Pulmonary Hypertension in Patients With Human Immunodeficiency Virus Infection

Comparison With Primary Pulmonary Hypertension

Patrick Petitprez, MD; François Brenot, MD; Réza Azarian, MD; Florence Parent, MD; Bernadette Rain, MD; Philippe Herve, MD; Gérard Simonneau, MD

Background
Previously reported cases of patients with pulmonary hypertension (PH) and human immunodeficiency virus (HIV) infection are poorly documented regarding baseline hemodynamics and potential for pulmonary vasodilatation. The purpose of this report was to compare HIV-infected patients who had PH with non–HIV-infected patients who had primary pulmonary hypertension (PPH) in terms of (1) clinical characteristics, (2) hemodynamics in baseline conditions and during a short-term vasodilator trial with epoprostenol, and (3) survival.

Methods and Results
Between April 1987 and August 1992, 20 HIV-infected patients with PH and 93 non–HIV-infected patients with PPH were referred to our department. At the time of referral, baseline right-side heart hemodynamics were obtained in addition to demographic variables and medical history. A short-term vasodilator trial with epoprostenol was performed in 19 of 20 HIV-infected and 86 of 93 non–HIV-infected patients. Outcome and survival were analyzed and compared for both groups (22 transplant recipients were excluded from the group of patients with PPH). At the time of diagnosis of PH, HIV-infected patients significantly differed from non–HIV-infected patients in age (32±5 versus 42±13 years; P<.05) and degree of disability (New York Heart Association functional class III or IV, 50% versus 75%; P<.01). The proportion of disease states known to be associated with PPH (Raynaud’s phenomenon, migraine, collagen disease without overt symptoms and signs, or a positive family history of PPH) was similar in the two groups. HIV-infected patients had a severe but significantly lower level of PH than patients with PPH. The percentage of responders to epoprostenol and the level achieved in pulmonary vasodilatation were similar in the two groups. PH was the cause of death in 8 of the 10 HIV-infected patients who died within 1 year after the diagnosis of PH. Overall survival was poor and not significantly different between the two groups. Pathological findings in lung tissue obtained from 3 HIV-infected patients were close to those seen in most of the lung specimens available from 27 patients with PPH and resembled plexogenic pulmonary arteriopathy.

Conclusions
These results support the view that HIV infection may now be regarded as another common disease state that can be associated with PPH development. The lower initial severity in HIV-infected patients may be due to the close medical attention usually devoted to such patients, who may account for an earlier diagnosis. However, the overall survival rate of HIV-infected patients with PH appeared to be as poor as in non–HIV-infected patients with PPH. (Circulation. 1994;89:2722-2727.)

Key Words
• mortality • prostacyclin • hemodynamics • hypertension • human immunodeficiency virus

Several reports of patients with human immunodeficiency virus (HIV) infection associated with pulmonary hypertension (PH) have recently been published.1-13 However, these reports have usually dealt with isolated cases or limited series with absent or incomplete hemodynamic evaluation, particularly in terms of response to vasodilators. Since April 1987, when we observed our first case of PH in a patient with HIV infection, we evaluated HIV-infected patients among patients referred to our department for primary pulmonary hypertension (PPH). We report 20 new cases of HIV-infected patients with PH in terms of clinical characteristics, complete hemodynamic evaluation including a short-term vasodilator trial with epoprostenol, and survival. These HIV-infected patients were compared with non–HIV-infected patients with PPH who had been referred during the same time period.

Methods

Patients

The diagnosis of PPH was established by the results of right-side heart catheterization (the presence of elevated mean pulmonary artery pressure >25 mm Hg and normal pulmonary wedge pressure) and after exclusion of potential causes of secondary PH according to the diagnostic criteria of the National Institutes of Health Registry on PPH.13 Patients with portal hypertension and/or cirrhosis were excluded, although there is debate as to whether this association disqualifies a patient from the diagnosis of PPH.

For each HIV-infected patient with PH, complete clinical information with follow-up to September 1992 was obtained by reviewing hospital and referring physician medical records, including age, sex, HIV risk factors, time between the discovery of HIV positivity and diagnosis of PH, staging of HIV infection according to the Centers for Disease Control and Prevention (CDC) classification, length of survival, and cause of death. Considering HIV risk factors, particular attention was paid to drug addiction (duration, route of administration, and type of drugs), especially seeking a history of injection of solutions derived from pills or tablets intended for oral use. At

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the time of diagnosis of PH, laboratory studies included routine blood tests, serum antibodies to HIV, HIV 

P2 anti-
genemia, and blood CD4+ T-lymphocyte count.

