CASE REPORTS

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Nitric Oxide Reduces Pulmonary Hypertension during Hepatic Transplantation

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AS orthotopic liver transplantation is used more often as accepted therapy for end-stage liver disease (ESLD), pulmonary hypertension as an associated complication will occur with increasing frequency. The optimal therapy has not been determined, and such patients continue to have a high rate of mortality and morbidity.[1–4] An emerging and promising pulmonary vasodilator is the inhaled gas nitric oxide.[5] We report on the use of nitric oxide in the treatment of pulmonary hypertension during liver transplantation.

Case Report

A 24-yr-old woman was referred for orthotopic liver transplantation. She had ESLD and portal hypertension due to cryptogenic cirrhosis. Approximately 1 yr before transplantation, the patient required repeated sclerosis of esophageal varices to control gastrointestinal bleeding. Because sclerosis failed to control the bleeding, she underwent splenorenal shunting. During surgery, pulmonary artery pressures ranged from 76 to 96 mmHg systolic (normal 15–30) and 45 to 62 mmHg diastolic (normal 0–12). Otherwise the patient was hemodynamically stable. Because of her increased pulmonary artery pressures, elective cardiac catheterization was performed postoperatively (table 1).

In the months following her splenorenal shunt, progressive hepatic failure developed, manifested by fatigue, ascites, and hepatic encephalopathy, which was responsive to medical management. Subsequently she underwent a multidisciplinary assessment for orthotopic liver transplantation.

At the time of evaluation, the patient was alert and ambulatory, though appearing cachectic and chronically ill. She had no symptoms of respiratory or cardiac disease. Her main complaints were of increasing abdominal girth and pruritus. Cardiac examination revealed a visible and palpable impulse associated with a thrill in the left second intercostal space, consistent with expansion of the main pulmonary artery. A III/VI holosystolic murmur was present at the left lower sternal border, which increased on inspiration.

Accompanying laboratory results included a hemoglobin of 9.7 g/dl (normal 13.5–16.7), hematocrit 32% (normal 40–49), platelets 118,000 mm^{-3} (normal 150–400 $\times 10^3$), prothrombin time 13.5 s (normal 9.7–12.1), partial thromboplastin time 36.4 s (normal 23.3–33.8), and antithrombin III level 31% (normal 70–120). The albumin was 3.1 g/dl, total protein 5.9 g/dl, and total bilirubin 1.1 mg/dl. Creatinine was 0.7 mg/dl and blood urea nitrogen 7 mg/dl. The electrolytes were within normal limits except for a bicarbonate of 17 mM/l. Arterial blood gases during room air breathing were: *p*H 7.44, arterial carbon dioxide tension 28 mmHg, and arterial oxygen tension 66 mmHg. Pulmonary function tests were within 20% of predicted except for a decreased diffusing capacity of 56%. Distal pruning of the pulmonary vessels was observed on chest x-ray.

The electrocardiogram showed a vertical axis, early R wave progression, and anterior ST-T wave changes consistent with right ventricular strain. An enlarged right atrium and ventricle with mild tricuspid regurgitation were observed on echocardiogram. Right ventricular ejection fraction was estimated to be 31%, and pulmonary hypertension was stated to be moderate. Results of a multigated acquisition scan indicated right ventricular hypokinesis with an ejection fraction of 30%. Left ventricular size and function were normal. A ventilation-perfusion scan was interpreted as indeterminate for pulmonary emboli, and no emboli were demonstrated by subsequent angiography.

Cardiac catheterization was repeated during transplant assessment. There were no significant changes in pulmonary or intracardiac pressures from the previous catheterization (table 1). To determine pulmonary vasoreactivity, a prostacyclin challenge was performed starting at 0.5 $ng \cdot kg^{-1} \cdot min^{-1}$. Testing was discontinued at 2.5 $ng \cdot kg^{-1} \cdot min^{-1}$ because of flushing, nausea, and abdominal pain. Despite early termination of testing, a 24% decrease in pulmonary vascular resistance with a 13% decrease in systemic vascular resistance was observed. Increasing oxygen concentrations also were administered to assess a vasoreactive response, but none was observed. Because of the stability of her pulmonary artery pressures and selective vasodilator response to prostacyclin, the patient was considered an acceptable but high-risk transplant candidate.

