Persistent elevated jugular venous pressure and pleural effusion following myocardial infarction

Case report

A forty-nine-year-old man without clinical symptoms nor relevant illnesses prior to the hospitalisation was admitted due to a large anteroseptal and apical infarct and presented with biventricular dysfunction. During follow-up ejection fraction of the patient with one vessel disease rose from 31 to 45% within four weeks following percutaneous revascularisation of an occluded middle left anterior descending artery (LAD) by use of a stent. Despite high doses of various diuretics such as furosemide and aldactone, in addition to angiotensin converting enzyme inhibitor, digoxin and betablockade, follow-up was characterised by persistent elevated jugular venous pressure (fig. 1) with pleural effusion mainly on the right side (fig. 2), indicating right ventricular dysfunction or hypotensive state. Suprasternal colour Doppler sonography revealed thrombosed proximal superior vena cava with collaterals (fig. 3). Computer tomography showed an enlarged azygos vein with thrombosed proximal superior vena cava and multiple collaterals (fig. 4) and retrosternal calcification (fig. 5). However, distal vena cava was open (fig. 6). Lymph node and adjacent tissue inflammation most probably due to tuberculosis in early childhood caused the thrombosis in the proximal part of the superior vena cava.

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Elevated jugular venous pressure is independently associated with adverse outcome in heart failure, including progression of the disease. However, if venous obstruction is limited to the upper extremities with visible collaterals (figure 7), obstruction in the superior vena cava has to be considered for the cause of persistent pleural effusion.

Key words: superior vena cava; thrombosis; pleural effusion; elevated jugular venous pressure

References