For the overall population, a complete clinical evaluation was conducted, including onset of symptoms, degree of dis-
ability assessed by the New York Heart Association (NYHA) functional classification, syncope or near syncope, chest pain, and right-side heart failure. Attention was also paid to the following clinical conditions commonly recognized to be asso-
ciated with PPH13: isolated Raynaud’s phenomenon, migraine, a positive family history of PPH, and significant serum anti-
clear antibody (ANA) titers (>1/80).

**Hemodynamic Study**

For each patient, all vasodilator or inotropic drugs previ-
ously administered were withdrawn at least 36 to 48 hours 

before catheterization. Central venous access was achieved 
through an antecubital vein (in most patients) or through an 
internal jugular vein. Right-side heart catheterization was 
performed under fluoroscopic guidance with a 7F triple-lumen 
flow-directed thermocatheter catheter (Swan Ganz, Baxter 
Edwards) with the tip positioned in a branch of a lower 
pulmonary artery. All parameters were then obtained in 
an intensive care unit in the patients supine, at rest, and breathing 
room air. Heart rate was monitored continuously. Systemic 
arterial pressure was measured intermittently by an external 
automated blood pressure cuff (Dinamap, Critikon).

Cardiac output (CO) was measured by the thermocatheter 
technique (frozen saline) and calculated as the mean value of 
at least three consecutive injections. Cardiac index (CI) was 
calculated as CO per squared meter of body surface area. 
Because the pulmonary artery wedge pressure could not be 
consistently recorded during the entire hemodynamic study in 
many patients, the total pulmonary vascular resistance (TPR) 
(instead of the pulmonary arteriolar resistance) was calculated 
as mean pulmonary artery pressure divided by CI.

A mean of three sets of baseline hemodynamic measure-
ments was obtained in each patient to appreciate the individ-
ual spontaneous hemodynamic variability. Then, a short-term 
vasodilator trial with epoprostenol (prostacyclin, Flolan, Well-
come) was performed in most patients. Epoprostenol is con-
sidered to be a sensitive screening agent in detecting the 
capacity to acutely vasodilate the pulmonary vasculature in 
patients with PPH.14,15 Epoprostenol was infused continuously 
in a peripheral vein with the dosage increased at increments of 
2.5 ng · kg⁻¹ · min⁻¹ every 10 minutes from 2.5 ng · kg⁻¹ · min⁻¹ 
to a maximal dosage of 10 ng · kg⁻¹ · min⁻¹. The infusion was 
stopped immediately if systemic blood pressure decreased by 
more than 30% or if the heart rate increased by more than 
50% compared with control values and/or if side effects 
precluded an increase in dosage. A significant individual 
vasodilator response was defined as a decrease in TPR of 
>20% compared with mean TPR baseline value. Subse-
quent, but only in significant responders to prostacyclin, 
other drugs (isoproterenol, nitroglycerin, phenolamine, dilitia-
zem, and hydralazine) were tested intravenously to select the 
optimal agent that can be taken orally to maintain pulmonary 
vasodilation chronically in combination with oral anticoagu-
lant therapy. Only the results of the short-term testing with 
epoprostenol will be discussed further.

**Statistical Analysis**

Values are given as mean±SD. Comparisons of selected 
variables were made using statistical methods including the 
Student’s t test and χ² test, where appropriate. Because the 
sample size of the HIV-infected group was small, all continu-
ous variables were retested with the nonparametric method of 
the Wilcoxon rank-sum test to confirm differences that were 
considered to be significant. Survival was estimated from first 
catheterization in our hospital until the end of the follow-up 
period (date of most recent information on the patient or 
death) by the Kaplan-Meier product-limit method. Lung or 
heart-lung transplant recipients in the PPH patient group were 
excluded from survival analysis. Differences in mean survival 
time were tested by the log-rank test. A value of P<.05 was 
taken as statistically significant.

