Tackling Inflammatory Heart Disease: How to Get Dirty Glasses Clean with Dirty Towels

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Inflammatory cardiomyopathy denotes a primary, acquired cardiomyopathy, defined by the presence of a chronic inflammatory process [1, 2]. Currently, inflammatory cardiomyopathy is estimated to account for 10–50% of cases with dilated cardiomyopathy [1–3]. Recent progresses in basic and clinical research support the concept that acute, mostly virus-mediated myocarditis triggers a complex system of interactions of host and infective agent-related factors, promoting heart failure development. In this context, heart-specific autoimmunity might play a particularly important role [3–5].

The term ‘inflammatory cardiomyopathy’ simply denotes the immunohistochemical detection of focal or diffuse mononuclear infiltrates with ≥14 leukocytes/mm² (CD3⁺ T lymphocytes and/or CD68⁺ macrophages) in the myocardium, but largely neglects variations in the histological phenotype [4, 6]. From this point of view, we still lack experience in linking morphological findings to a presumed specific clinical disease course. As an exception, we have to mention giant cell myocarditis, a distinct disease entity with typical histological appearance and unfavorable prognosis [7].

The availability of novel molecular-biological techniques has allowed the identification of genomes of various infective agents supposed to trigger and promote inflammatory heart disease [4]. Whereas historical studies failed to show an effect of immunosuppressive treatments in patients with active myocarditis [8, 9], several, albeit small, studies have suggested a potential effect of immunosuppressive treatments in subgroups of patients with inflammatory cardiomyopathy [10, 11]. In fact, it appears that patients with histological evidence for ongoing inflammation in the absence of residual viral genome in heart biopsy samples or evidence for heart-specific autoimmunity might benefit from immunosuppression. Antiviral treatment, on the other hand, might be beneficial for patients suffering from acute viral myocarditis [12]. Unfortunately, these studies were rather small and do not yet provide sufficient evidence to draw firm conclusions regarding generally accepted recommendations for the management of all patients with inflammatory heart disease. In order to translate recent progress in basic and preliminary clinical research into clinical practice, we therefore urgently need more data on these patients.

The German Transregional Collaborative Research Centre ‘Inflammatory Cardiomyopathy – Molecular Pathogenesis and Therapy’ [13], details of which are published in Cardiology, aims to establish a comprehensive research registry on the diagnostics, therapy and disease outcomes of patients with inflammatory cardiomyopathy. Unfortunately, data collection and interpretation are greatly complicated by the low incidence of diagnosed acute myocarditis, the clinical variability, the lack of sensitivity and specificity of routine cardiac tests and the
changing patterns of pathogenic cardiotropic microorganisms [3, 4, 14, 15]. From this point of view, it is practically impossible to cover all variables which might become important for the understanding and management of inflammatory heart disease. In addition, the successive improvement of diagnostic modalities, i.e. cardiac MRI [16] or molecular biology [4, 14], warrants ongoing and critical re-evaluation of data validity. As such, the currently available methodology will always be unsatisfactory to answer our most recent questions. Thus, we are forced to – as the famous Danish physicist Niels Bohr (1885–1962) put it – get ‘dirty glasses clean with dirty towels and dirty dishwater’. Nevertheless, the described registry already covers a fairly large number of included cases and most of the variables currently considered as important in inflammatory cardiomyopathy. Despite important drawbacks, the registry might therefore represent a first step to the acquisition of relevant clinical, microbial and pathological data on inflammatory cardiomyopathy patients, to the analyses of specific sub-groups as well as to potential therapeutic regimens.

I hope that the publication of this methodology paper prompts other centers in Europe to either join an existing registry or to build up their own collaborative databases – ideally by using the same inclusion criteria and standardized protocols. This will be the only way to advance our knowledge on the disease course, diagnostic strategies and appropriate treatments in such a complex disease entity as inflammatory cardiomyopathy. Getting dirty glasses clean with dirty towels, it works somehow – but a philosopher would not believe it.

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