Clinical Reports

Reversibility of myocardial dyskinesia due to severe hypertension

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Summary: Two cases of acute left ventricular failure associated with severe essential hypertension are presented. On admission echocardiography indicated severe dyskinesia of all wall segments of the heart. Anti-hypertensive treatment resulted in significant improvement in clinical and echocardiographic findings.

Introduction

Echocardiography has become an important tool in the work-up of patients with hypertension. As the incidence of left ventricular wall thickening increases with age and the height of systolic blood pressure,¹ it is often believed that cardiac hypertrophy reflects longstanding pressure overload of the heart. On the other hand, in hypertension of rapid onset left ventricular mass usually is normal, although marked functional abnormalities may be found.²

We describe here two patients with an acute hypertensive state in whom the most prominent echocardiographic finding was severe myocardial hypokinesia which turned out to be almost completely reversible during anti-hypertensive treatment.

Case reports

Patient 1

A 38 year old Caucasian female with a history of allergic asthma but without coronary complaints was referred to the hospital because of acute dyspnoea. On admission, she showed signs of confusion and reduced consciousness. Blood pressure was 240/150 mmHg and pulse rate 102/minute. A third heart sound was present and diffuse crackles were heard. Fundoscopic examination was unremarkable. Electrocardiography showed left

and right atrial overload, but no signs of ventricular hypertrophy. Laboratory tests revealed normal renal function and no albuminuria. Plasma noradrenaline was markedly elevated: 18.47 nmol/l (normal < 3.0 nmol/l). Plasma adrenaline and dopamine were only slightly increased and renin levels were within the normal range.

Echocardiography showed severe hypokinesia of all wall segments (Figure 1) and a decrease of transmitral flow. Continuous wave Doppler examination of mitral flow displayed a significant increase in the A-top, indicating a delay in mitral valve opening. Left atrial dimensions were at upper normal limits. There were no signs of left ventricular hypertrophy or valvular disease. Early diastolic velocity (*E*) was reduced and late velocity following atrial contraction (*A*) increased, yielding an *E/A* ratio of less than one. On haemodynamic evaluation increases in wedge pressure (22 mmHg) and right atrial pressure (12 mmHg) were found.

The patient was treated initially with intravenous nitroprusside and diuretics, and afterwards with nifedipine and enalapril orally. During follow-up control echocardiograms indicated remarkable recovery of myocardial contractility. Four months after admission the medication was discontinued temporarily to allow a more thorough evaluation of suspected secondary hypertension. Repeat haemodynamic measurements now yielded a blood pressure of 150/100 mmHg and a wedge pressure of 10 mmHg. Right atrial pressure turned out to be 5 mmHg. At that time a repeat echocardiogram showed only slight hypokinesia of the interventricular septum (Figure 1). Plasma noradrenaline was within normal limits (1.45 nmol/l). Further work-up for secondary hypertension was negative.

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Figure 1 M-mode echocardiographic scan with the transducer placed in the fourth intercostal space. Septal motion (LS) and posterior left ventricular endocardium (EN) motion are markedly decreased on admission (left) and returned to near normal after treatment (right).

Patient 2

A 45 year old Caucasian female was referred because of progressive dyspnoea. During the previous month she had experienced slight dyspnoea on exertion but otherwise her history was unremarkable. Blood pressure on admission was 220/140 mmHg and pulse rate 120/min. A third heart sound was present and crackles were audible at the lung bases. Fundoscopy showed grade II hypertensive abnormalities.

Serum creatinine was within normal limits and no albuminuria was found. Plasma noradrenaline was 5.06 nmol/l; normal values were obtained for other pressor hormones, including adrenaline and renin. On echocardiography severe hypokinesia of all myocardial wall segments was noted with only borderline left heart enlargement. The *E/A* ratio was below one. A thermodilution catheter was inserted, revealing that wedge pressure was 24 mmHg, right atrial pressure 12 mmHg and cardiac index 1.71 litres/minute/m².

Treatment was started with frusemide and nifedipine. Blood pressure fell to 130/85 mmHg and physical signs of cardiac failure abated. A control echocardiogram, obtained 4 weeks after starting therapy, indicated significant recovery of left ventricular wall contractility. Work-up for secondary hypertension was negative.

Discussion

Severe or established hypertension can be complicated by structural and functional cardiac abnormalities such as reduced ventricular compliance, elevated left ventricular end diastolic filling pressures with dilatation of the heart, decreased transmitral flow and ultimately symptomatic heart failure.^{2,3} The two patients described here presented with overt left ventricular failure as the initial clinical manifestation of severe essential hypertension. Neither one of them had a history of coronary heart disease. Still, stunned myocardium was documented echocardiographically in the absence of significant hypertrophy or secondary organ damage. In our first patient we found remarkable improvement on a repeat echocardiogram at a time that she was temporarily untreated. Blood pressure at that time was only marginally elevated which, however, is not too surprising because it may take several months before pressure rises again after cessation of treatment.

Echocardiographic features of malignant hypertension have been reported by Shapiro and co-workers.² From their data it is apparent that left ventricular cavity dimensions in systole and diastole may remain normal, but that mitral valve opening may be significantly delayed (as we also observed in our first case). In our patients the severe

increase in pressure probably was of recent origin but despite normal cardiac dimensions left ventricular function was grossly impaired with extreme hypokinaesia of the left ventricular wall. The E/A ratio was below one in both of them, suggesting reduced left ventricular compliance.

A remarkable finding in our cases was the isolated increase in plasma noradrenaline in the absence of a phaeochromocytoma. Although we cannot entirely exclude the possibility that this was secondary to the pulmonary oedema (especially not in patient 2), we think such a mechanism to be less likely because one would have expected a comparable rise in adrenaline and activation of renin.⁴ The question arises, therefore, whether acute

oversecretion of noradrenaline was the primary event that has induced contraction of the coronary arteries with subsequent ischaemia and hypokinaesia of the heart (stunning). In other words, left ventricular dysfunction during acute episodes of hypertension may be due to a direct effect of catecholamines on the heart rather than to pressure overload as is also observed in patients with phaeochromocytoma.^{5,6}

In conclusion, the present findings demonstrate that sometimes severe myocardial hypokinaesia (stunning) may accompany severe hypertension in the absence of overt coronary disease. Perhaps excess secretion of noradrenaline is involved in the pathophysiology of this phenomenon.

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A feminizing adrenocortical carcinoma presenting with gynaecomastia

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Summary: A case is presented in which gynaecomastia was the sole initial presenting symptom of a feminizing adrenocortical carcinoma. This rare pathological lesion is discussed.

Introduction

Adrenocortical neoplasms are rare. They may be classified into benign and malignant, and functioning and non-functioning types. All may produce

local signs and symptoms, such as loin pain, a mass, or even haematuria if the kidney is invaded.¹ Malignant tumours of the adrenal cortex are considered to have a very poor prognosis. The functioning types may manifest themselves through their hormonal effects; indeed, these may be the only presenting features in some cases. A case is presented in which gynaecomastia was the

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