**Results**

Between April 1987 and August 1992, 20 HIV-in-
fected patients with PH and 93 non–HIV-infected 
patients with PPH have been referred for diagnostic and 
therapeutic considerations.

Demographic, clinical, and biological characteristics of 
HIV-infected patients are presented in Table 1. Patients are classified according to the date of diagnosis of 
PH. There were 11 women and 9 men with a mean age 
at the time of diagnosis of 32±5 years (range, 24 to 46 years). In 5 patients, the diagnosis of PH led to the 
 discovery of HIV infection. In the 15 other patients, the 
time mean between the diagnosis of HIV infection and 
PH was 5±2 years (range, 1 to 9 years). At the time of 
referral, all HIV-infected patients were free of concom-
tant evolute pulmonary infection or Kaposi’s sar-
coma. Twelve of the 20 HIV-infected patients had a history of intravenous drug addiction as a risk factor for 
HIV infection. All 12 patients had a mean duration of 
heroin addiction of 7±4 years. At the time of diagnosis 
of PH, 4 were still active heroin abusers, 8 had stopped 
2 to 7 years previously, and all denied having injected 
particles from pills or tablets intended for oral use. 
According to CDC classification, HIV status was group 
II for 7 patients; group III, 6 patients; and group IV, 7 
patients. In 12 of 20 patients, blood CD4+ T-lympho-
cyte count was <200 cells/mm³.

The main clinical characteristics of patients are re-
ported in Table 2. Only the symptoms considered as 
severity indexes of PPH are indicated. HIV-infected 
patients significantly differed from patients with PPH 
only in age (32±5 versus 42±13 years, respectively; 
P<.05) and degree of disability (NYHA class III or IV, 
50% versus 75%, respectively; P<.01). Although the 
time between onset of symptoms and diagnosis of PH 
appeared to be twice as short in HIV-infected than in 
non–HIV-infected patients, this did not reach the level 
of statistical significance. Patients also did not differ in 
terms of clinical conditions known to be associated with 
PPH.

Baseline hemodynamic findings are presented in Ta-
ble 3. Although HIV-infected patients with PH had 
markedly elevated TPR levels, their level of PH was 
significantly lower than in patients with PPH (P<.01). 
Hemodynamic changes observed during short-term 
infusion of prostacyclin in the 19 HIV-infected and 86 
non–HIV-infected patients tested are presented in Ta-
ble 4. The proportion of responders to prostacyclin was 
equal in the two groups, and the level of acute pulmo-
nary vasodilatation achieved with prostacyclin (percent 
fall in TPR) in HIV-infected and non–HIV-infected 
patients was similar and unrelated to the level of control 
TPR.

Follow-up of HIV-infected patients from the diagno-
sis of PH to September 1992 showed that 9 patients 
were alive (range, 2 to 43 months) and 11 had died. One 
committed suicide 16 months after the diagnosis of PH, 
and the 10 other patients had died 8±9 months after 
diagnosis (range, 1 to 33 months). The cause of death in
8 of 10 patients could be directly related to PH (right-side heart failure, sudden death), and in the 2 remaining patients, the cause of death was acute sepsis. Twenty-two lung transplant recipients were excluded from the PPH group for survival analysis. Overall survival was poor and not significantly different between the 20 HIV-infected patients with PH and the 71 patients with PPH, with a 46% and 53% survival rate, respectively, at 2 years (Fig 1).

Lung pathological specimens were obtained from 3 HIV-infected patients (patients 4, 15, and 20). Examinations showed features of pulmonary arteriopathy with concentric laminar intimal fibrosis, medial hypertrophy, and numerous plexiform lesions in 2 patients (15 and 20; Fig 2). These findings resembled the pathological changes seen in lung tissue obtained from 27 patients with PPH except for the 1 patient who had pulmonary veno-occlusive disease. In addition, no foreign body granulomas were found in the lungs of HIV-infected patients 4 and 20, who had a long-term history of intravenous drug addiction.

