituto Nacional de Cardiologia in Rio de Janeiro, Brazil report the results of a multi-centre, randomized, double-blind, placebo-controlled trial on the effects of intracoronary injection of autologous bone marrow mononuclear cells on pump function in 160 patients with a left ventricular ejection fraction of <35%.18 One hundred and fifteen patients completed the study. Left ventricular ejection fraction decreased from 24% to 20% in the bone marrow mononuclear cell group and from 24% to 22% in the placebo group. There were no significant differences in changes between cell and placebo groups for left ventricular systolic and diastolic volumes and ejection fraction. The mortality rate was 20% with placebo and 21% with bone marrow mononuclear cells. The authors conclude that intracoronary injection of autologous bone marrow mononuclear cells does not improve left ventricular function in patients with non-ischaemic dilated cardiomyopathy. The manuscript is accompanied by an informative Editorial authored by Roberto Bolli from the University of Louisville in Louisville, Kentucky, USA.19

Viral myocarditis is an important cause of heart failure and sudden cardiac death in young healthy adults. Furthermore, it also is a precursor of dilated cardiomyopathy. In a Basic Science article ‘The microRNA-221/-222 cluster balances the antiviral and inflammatory response in viral myocarditis’20 Ward Heggiesmont and colleagues from the University of Leuven in Belgium explored the role of the miR-221/-222 family that is up-regulated in viral myocarditis. Interestingly, miR-221 and miR-222 levels were elevated during acute viral myocarditis caused by Coxackie virus B3. Both miRs were expressed by different cardiac cells and by infiltrating inflammatory cells, but their up-regulation in myocarditis was mostly found in cardiomyocytes. Systemic inhibition of miR-221/-222 in mice increased cardiac viral load, prolonged the viraemic state, and strongly aggravated cardiac injury and...
References