



Biomarkers: past, present and future

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Coronary artery disease (CAD) continues to be a major cause of morbidity and mortality in both men and women in developed and developing countries. The extent of myocardial damage after an acute myocardial infarction (AMI) determines prognosis. The diagnosis of an AMI is based on a combination of symptoms, electrocardiographic changes, and biomarkers. In this issue of *Heart and Metabolism*, we focus on established and novel biomarkers and their role in diagnosis, risk stratification, and prognosis.

Drs Wierzbicki and Viljoen provide us with a comprehensive clinical overview, pointing out that, in addition to a diagnostic role in AMI, biomarkers are also used to monitor drug treatments and their potential toxicity. We tend to think in terms of C-reactive protein, troponin (T, I, or C), and brain natriuretic peptide (BNP), but a wider panel of markers is available to aid both diagnosis and risk evaluation.

In the Basic Article, Drs Vasile and Jaffe describe in detail the molecular biology of troponin in cardiac disease, and the potential for using troponin fragments to delineate differing pathological processes, leading to distinct diagnostic and therapeutic potential.

The time course of biomarker release is important, and differences that may occur, and their clinical

relevance, are the subject of the Refresher Corner. We know that the release of protein at the time of AMI provides both diagnostic and prognostic information, so it is helpful to have a review of the time courses of individual biomarkers, which in turn provides a framework for evaluating therapeutic intervention (whether mechanical or pharmacological). The Case Report shows how these time courses or increasing trends are a valuable adjunct to management.

In the drug focus article, importantly we are reminded of the role of trimetazidine in reducing left ventricular remodeling and potentially improving prognosis. The significant reductions in troponin T and BNP concentrations after 6 months of treatment (60 mg daily) compared with no trimetazidine treatment are associated with preservation of left ventricular function. This signals the need for a large-scale study to evaluate the symptomatic and prognostic role of trimetazidine in patients with reduced left ventricular function.

In the future, genetic biomarkers may transform our understanding and management of patients with CAD, so the New Therapeutic Approaches article by Professor McManus is both timely and enlightening.