**Discussion**

Since 1987, 33 patients with PH associated with HIV infection have been reported in the literature. We report 20 additional cases, for a total of 53 patients. The evidence for a cause-and-effect relation between these two diseases would require a controlled epidemiological study, which is not presently available. However, in a recent retrospective study from a cohort of 1200 HIV-infected patients, the incidence of PH was estimated to be as high as 0.5%, a rate that is higher than the

**Table 1. Characteristics and Outcome of Patients With Pulmonary Hypertension and HIV Infection**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y/ Sex</th>
<th>Risk Factors</th>
<th>Date of HIV Positivity</th>
<th>CDC Stage</th>
<th>CD4+ (cells/mm³)</th>
<th>Date of PH Diagnosis</th>
<th>Outcome to September 1992</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>36/F</td>
<td>Heterosexual</td>
<td>1987</td>
<td>III</td>
<td>350</td>
<td>April 1987</td>
<td>Died January 1990 (RHF)</td>
</tr>
<tr>
<td>2</td>
<td>28/F</td>
<td>Intravenous heroin abuser</td>
<td>1989</td>
<td>II</td>
<td>174</td>
<td>February 1989</td>
<td>Alive</td>
</tr>
<tr>
<td>4</td>
<td>29/F</td>
<td>Intravenous heroin abuser</td>
<td>1986</td>
<td>III</td>
<td>160</td>
<td>January 1990</td>
<td>Died March 1990 (sepsis)</td>
</tr>
<tr>
<td>5</td>
<td>32/M</td>
<td>Intravenous heroin abuser</td>
<td>1985</td>
<td>IV C₃⁺</td>
<td>222</td>
<td>February 1990</td>
<td>Died March 1990 (RHF)</td>
</tr>
<tr>
<td>6</td>
<td>34/F</td>
<td>Transfusion (anemia)</td>
<td>1990</td>
<td>III</td>
<td>120</td>
<td>October 1990</td>
<td>Alive</td>
</tr>
<tr>
<td>7</td>
<td>34/F</td>
<td>Intravenous heroin abuser</td>
<td>1986</td>
<td>III</td>
<td>340</td>
<td>October 1990</td>
<td>Died December 1990 (sd)</td>
</tr>
<tr>
<td>8</td>
<td>34/M</td>
<td>Homosexual</td>
<td>1987</td>
<td>IV C₂⁺</td>
<td>150</td>
<td>November 1990</td>
<td>Died August 1991 (sepsis)</td>
</tr>
<tr>
<td>9</td>
<td>31/M</td>
<td>Intravenous heroin abuser</td>
<td>1989</td>
<td>IV C₁⁺</td>
<td>6</td>
<td>November 1990</td>
<td>Died December 1990 (RHF)</td>
</tr>
<tr>
<td>10</td>
<td>30/F</td>
<td>Intravenous heroin abuser</td>
<td>1986</td>
<td>II</td>
<td>380</td>
<td>February 1991</td>
<td>Alive</td>
</tr>
<tr>
<td>11</td>
<td>31/M</td>
<td>Intravenous heroin abuser</td>
<td>1985</td>
<td>III</td>
<td>85</td>
<td>February 1991</td>
<td>Died June 1992 (suicide)</td>
</tr>
<tr>
<td>12</td>
<td>31/M</td>
<td>Intravenous heroin abuser</td>
<td>1985</td>
<td>IV C₂⁺</td>
<td>20</td>
<td>May 1991</td>
<td>Died December 1991 (RHF)</td>
</tr>
<tr>
<td>13</td>
<td>24/M</td>
<td>Transfusion (anemia)</td>
<td>1983</td>
<td>II</td>
<td>0</td>
<td>September 1991</td>
<td>Alive</td>
</tr>
<tr>
<td>14</td>
<td>43/M</td>
<td>Transfusion (hemophilia)</td>
<td>1983</td>
<td>II</td>
<td>110</td>
<td>January 1992</td>
<td>Alive</td>
</tr>
<tr>
<td>16</td>
<td>31/F</td>
<td>Intravenous heroin abuser</td>
<td>1987</td>
<td>IV C₁⁺</td>
<td>105</td>
<td>January 1992</td>
<td>Alive</td>
</tr>
<tr>
<td>18</td>
<td>34/M</td>
<td>Homosexual</td>
<td>1985</td>
<td>IV C₁⁺</td>
<td>7</td>
<td>April 1992</td>
<td>Alive</td>
</tr>
<tr>
<td>19</td>
<td>30/F</td>
<td>Heterosexual</td>
<td>1985</td>
<td>II</td>
<td>350</td>
<td>July 1992</td>
<td>Alive</td>
</tr>
</tbody>
</table>

HIV indicates human immunodeficiency virus; CDC, Centers for Disease Control and Prevention; PH, pulmonary hypertension; RHF, right-side heart failure; and sd, sudden death.

