IS CAPTOPRIL EFFECTIVE IN PRIMARY PULMONARY HYPERTENSION?

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Several vasodilating agents have been used for the treatment of primary pulmonary hypertension (PPH). However, no effective therapy is currently available for this distressful disease. We tried to use an angiotensin-I converting enzyme inhibitor, captopril, for one woman suffering from PPH. Her initial treatment with furosemide, digoxin and spironolactone had shown little effect. Further administration of captopril improved her clinical condition from NYHA IV to II and made it possible to treat her at our outpatient clinic. No adverse effect has been found. We would like to appreciate captopril as one of the effective drugs in the treatment of PPH.

Many reports concern the etiology and pathophysiology of primary pulmonary hypertension (PPH). However, they still remain obscure. In its natural course, death occurs several years after the onset of symptoms such as exertional dyspnea, syncope and chest pain. Although several pulmonary vasodilatating agents have recently contributed to the treatment of PPH, an effective therapy has not been established for this distressful disease. In this report, we will present a patient who showed a significant improvement of symptoms and laboratory data with an oral administration of angiotensin-I converting enzyme inhibitor, captopril.

**Case Report**

The patient, a 53 year old female, had dyspnea at rest, syncopal attack and chest pain, with a functional capacity of class IV of the NYHA. Her hematological and routine laboratory findings were normal. A chest X-ray showed marked cardiac enlargement and protrudent main pulmonary artery with peripheral tapering. Her ECG exhibited typical right ventricular hypertrophy. Echocardiography revealed the right ventricular dilatation and marked pericardial effusion. Cardiac catheterization confirmed her diagnosis of PPH (Table I).

The treatment, started initially with a combin-

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<th>TABLE I DATA ON CARDIAC CATHETERIZATION</th>
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<tr>
<td>Pulmonary capillary wedge pressure</td>
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<td>Pulmonary artery pressure</td>
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<td>Right ventricular pressure</td>
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<td>Right atrial pressure</td>
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<td>Aortic pressure</td>
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<td>Cardiac index</td>
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Key Words: Primary pulmonary hypertension Captopril

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ed administration of 0.25 mg of digoxin and 40 mg of furosemide daily had brought no remarkable improvement. Further administration of 75 mg of spironolactone and an increment of furosemide doses up to 80 mg daily, still showed little effect. However, the administration of captopril, 25 mg orally, three times daily, significantly improved the symptoms and laboratory data. After 4 weeks of captopril therapy, dyspnea at rest disappeared. The central venous pressure and the cardiothoracic ratio decreased from 15 to 7.5 cmH₂O and from 72 to 58%, respectively. Pericardial effusion was also reduced. Her clinical course is summarized in Fig. 1.

Hemodynamic parameters were analyzed one hour after a single oral administration of 50 mg of captopril. Mean pulmonary artery pressure fell from 64 to 49 mmHg. Pulmonary vascular resistance decreased from 1131 to 818 dyn·s·cm⁻⁵. Cardiac index did not change significantly (2.46 to 2.56 L/min/m²) (Table II). The results observed in this patient, may suggest the noteworthy effectiveness of captopril in PPH. After discharge, she is in fairly good condition on 75 mg of captopril. No adverse effect of the drug has been found.

The effectiveness of captopril on congestive heart failure has been reported. The action mechanism of captopril on pulmonary vascular beds is not fully understood. From our present experience, administration of captopril, may be considered one of the promising therapies for PPH.

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