On the basis of these observations, the role of impaired diastolic suction in the genesis of changes in right ventricular filling dynamics that we described during PTCA doesn’t seem to be supported by the available data.

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References

Harmful effect of enalapril on left ventricular remodelling in patients without a severe residual stenosis after acute anterior wall infarction?

Baur et al.[1] have shown in their placebo-controlled study that enalapril therapy in patients with acute anterior wall myocardial infarction treated with primary angioplasty or thrombolytic therapy, had no effect on left ventricular end-diastolic volume index (EDVI) at 3 weeks and after 1 year. However, in a subgroup of patients (36 of 56) with a residual stenosis of ≥70% in the infarct related artery (IRA) the enalapril treated patients showed a significantly less increase in EDVI during follow-up (4.2 ml·m⁻² vs 18.8 ml·m⁻²), and the authors concluded that enalapril prevented left ventricular remodelling in this subgroup.

Results for the remaining patients (20 of 56), i.e. with a residual stenosis <70% of IRA, were not presented and thereby the authors entered a rather common pitfall regarding subgroup analysis. Thus, for these 20 patients, based on data from Table 3, I calculate the mean increase in EDVI after 1 year to 39.3 ml·m⁻² for the enalapril treated group and to 12.5 ml·m⁻² for the placebo treated group (statistical significant P<0.05 with an estimated coefficient of variance of 90%). Should this sizable difference not be statistically significant, the statistical power of the study to justify subgroup analysis might be questioned. Accordingly, in my opinion, either or neither of the following conclusions should be drawn from the present study:
(1) In patients with an anterior wall infarction and a severe residual infarct-related stenosis following reperfusion, treatment with enalapril prevents the process of ventricular remodelling.
(2) In patients with an anterior wall infarction without a severe residual infarct-related stenosis following reperfusion, treatment with enalapril augments the process of ventricular remodelling.

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Serum lactate dehydrogenase activity: indicator of the development of pneumonitis induced by amiodarone

Many drugs including amiodarone are known to adversely affect the lung parenchyma. The incidence of this pulmonary toxicity is 5% to 10%[1]. It may take months to resolve after discontinuation of amiodarone due to the long half-life (up to 60 days)[1]. It is important to determine the risk for each patient to develop pulmonary adverse effects. However, until now no appropriate parameters indicating pulmonary side effects are available. We report an increase of the serum lactate dehydrogenase (LDH) activity prior to and during the disclosure of a drug-induced pneumonitis caused by amiodarone. A 72-year-old female was admitted to hospital because of complaints of shortness of breath. She was treated with amiodarone for 2 years because of a history of atrial fibrillation. A chest roentgenogram showed a diffuse reticulonodular pattern, indicative of interstitial abnormalities, which was confirmed by a HRCT. Bronchoalveolar lavage fluid (BALF) analysis revealed an increased number of cells, the presence of plasma cells and lipid-laden, ‘foamy’ alveolar macrophages. Culture remained sterile. Both in serum and BALF, the LDH level was increased. In serum mainly LDH₁ and LDH₂ were increased, whereas in BALF LDH₁ and LDH₄ were high. Moreover, the arterial paO₂ was decreased (63 Kpa). All other laboratory tests — including liver function tests — were normal. The diagnosis drug-induced pneumonitis was considered based on the clinical picture and the BALF analysis results. After discontinuation of amiodarone, the clinical condition improved, in addition to the paO₂ and chest roentgenogram. Moreover, the serum LDH activity decreased gradually (Fig. 1). The extracellular appearance of LDH, a cytoplasmatic enzyme, indicates cell damage or cell death[3]. Although the increase in total serum LDH activity is rather nonspecific, measurement of LDH activity in serum, pleural effusion and BALF has been reported useful in monitoring pulmonary cell injury[3-5]. It has been suggested that the influx of inflammatory cells causes increase of LDH activity[3-4]. Like paraffins, the phospholipid inclusions — indicated by the ‘foamy’ appearance of the alveolar macrophages — are noncorrosive, but interact with pulmonary surfactant, which probably causes damage to the alveolar walls, and additionally, the alveolar/blood barrier[2-3]. This causes leakage of LDH from the pulmonary interstitium to the blood.


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