Five months after transplant evaluation, a donor organ became available. Cardiac catheterization was performed immediately before surgery. Significant increases in pulmonary artery and right ventricular pressures were noted compared to prior catheterizations (table 1). Cardiac output remained stable.

After insertion of an oximetric pulmonary artery catheter and right and left 20-G radial arterial catheters, anesthesia was induced with

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Table 1. Cardiac Catheterization Data

	Months before Transplantation			12 Months after Transplantation
	12	6	0	
Event	Splenorenal shunt	Transplant assessment	Pretransplant	Follow-up
Blood pressure (mean)(mmHg)	124/64 (84)	101/61 (74)	100/65 (76)	123/84 (97)
Heart rate (beats/min)	77	83	90	75
Cardiac output (L/min)	7.5	6.8	7.1	6.1
Right atrial pressure (mmHg)	3	5	6	6
Right ventricular pressure (mmHg)	60/5	67/1	91/11	66/1
Pulmonary artery pressure (mean) (mmHg)	60/30 (940)	63/26 (43)	86/35 (56)	66/29 (45)
Pulmonary wedge pressure (mmHg)	4	6	4	10
Systemic vascular resistance (dyne.s.cm^{-5})	864	811	788	1192
Pulmonary vascular resistance (dyne.s.cm^{-5})	384	435	585	459

250 mg sodium thiopental. After tracheal intubation facilitated by 100 mg succinylcholine, relaxation was supplemented with 10 mg doxacurium and anesthesia maintained with 2 mg fentanyl and 0.5% isoflurane. Additional intravenous access was achieved through placement of 9.0-Fr catheters in the right antecubital and left internal jugular veins, which were connected to a rapid infusion system. A transesophageal echo probe was placed to view the short axis and right ventricular volume. Right ventricular function as assessed by transesophageal echo appeared unchanged from pretransplantation evaluation. Initial hemodynamic measurements were recorded (table 2).

A 20-min trial of nitric oxide at 40 ppm was started before surgical incision. Chemiluminescence analysis was used to monitor the administered dose. Pulmonary artery pressures decreased in response to inhaled nitric oxide and increased after its discontinuation (table 2). Pulmonary artery wedge pressures were not obtained because of concern for possible vascular rupture. Surgical dissection was difficult because of adhesions. Five units of packed erythrocytes and 5 U of fresh frozen plasma were administered to maintain stability during stage 1, the duration of that stage being 4 h, 12 min. Venovenous bypass was not instituted. After clamping of the supra- and infrahepatic vena cavae, she required 2 U of packed erythrocytes, 3 U of fresh frozen plasma, and 400 μg Neo-Synephrine in divided doses to maintain hemodynamic stability. Pulmonary artery pressures ranged at about 55–60/25–30 mmHg.

The grafted liver was received from a 36-yr-old donor. The liver had been perfused with 450 ml of University of Wisconsin solution. Cold ischemic time was approximately 7 h, warm ischemic time 53 min. The duration of stage 2 was 1 h, 40 min.

