greater than 4 cm is always undertaken, whether in adults or younger subjects. The occurrence of primary lung tumors in children younger than 16 years is uncommon, and the finding of primary pulmonary teratomas in this population group is even more infrequent. Therefore, only 2 of the 230 primary pulmonary neoplasms in children reported by Hartman and Shochat were classified as true primary lung teratomas. Before accepting a diagnosis of primary lung germ-cell tumor, it is mandatory to exclude, through an exhaustive search, a possible primary gonadal or extragonadal localization that could have escaped clinical detection.

Despite its rarity, it is known that a real primary mediastinal, sacrococcygeal, or even gonadal teratoma can be clinically silent and therefore remain undiagnosed. This difficulty in the right diagnosis of germ-cell tumors is increased by a frequently atypical presentation of extragonadal teratomas. The mediastinum is the most common primary localization of extragonadal teratoma in the adult, while the sacrococcygeal localization is the usual primary area of involvement in children, with just a few well-documented cases of the same diagnosed later in life. Only one of the 24 cases of sacrococcygeal teratomas in adults reviewed by Miles and Stewart were malignant, while it is known that those tumors diagnosed in children are characterized by their tendency for malignant change with increasing age, especially after the age of 4 months. It is considered that about 10 percent of sacrococcygeal teratomas in adults will undergo a malignant transformation. Opposite to what happens in the infantile variety, sacrococcygeal teratomas in adults are usually completely intrapelvic, without any externally visible tumor. Although many patients are asymptomatic, pain is the most common symptom. The mental retardation of our patient may suggest an explanation for the absence of complaints despite the wide sacrococcygeal bone involvement on roentgenographic examination. Not one of the previously reported cases presented as a primary lung process. The clinical respiratory features of the rare reported cases of primary lung germ-cell tumors or pulmonary metastasis from primary mediastinal and gonadal teratomas are usually not specific. In general, thoracic radiologic manifestations are also not diagnostic.

The presence of the air-crescent sign (Monod's sign) raises a quite broad differential diagnosis. To our knowledge, this is the first description of this classic radiologic sign due to pulmonary teratoma. Since that sign has been reported in some tumors characterized by the presence of intratumor necrosis, it seems logical to find it in pulmonary localizations of germ-cell tumors because of the potentially high biologic aggressiveness of these tumors, particularly when a yolk-sac tumor component is present. The identification of these histologic areas of yolk-sac cells, as we observed in our case, shows a very good correlation with the finding of high α-fetoprotein blood levels and a malignant behaviour of the tumor.

REFERENCES

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Acute Nonhemodynamic Pulmonary Edema with Nifedipine in Primary Pulmonary Hypertension*

Thierry Prigogine, M.D.; Yves Waterlot, M.D.; Paul Gottignies, M.D.; Anne Verhoeven, M.D.; and Philippe Decroly, M.D.

A 34-year-old man with primary pulmonary hypertension developed acute nonhemodynamic pulmonary edema after a loading dose of nifedipine. Changes of the vascular permeability induced by the drug acting on the arteriolar wall of the capillary system could be an explanation.

(Chest 1991; 100:563-64)

PPH = primary pulmonary hypertension

Nifedipine, a calcium channel blocker, is considered to be a potentially useful agent in the management of some patients with primary pulmonary hypertension (PPH). Transient mild side effects attributable to its vasodilator action have been reported in the form of headache, hypotension, dizziness, and flushing. More serious reaction, such as excessive hypotension and pulmonary edema, can also occur.

We describe a patient with PPH who developed acute pulmonary edema after a loading dose of nifedipine without significant change of pulmonary vascular resistance or filling pressures of the left ventricle.

CASE REPORT

A 34-year-old white man fulfilling the diagnostic criteria for PPH based on history, physical examination, chest roentgenogram, lung scan, pulmonary function testing, two-dimensional echocardiography, and absence of left-to-right shunting or elevated left heart pressures on catheterization was admitted to the ICU to study the clinical and hemodynamic effects of oral nifedipine with a thermodilution flow-directed catheter in the pulmonary artery. Basal measurements were recorded (Table 1).

