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Review article

Twenty years of alcohol septal ablation document more than a history of a single interventional procedure

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ABSTRACT

The first alcohol septal ablation (ASA) was performed in 1994. The concept of "therapeutic acute myocardial infarction" caused by application of concentrated alcohol into coronary artery is not quite unique since certain arrhythmias were treated in a similar way in the past; however, ASA has been discussed widely since its introduction and refused for many years by many cardiologists. Sufficiently robust data from long-term follow-up of patients with hypertrophic obstructive cardiomyopathy treated with ASA were obtained in the past few years only, demonstrating the safety and long-term efficacy of ASA. This article deals with a history of the above-mentioned interventional procedure, its indications, technical aspects, safety, and long-term results.

Contents

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Characteristics of hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the most common cardiovascular disorder with a monogenic inheritance, characterized by myocardial hypertrophy without a clear external cause like hypertension or aortic stenosis [1,2] (Fig. 1). This definition is very general and aims to make the diagnosis of HCM as easy as possible while leaving out the fact that hypertrophy may be caused by other factors, such as storage of certain substances in the extracellular space (amyloidosis), or that it can only involve the basal segment of the interventricular septum like in older hypertonics with sigmoid septum. On the other hand, it makes the diagnosis easier for cardiologists despite the fact that it ''pulls together'' patients suffering from obviously different disorders.

The genetic background of HCM is extremely heterogeneous and the same statement applies to the phenotype of the disease. Heart morphology may vary greatly, especially concerning the extent and localization of myocardial hypertrophy, anatomy of papillary muscles, length of tendinous chords and of mitral valve cusps, angle between the root of aorta and interventricular septum, and presence/extent of largely fibrotic myocardial foci and of foci consisting of myocardial disarray. Hypertrophy of the interventricular septum, longer cusps of the mitral valve, and abnormalities of papillary muscles are the most common reasons why an obstruction of the left ventricular outflow is seen in about two thirds of patients [3]. Heterogeneity of the genetic substrate and of heart morphology is mirrored by the wide range of clinical manifestations and courses of this cardiomyopathy. Many individuals with HCM are asymptomatic, the prognosis of some is similar to that of the general population while other subgroups have much worse expectations including the risk of sudden cardiac death or heart failure [4].

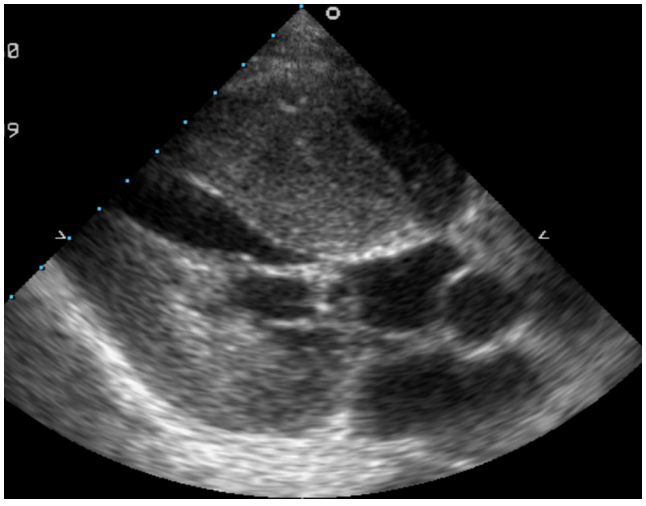

Fig. 1 – Transthoracic echocardiography, parasternal long axis view. Typical presentation of severe HOCM with marked hypertrophy of the interventricular septum and subaortic obstruction. This obstruction is caused by anterior movement of the anterior cusp of the mitral valve and its tendinous chords together with the thickened interventricular septum.

History and nomenclature

Opinions concerning who was the first to describe HCM may vary but most agree that the first description was published by forensic pathologist Teare more than 50 years ago [5]. He was not sure himself what illness he was describing when being repeatedly confronted with cardiac hypertrophy in young victims of sudden death. He thought that cardiac tumors may have been present. Given the above mentioned heterogeneity of the heart morphology, many other publications dealing with the same disorder each used their original name for it. The nomenclature thus became completely chaotic but fortunately it was unified in quite a determined way by other authors in the 1970s, pursuing the term ''hypertrophic cardiomyopathy''. Thanks to professor Gregor, this term was adopted in the Czech Republic as well and we have no doubt about what disorder is being discussed when HCM is mentioned today.

Therapy of hypertrophic cardiomyopathy

Studies dealing with HCM are usually characterized by small numbers of participants – tens of subjects are typical, hundreds exceptional, more than thousand represent a rarity. The number of randomized trials in the history of HCM could also be counted using the fingers of two hands [6]. This fact – along with the pronounced morphological and clinical heterogeneity of the disease – has a profound effect on the robustness of our knowledge and on the assessment of effectiveness of our therapeutic approaches, too.

Pharmacotherapy of HCM has not changed nor developed much since its earlier diagnosis based on echocardiography findings. Logically, symptomatic patients are given medications decreasing the use of oxygen and prolonging the diastole. We have been doing this for decades using beta blockers and verapamil. Attempts to decrease the risk of sudden death with pharmacotherapy have failed; patients who were symptomatic despite taking the appropriate medications or at risk of sudden death were thus offered non-pharmacological therapeutic approaches [1,2].

One of the first was myectomy, which – especially in its extensive form – proved to be a very effective therapeutic tool in the hands of experienced surgical teams, diminishing or even eliminating the intraventricular obstruction [7,8]. Myectomy is based on excision of several grams of myocardium, usually protruding from the hypertrophic basal segment of interventricular septum into the outlet tract of the left ventricle. The growing body of experience of several surgical teams in large countries where it was possible to concentrate patients with HCM has led to improvements in the surgical technique and widening of the originally small myotomy, then excision and eventually plastic of the papillary muscles and the entire mitral apparatus. Good surgical results were followed by publications describing elimination of intraventricular obstruction, further decrease of left ventricular hypertrophy and marked reduction in symptoms [9]. One problem concerning myectomy has not been solved until now, namely the relative complexity of the entire procedure and the