Forty minutes following caval cross-clamping, nitric oxide was restarted at 40 ppm. At the conclusion of stage 2, the portal venous and suprahepatic caval clamps were released slowly to limit the rate of return of blood to the pulmonary circulation. This was well tolerated, and subsequently the infrahepatic vena cava was slowly unclamped. At this stage and during nitric oxide administration, pulmonary artery pressures increased precipitously whereas systemic

Table 2. Intraoperative Hemodynamics

	60 min Postinduction	90 min Postinduction	30 min into Stage 2	50 min into Stage 2	Reperfusion	60 min Postanhepatic	90 min Postanhepatic
Intervention with nitric oxide (ppm)	0	40	0	40	40	40	0
Operative stage	Preanhepatic	Preanhepatic	Anhepatic	Anhepatic	Reperfusion	Reperfusion	Reperfusion
Blood pressure (mean) (mmHg)	120/65 (80)	130/65 (86)	130/70 (90)	130/65 (86)	78/44 (55)	110/50 (70)	120/70
Heart rate (beats/min)	90	90	110	120	120	110	100
Cardiac output (L/min)	7.6	8.8	7.5	8.5	3.2	7.2	8.6
Central venous pressure (mmHg)	5	5	5	6	9	7	6
Pulmonary artery pressure (mean) (mmHg)	87/27	57/24	68/28 (41)	60/24 (36)	105/87 (93)	46/22	72/25 (46)
Mixed venous hemoglobin oxygen saturation (%)	88	91	88	91	74	89	93

blood pressure decreased (table 2). The transesophageal echo revealed marked global right ventricular hypokinesis. What were thought to be air emboli were observed only fleetingly. A total of 300 μg epinephrine was given with improvement in ventricular contractility, although the systemic blood pressure remained low. Systemic pressure gradually increased as phenylephrine was administered in divided doses to a total of 400 μg. Pulmonary artery pressures gradually decreased (table 2). Continued improvement in right ventricular function was observed by transesophageal echo.

One hour after reperfusion, with nitric oxide administration continuing, the patient was hemodynamically stable; pulmonary artery pressures were 46/22 mmHg. The liver appeared to be functioning well and producing bile. The nitric oxide was discontinued to determine pulmonary vascular response. The pulmonary artery pressures increased to 72/25 mmHg with a slight decrease in cardiac output (table 2). As cardiac output and mixed venous oxygen saturations remained within acceptable limits, nitric oxide therapy was not reinstituted. Stage 3 duration was 5 h, 29 min. Fluid replacement during the entire procedure included 4,500 ml of balanced crystalloid, 19 U of packed erythrocytes, 24 U of fresh frozen plasma, 40 U of platelets, 10 U of cryoprecipitate, and 2.75 l 5% albumin. Estimated blood loss was 8 l, and urine output was approximately 3 l.

The patient was transferred to the intensive care unit, where pulmonary artery pressures and cardiac output remained about 72/33 mmHg and 8.34 l/min, respectively. She remained hemodynamically stable and was separated from ventilatory support 26 h postoperatively. The patient was discharged from the intensive care unit by 72 h. The duration of hospitalization was 19 days. Subsequently, she has experienced no episodes of rejection. One year after transplantation, she is mildly dyspneic on exertion, has returned to work, and is rated as New York Heart Association class II. An echocardiogram performed 12 months after the transplant, estimated her pulmonary hypertension to be moderate. Cardiac and portal catheterizations demonstrated moderate pulmonary hypertension (table 1), inferior vena cava pressure of 8 mmHg, and hepatic vein pressure of 7 mmHg. She is receiving nifedipine for her pulmonary hypertension.

Discussion

The association between portal and pulmonary hypertension has been well documented.[6–11] As symptoms may be nonspecific or attributed to the ESLD,[12] evidence of pulmonary hypertension must be sought carefully. The chest radiograph and electrocardiogram often have findings suggestive of pulmonary hypertension.[13,14] Echocardiography is useful for detecting pulmonary hypertension and evaluating ventricular function and is an excellent screening tool in the preoperative assessment of these patients.[15,16] Mean survival after diagnosis of pulmonary hypertension associated with ESLD is 15 months with a 6-month mortality of 50%.[17]

When transplantation is considered, patient selection is particularly important.[4] There is insufficient case history and no established guidelines for deciding which transplantation candidates need further invasive evaluation once pulmonary hypertension is detected. However, in the National Institutes of Health Registry on Primary Pulmonary Hypertension, an algorithm has been developed that may guide evaluation.[18] Once an echocardiogram has established pulmonary hypertension, and valvular disease and shunts have been eliminated as a likely etiology, a ventilation-perfusion scan should be performed to evaluate for embolic disease. Patients with more than one segmental defect should receive pulmonary angiography. If pulmonary emboli is eliminated as an etiology, the patient should undergo cardiac catheterization.