*Group IV CDC stage; patient 5, cryptococcal meningitis; patient 8, oral thrush; patient 9, brain toxoplasmosis; patient 12, oral thrush; patient 15, pneumocystis carinii pneumonia; patient 16, cytomegalovirus infection; and patient 18, disseminated Mycobacterium avium-intracellular infection.
estimated incidence (0.02%) of PPH in the general population. 16

Some disease states are known to be associated with PPH development, such as isolated Raynaud’s phenomenon, migraine, collagen disease without overt symptoms or signs, and a positive family history of PPH. 13,17 The proportion of such clinical conditions was similar in our two patient populations, suggesting that PH had arisen in equally “susceptible” patients. A female-to-male predominance is well known in PPH 13,16,18 and was also found in our HIV-infected patients, even though evident sexual bias usually leads to a male predominance in the general distribution of HIV risk factors (eg, hemophilia, homosexuals).

Hemodynamic changes observed during a short-term vasodilator trial with epoprostenol were quite similar in both groups, providing further argument that they may suffer from the same pulmonary vascular disease. This can also be supported by the close pathological changes observed in the pulmonary vasculature of our HIV-infected and non-HIV-infected patients, in keeping with findings previously reported in the literature. 3,6,8

The first major report of PH associated with HIV infection concerned only patients with classic hemophilia, raising questions about a cause-and-effect relation between PH and the use of factor VIII concentrates of low purity. 2 Since then, it has been shown that PH may develop in HIV-infected patients regardless of the degree of immunosuppression and/or risk factor. 1-12 Patients with a history of chronic intravenous drug abuse may develop PH 19-22 and are classically excluded from PPH. 13 Pulmonary angiography is the main

| TABLE 2. Clinical Characteristics of Patients at the Time of Diagnosis of Pulmonary Hypertension |
|----------------------------------|----------------------------------|----------------------------------|
| Patient Characteristic | PH-HIV (n=20) | P | PPH (n=93) |
| Age, y | 32±5* | <.05 | 42±13* |
| Sex | | | |
| Women | 11 | 65 | |
| Men | 9 | 28 | |
| Ratio of women to men | 1.2:1 | NS | 2.3:1 |
| Associated conditions | | | |
| Raynaud’s phenomenon, n (%) | 5 (25) | NS | 20 (21) |
| ANA tilters >1/80, n (%) | 2 (17) | NS | 9 (11) |
| Migraines, n (%) | 2 (10) | NS | 14 (15) |
| Family history of PPH, n (%) | 0 (0) | NS | 3 (3) |
| Symptoms | | | |
| Duration, mo | 14±15* | NS | 30±32* |
| NYHA functional class | | | |
| I or II, n (%) | 10 (50) | <.05 | 23 (25) |
| II or IV, n (%) | 10 (50) | <.05 | 70 (75) |
| Syncope, n (%) | 6 (30) | NS | 46 (49) |
| Chest pain, n (%) | 4 (20) | NS | 31 (33) |
| Hemoptyisia, n (%) | 1 (5) | NS | 10 (11) |
| Right-side heart failure, n (%) | 4 (20) | NS | 43 (46) |

PH indicates pulmonary hypertension; HIV, human immunodeficiency virus; PPH, primary pulmonary hypertension; ANA, antinuclear antibodies (percentage is given for the number of patients tested); and NYHA, New York Heart Association. *Values are mean±SD.

| TABLE 3. Baseline Hemodynamic Findings at First Catheterization |
|----------------------------------|----------------------------------|----------------------------------|
| Patients (n) | RAP, mm Hg | PAP, mm Hg | PWP, mm Hg | CI, L·min⁻¹·m⁻² | TPR, mm Hg·L⁻¹·min⁻¹·m⁻² | SAP, mm Hg | HR, bpm |
| PH-HIV (20) | 8±4 | 50±11 | 7±2 | 2.6±0.5 | 20±7 | 87±10 | 91±13 |
| NS | * | NS | * | * | NS | NS |
| PPH (93) | 9±4 | 62±15 | 7±3 | 2.2±0.6 | 31±11 | 93±12 | 87±13 |

RAP indicates mean right atrial pressure; PAP, mean pulmonary arterial pressure; PWP, mean pulmonary wedge pressure; CI, cardiac index; TPR, total pulmonary vascular resistance index; SAP, mean systemic arterial pressure; HR, heart rate; bpm, beats per minute; PH, pulmonary hypertension; HIV, human immunodeficiency virus; and PPH, primary pulmonary hypertension. Values are given as mean±SD.