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Table 1—Hemodynamic Changes After Nifedipine

<table>
<thead>
<tr>
<th></th>
<th>PaO₂, mm Hg</th>
<th>Cardiac Output, L/min</th>
<th>Pulmonary Arterial Pressure, mm Hg</th>
<th>Pulmonary Vascular Resistance, dyn cm⁻¹</th>
<th>Capillary Wedge Pressure, mm Hg</th>
<th>Systemic Systolic Blood Pressure, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>62</td>
<td>4.9</td>
<td>102</td>
<td>70</td>
<td>985</td>
<td>9</td>
</tr>
<tr>
<td>10 h</td>
<td>59</td>
<td>4.7</td>
<td>115</td>
<td>72</td>
<td>1050</td>
<td>10</td>
</tr>
<tr>
<td>11 h (1h after cessation of drug challenge)</td>
<td>ND</td>
<td>4.9</td>
<td>127</td>
<td>76</td>
<td>1190</td>
<td>3</td>
</tr>
<tr>
<td>14 h</td>
<td>ND</td>
<td>4.8</td>
<td>70</td>
<td>49</td>
<td>650</td>
<td>10</td>
</tr>
<tr>
<td>18 h</td>
<td>38</td>
<td>5.1</td>
<td>125</td>
<td>68</td>
<td>988</td>
<td>5</td>
</tr>
</tbody>
</table>

Nifedipine was administered according Rich's protocol using high doses of the calcium channel blocking drug. After 10 h, the patient received 200 mg nifedipine without any positive response. Therefore, drug challenge was stopped, the patient remaining under close surveillance. A transitory decrease of the pulmonary arterial pressure and of the pulmonary vascular resistance without change of the capillary wedge pressure was observed 4 h after cessation of drug challenge.

Four hours later, he became dyspneic with clinical and roentgenographic evidence of pulmonary edema (Fig 1).

Hemodynamic measurements demonstrated a recurrence of the severe pulmonary hypertension, lower PaO₂ without change of the capillary wedge pressure which remained low (Table 1).

The patient recovered after administration of corticosteroids, furosemide, and oxygen. Combined heart-lung transplantation was done three months later. Histologic studies revealed typical aspects of advanced plexogenic pulmonary arteriopathy.

**DISCUSSION**

Despite frequently serious adverse hemodynamic and clinical reactions, patients with PPH continue to receive vasodilators in the hope that some will benefit. More recently, high doses of calcium channel blocking agents were recommended in order to identify those patients in whom conventional doses appear inadequate.

Nifedipine, at low dosage, by its vasodilatory effects on resistance vessels, represents a very effective drug in the treatment of hypertension with acute pulmonary edema, where left ventricular after-load reduction is mandatory. Such patients benefit from its systemic effects without undue deleterious consequences from its subtle inotropic action.

Few cases of pulmonary edema induced by nifedipine are reported in the literature especially in patients with outflow tract obstruction due to aortic stenosis, hypertrophic cardiomyopathy, or in PPH.

Since nifedipine has negative inotropic effects, it would be conceivable that our patient developed cardiogenic pulmonary edema and that his left ventricular function had compensated by the time hemodynamic measurements were made. Such hypothesis would be improbable as repeated capillary wedge pressure measures and cardiac outflow remained stable.

Moreover, aspiration of gastric content is also excluded by the absence of any digestive disturbances.

Ankle edema is a classic but infrequent complication of nifedipine and other dihydropyridine treatment; its pathogenesis remains unclear; sodium retention has been excluded by renin-angiotensin studies. Moreover, salidriuretic administration does not have any effect on their evolution; changes of the vascular permeability induced by the drug acting on the arteriolar wall of the capillary system could be an explanation. The wet lung that we report could be caused by such mechanism on deteriorated pulmonary vessels.

This report emphasizes the necessity of testing high doses of calcium channel blockers with direct hemodynamic monitoring to watch for adverse hemodynamic effects.

**REFERENCES**


**FIGURE 1.** PA chest x-ray film showing features of pulmonary edema.