

Case Report

Pleural effusion as a result of chronic renal ischemia

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ABSTRACT

We would like to present a case of patient with a transudative pleural effusion as a result of atherosclerotic occlusion of renal arteries. About 50 liters of fluid were drained from the right pleural cavity during 10 months period of observation. Successful revascularization of kidneys improved left ventricular function, stabilized hemodynamic of the pulmonary circulation and thus led to elimination of pleural effusion.

Key Words:

pleural effusion; transudate; renal artery

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Introduction

The diagnosis and treatment of pleural effusion is one of the most relevant problems in pulmonary medicine. Pleural effusions can be developed as a complication of at least 50 different diseases, but chronic renal ischemia was not mentioned in already published data as one of the predisposing conditions of pleural effusion (1,2). We would like to present a clinical case of pleural effusion, which appeared to be a result of atherosclerotic obstruction of renal artery, and to discuss the mechanism of its development.

Case report

A 67-year-old man admitted hospital in April 2007 with complains on dyspnea associated with reoccurring accumulation of fluid in the right pleural cavity, and persistent arterial hypertension (170/100 mmHg) in spite of ongoing three-drug antihypertensive therapy.

A year earlier (May 2006) patient underwent coronary artery bypass surgery. Pleural effusion in the right pleural cavity appeared first time in June 2006. Since then, over the past 10 months, about 50 liters of transudative fluid were drained from the right pleural cavity.

Physical examination at admission revealed that breath sounds on the right side were sharply weakened. Chest X-ray examination diagnosed fluid in the right pleural cavity coming up to the 5th rib. Serum creatinine level was 0.22 mmol/l, urea - 11.6 mmol/l, tubular filtration rate - 29 ml/min. Investigation of fluid from the pleural cavity revealed a transudate with high creatinine concentration (0.20 mmol/L or 2.3 mg/dL).

Ultrasound examination founded out that size of the left kidney was decreased. Echocardiography showed increase of the left ventricle up to 6.9 cm, left ventricular ejection fraction appeared to be - 36%, systolic pressure in the pulmonary artery - 42 mmHg. Aortoangiography revealed marked atherosclerosis of the abdominal aorta, subtotal occlusion of the left renal artery, stenosis of the right renal artery up to 85% of its diameter.

The patient underwent transcatheter endovascular angioplasty with stent implantation in both renal arteries. Immediately after stent implantation in the renal arteries we observed marked stabilization of the systemic arterial pressure with mean level of 120/80 mmHg. The patient felt himself much better, dyspnea was decreased. According to the results of follow-up X-ray examination of the thorax, there was no evidence of fluid in the pleural cavity. The patient was discharged from the hospital in a satisfactory condition.

During examination after twelve months since angioplasty patient had no complains. Computed tomography revealed no evidence of pleural effusion. Mean arterial pressure was 130/70 mmHg, and appeared to be stable on three-drug antihypertensive therapy in average doses. Echocardiography revealed increase of left ventricular ejection fraction up to 64%, systolic pressure in the pulmonary artery was reduced to 28 mmHg. However overall renal function remained unchanged (serum creatinine 0.20 mmol/l, tubular filtration rate 29 ml/min).

No potential conflict of interest.

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Discussion

This case demonstrates clinical relation between atherosclerotic stenoses of renal arteries and pleural effusion.

It is well known that the most common cause of transudative pleural effusion is circulatory insufficiency (3). The patient had complications of ischemic heart disease, pulmonary hypertension, decreased ejection fraction of the left ventricle, and all the attempts of therapeutic treatment of these conditions had no influence on pleural effusion. Chronic renal insufficiency, considered as a cause of pleural effusion is affecting circulation by means of hypervolemia, which appears to be caused by water-electrolyte disbalance (1,2,4). But in the presented case, it was observed that the serum creatinine level and tubular filtration rate were unchanged despite renal revascularization. So we can exclude renal insufficiency as the primary cause of pleural effusion.

Also stenoses of the renal arteries can lead to activation of renin - angiotensin - aldosterone system (5).

It was already published that diastolic dysfunction of the left ventricle develops faster and is more severe in patients with occlusion of the renal artery, than in patients with essential arterial hypertension (6). Furthermore diastolic dysfunction of the left ventricle causes chronic congestive heart failure. Thus we can speculate that decrease in activity of renin - angiotensin

-aldosterone system after renal revascularization caused an improvement of left ventricular function and dissolution of pleural effusion.

It is possible to suppose that in the same clinical cases renal revascularization can be profitable despite of kidneys excretory function.

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