*P<.01.

Hemodynamic parameters were recorded in patients at rest, breathing room air, and with no supportive drug.
pathological finding in such a condition\(^{19}\) and is believed to be due to foreign particle pulmonary emboli following injections of solutions derived from tablets or pills containing insoluble microcrystals.\(^{19}\) Ordinary heroin does not contain enough crystalline debris to induce extensive pulmonary angiothrombosis,\(^{19}\) and because fewer than 5% of drug addicts frequently inject tablet derivatives,\(^{20,22}\) PH is supposed to be rare among them. This was confirmed in the late 1970s before HIV infection was epidemic.\(^{23}\) Repeated injections of foreign particles were denied by all of our drug addicts. Moreover, lung pathological specimens obtained from 4 HIV-infected drug abusers with PH reported in the literature\(^{3,6,8}\) did not show any foreign body granulomas (like in our patients 4 and 20) (Fig 2).

HIV-infected and non-HIV-infected patients differed significantly in age, functional class, and initial hemodynamic severity (Tables 2 and 3). This may be explained, as well as the shorter time interval between onset of symptoms and diagnosis of PH, by the close medical attention usually devoted to HIV-infected patients who may account for an earlier diagnosis, although both conditions were simultaneously diagnosed in 5 of the 20 patients reported here.

Survival in PPH is known to be poor\(^{24}\) and has been reported to be limited as well in HIV-infected patients with PH.\(^{1,3,5-9}\) Survival in our HIV-infected patients was not significantly different than that in patients with PPH who had been evaluated and treated in the same manner. However, considering that HIV-infected patients had a shorter average duration of symptoms attributable to PH before initial catheterization, a faster disease evolution in HIV-infected patients cannot be ruled out.

The pathogenesis of the pulmonary arterial disease associated with HIV infection is unknown, as it is in PPH.\(^{17}\) The hypothesis of a direct impact of the virus on the pulmonary vascular smooth muscle and/or endothelial cells has not been demonstrated.\(^{12}\) An indirect role of HIV in the production of growth factors leading to abnormal endothelial and smooth muscle cell proliferation has also been discussed.\(^{7}\)

**TABLE 4.** Hemodynamic Changes Observed During Short-term Infusion of Epoprostenol

<table>
<thead>
<tr>
<th>Patients Tested (n)</th>
<th>TPR Baseline, mm Hg (\cdot) L(^{-1}) (\cdot) min(^{-1}) (\cdot) m(^{-2})</th>
<th>TPR During Epoprostenol, mm Hg (\cdot) L(^{-1}) (\cdot) min(^{-1}) (\cdot) m(^{-2})</th>
<th>Decrease in TPR, %</th>
<th>Responders to Epoprostenol, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH-HIV (19)</td>
<td>19±5 *</td>
<td>15±5</td>
<td>20±13</td>
<td>11 (58)</td>
</tr>
<tr>
<td>PPH (86)</td>
<td>30±11 *</td>
<td>22±10</td>
<td>24±18</td>
<td>49 (57)</td>
</tr>
</tbody>
</table>

TPR indicates total pulmonary vascular resistance index; PH, pulmonary hypertension; HIV, human immunodeficiency virus; and PPH, primary pulmonary hypertension. Values are given as mean±SD.

For a definition of "responders," see "Methods."

*\(P<.01\).
In conclusion, the clinical, hemodynamic, and pathological similarities between HIV-infected patients with PH and non–HIV-infected patients with PPH suggest that HIV infection must be regarded as another common risk factor for PPH development. Subsequently, it seems reasonable to routinely perform HIV tests in patients presenting with PPH. Finally, this likely association obviously raises the question of a viral involvement in the pathogenesis of PPH in general.

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