‘Myocardial Depression’ or ‘Septic Cardiomyopathy’?

K. WERDAN, A. OELKE, and U. MÜLLER-WERDAN

Introduction

‘Septic acute myocarditis’ in the pre-antibiotic era was a purulent disease of the heart. Nowadays, non-specific pathomorphological and pathohistological alterations characterize the myocardium of patients whose hearts have failed in septic shock. For decades, septic myocardial depression in animal models was attributed to the release of cardiodepressant factors into the blood stream, while the existence of human septic myocardial depression was only unequivocally proven in the early 1980s by the group of Parrillo [1], who had examined patients in the ICU with nuclear imaging techniques. Since then, experimental and clinical evidence has accumulated arguing for a more complex alteration of the heart in sepsis than exclusive myocardial depression. The concept of a “septic cardiomyopathy” was proposed [2], which emphasizes alterations of cardiac cellular phenotype as a basis of organopathy in response to a variety of agents acting on heart cells, like bacterial toxins and endogenous cytokines, hormones, mediators, and cardiodepressant factors. Not only is impairment of complex intrinsic heart function a consequence, but regulation of cardiac function is also severely disturbed due to excessive autonomic dysfunction [3].

The intention of this chapter is to highlight newer aspects of cardiac involvement in severe sepsis and septic shock and its impaired regulation. Organ-related infectious heart diseases, like viral myocarditis or bacterial endocarditis, are not the focus of this article, rather the uniform reaction of the heart to the generalized inflammatory processes seen in sepsis.

Septic Cardiomyopathy: A Secondary Cardiomyopathy in the Scope of the Systemic Disease, ‘Sepsis’

It had long been denied that cardiac involvement forms part of septic multiple organ dysfunction syndrome, as cardiac output values of septic patients are usually apparently normal or may even be enhanced in comparison to the physiological range (Fig. 1). However, heart failure becomes evident when cardiac output is considered in relation to the systemic vascular resistance (SVR), which is severely lowered due to sepsis-induced vasodilatation (Fig. 2). A healthy heart may compensate for the pathological decrease in afterload down to one-third or one-fourth of the normal value by an up to three- or fourfold increase in cardiac output (Fig. 2), while, very often, the observed values in our septic patients are considerably lower (Fig. 2); the compensatory increase in pump activity is not large enough to stabilize blood pres-
Fig. 1. Case report: Cardiovascular changes in *Pseudomonas* sepsis. This patient suffered from an aspiration pneumonia on day 1. After initial stabilization, cardiovascular deterioration occurred, resulting in septic shock around day 7. Thereafter, the patient had an uneventful recovery. CO: cardiac output.

Fig. 2. Correlation of cardiac output and systemic vascular resistance (SVR) in patients with septic multiple organ dysfunction syndrome. In 31 patients with septic multiple organ dysfunction syndrome, cardiac output was measured repeatedly during the course of the disease and here is plotted against the respective SVR. With decreasing afterload (fall in SVR) cardiac output values increase, with considerable variation for any specific SVR value. The upper line represents the maximal cardiac output values achieved by hearts minimally or unimpaired related to the respective SVR, while the values below indicate reduced cardiac output values in the sense of more or less severe septic cardiomyopathy.

sure. This observation is the consequence of the complex pattern of septic cardiomyopathy and the impaired regulation of heart function (Table 1). Left ventricular stroke work indices were reported to be reduced to a similar degree in patients with various forms of Gram-negative, Gram-positive or fungal sepsis [2, 4], indicating that it is not so much bacterial virulence factors but rather the common mediator network that determines the occurrence and severity of the disease. Although septic cardiomyopathy is potentially completely reversible – described as myocardial hiber-