If the patient has moderate or severe pulmonary hypertension, vasodilator potential must be assessed.[3] Optimally, reductions in pulmonary artery pressures or increases in cardiac output are observed with preservation of systemic pressures. Failure to achieve reductions in pulmonary artery pressures with vasodilators suggests the vascular disease has become fixed and is a poor prognostic sign.[3,4] In contrast, patients with ESLD and mild to moderate pulmonary hypertension (pulmonary vascular resistance within 120–275 dyne·s·cm^{-5}) have survived orthotopic liver transplantation without difficulty.[19]

Short-acting intravenous vasodilators, such as prostacyclin, prostaglandin E1, adenosine, sodium nitroprusside, and nitroglycerin, have been administered to treat pulmonary hypertension but may produce unacceptable systemic hypotension.[20,21] Nitric oxide, or endothelium-derived relaxing factor,[22–24] offers a novel therapy for pulmonary hypertension associated with many conditions.[25–34] Nitric oxide, when inhaled, crosses the alveolar membrane and produces smooth muscle dilation. However, when it crosses into the circulation, nitric oxide is bound to hemoglobin with an avidity 1,500 times greater than that of carbon monoxide, preventing its action in the systemic circulation.[35,36]

The ideal dose of nitric oxide has yet to be determined. Current trends suggest doses as small as 2 ppm may be effective in producing pulmonary vasodilation and improving oxygenation.[37–39] What constitutes a satisfactory dose may vary depending on the chronicity of the condition, the particular disease process, and the desired objectives (oxygenation *vs.* vasodilation). Optimally, within any individual, dose-response evaluation should be undertaken to determine the minimum effective dose.

During transplantation, numerous factors may exacerbate pulmonary hypertension. Pulmonary artery

pressures may increase precipitously secondary to acidosis and fluid shifts.[40,41] Reperfusion of the donor liver is a time of increased risk because vasoactive substances returning to the heart may result in systemic vasodilation and myocardial depression.[42,43] Right ventricular decompensation may be poorly tolerated when there is preexisting ventricular dysfunction and is a major cause of death in pulmonary hypertension.[44] Preservation of right ventricular function may be the determining factor in survival of these patients.[1] Reduction in pulmonary pressures with nitric oxide may prevent right ventricular deterioration.

In summary, patients with end-stage liver disease have an incidence of pulmonary hypertension higher than the normal population and may pose difficult management problems during liver transplantation. Right heart catheterization and vasodilator testing will establish the degree of pulmonary hypertension and responsiveness to therapy and may influence decisions concerning transplant candidacy and intraoperative intervention. The significant hemodynamic changes occurring during orthotopic liver transplantation may be poorly tolerated by such individuals. We have demonstrated that inhaled nitric oxide, through its selective pulmonary vasodilation, is an effective agent for treating pulmonary hypertension in patients undergoing liver transplantation.

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Paraplegia in a Patient with an Intrathecal Catheter and a Spinal Cord Stimulator

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This article is accompanied by a Highlight. Please see this issue of ANESTHESIOLOGY, page 27A.

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NEW technical devices are adopted frequently and enthusiastically, while possible serious risks are overlooked. This has happened with intrathecal catheters connected to infusion pumps[1] and spinal cord stimulators.[2]

Herein, we describe a sudden spinal cord derangement that occurred in a patient in whom both an intrathecal catheter infusing morphine by an implanted computerized pump and a spinal cord stimulator had been inserted.

Case Report

A 73-yr-old man described an acute onset of motor and sensory loss in January 1991 but still suffered from thoracic back pain,