Intrapericardial treatment of persistent autoreactive pericarditis/myopericarditis and pericardial effusion

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For the quite small international group of cardiologists specializing in diseases of the pericardium, the most vexing of these is recurrent pericarditis. Virtually never due to an identifiable microorganism or systemic disease (at least after the index attack), most patients have either recurrent pericardial effusions or, more often, extremely unpleasant, disabling chest pain which nearly always yields to systemic administration of a corticosteroid, especially after failure of non-steroidal agents. This is followed by one of two courses: either recurrent pericarditis: discrete recurrences after days, weeks or months or incessant pericarditis: resistance to attempts to completely taper the corticosteroid, with symptoms repeatedly returning at each patient’s customary threshold level[1].

The latter patients may be fairly described as ‘steroid hooked’[1] and have a wide variety and intensity of corticosteroid side effects making life even more unpleasant. While methods are under development to permit entering the ‘dry’ pericardium[2], Professor Maisch and his collaborators have had extraordinary success in managing recurrent, presumably immunopathic (autoreactive) pericardial effusions with direct intrapericardial therapy[3]. Whereas most diagnostic evaluations of any kind of pericardial disease are limited to familiar laboratory procedures — negative results leaving a large number of bacteriologic, virologic ‘idiopathic’ cases — Maisch et al. have performed the most thorough set conceivable of hematologic, chemical and immunologic tests on pericardial fluid and tissue as well as blood[3].

Corticosteroid therapy of pericardial disease

Intrapericardial corticosteroid therapy has a long history, particularly for patients in whom hydrocortisone was employed in attempts to prevent constriction following tuberculous pericarditis and in some patients with uremic effusions[4-5]. (Oral or parenteral corticosteroids have failed to prevent constriction in tuberculous cases and the limited use of intrapericardial hydrocortisone was inconclusive.) Maisch and colleagues have successfully used triamcinolone which is usually non-absorbable (30% transient Cushing syndrome in a high dose group and only 13% in a lower dose group.) They concluded that the lower dose, 300 mg . m\(^{-2}\).24 h\(^{-1}\), prevented recurrences of symptoms and relapses of effusion equally as effectively as 600 mg . m\(^{-2}\).24 h\(^{-1}\), but with less than half the side effects[3].

After one year, successful treatment remained in over 80% of the patients, a relatively small slippage from the approximately 90% success after 3 months. The investigators made meticulously thorough diagnostic efforts to exclude viruses and bacteria in the effusion fluid and in epicardial biopsies as well as by immunohistochemistry and immunocytochemistry of epicardial and endomyocardial biopsies. These resulted in 84 out of a total group of 260 patients who could be firmly classified as ‘autoreactive’[3]. Without the specialized immunologic investigations such patients would have been consigned to the ‘idiopathic’ category. That category should always be regarded as unlikely since no disease is truly idiopathic or *sui generis* — labels that are always a confession of diagnostic defeat.

**Effusions and the visceral pericardium**

It is particularly significant that these meticulous investigators biopsied the epicardium (visceral pericardium)[3], since effusions uniformly develop across this membrane and its adjacent myocardium[6]. (This probably accounts for the frequently disappointing biopsy results in previous years, which were mainly of the parietal pericardium.) Of course, the use of a thoracoscope was essential so that the tissue could be removed with this visual aid — it would not do to biopsy an epicardial coronary vessel. The current report is the latest achievement in this outstanding group’s over 20 years of investigations of many, but particularly immunopathic, aspects of pericardial disease.
Recurrent and incessant pericarditis: intrapericardial triamcinolone therapy

In my experience the largest individual group of sufferers from recurrent and especially incessant pericarditis have been those without recurrent effusions who are presumed, though rarely firmly demonstrated (as here), to have an immunopathy, and especially those who are ‘steroid hooked’[1]. Indeed, there is some evidence that corticosteroid treatment has been necessary to initiate the cycle of recurrences[7–9]. Moreover, recurrent episodes appear to be more frequent when corticosteroid therapy is prescribed during the first attack of pericarditis compared to patients not so managed[7–9]. It is here that one hopes that either a thoracoscope or the specialized instrument for penetrating the ‘dry’ pericardium (PerDUCER[2]) can be used to approach this problem. Since the patients almost uniformly respond to systemic corticosteroid there is every hope for the success of an agent like triamcinolone safely deposited in the wet or dry pericardium in a concentration that would be unreachable with oral or parenteral therapy and usually without detectable side effects, which, in any case, could not become chronic. Professor Maisch and colleagues[3] have established the efficacy and safety of intrapericardial triamcinolone for autoreactive effusive pericarditis and myopericarditis. It is to be hoped that in appropriate clinical trials they and other investigators can broaden its indications.

References


Homocysteine and vascular disease

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Although case control studies remain robust in linking an elevated plasma homocysteine concentration to the development of premature atherosclerosis and thrombosis, prospective studies have been less convincing. On balance, however, it is generally accepted that an elevated level of this amino acid, common in patients with atherosclerosis, constitutes a weak but independent risk factor for the development of vascular disorders and may also be a determinant of prognosis. The concentration of plasma homocysteine is inversely related to that of folate as well as plasma vitamins B12 and B6[1]. Inadequate concentrations of B vitamins are common among those with high homocysteine levels. Hence vitamin therapy has received much attention as a possible treatment option in patients with vascular disorders.

Treatment studies — a perspective

Early intervention studies by Wilcken et al.[2], Boers et al.[3] and Brattstrom et al.[4] showed the usefulness of folic acid treatment in patients with vascular