

ation and pulmonary hypertension in patients with adult respiratory distress syndrome. *Eur J Clin Invest* 1993; 23: 499-502

Pregnancy and Primary Pulmonary Hypertension*

Successful Outcome With Epoprostenol Therapy

Rosalyn Stewart, MD; Divina Tuazon, MD; Gayle Olson, MD; and Alexander G. Duarte, MD

Primary pulmonary hypertension (PPH) associated with pregnancy carries a high maternal mortality rate. Short-term epoprostenol infusion has been demonstrated to improve the hemodynamic profile in patients with PPH. We report a successful maternal-fetal outcome with epoprostenol therapy during pregnancy, cesarean section, and postpartum in a patient with PPH. Epoprostenol therapy did not produce any physical or developmental abnormalities in the fetus. A favorable maternal-fetal outcome may occur with a multidisciplinary approach.

(*CHEST* 2001; 119:973-975)

Key words: cesarean section; epoprostenol; pulmonary hypertension; pregnancy

Abbreviations: PAC = pulmonary artery catheter; PPH = primary pulmonary hypertension

Primary pulmonary hypertension (PPH) is a rare, progressive condition aggravated by the physiologic changes occurring during pregnancy and surgery. The maternal mortality rate associated with pregnancy and pulmonary hypertension ranges from 30 to 50%.^{1,2} The administration of IV epoprostenol has been well-demonstrated to improve hemodynamics in nonpregnant patients with PPH.³ We report a successful maternal-fetal outcome in a pregnant woman in whom PPH was diagnosed who was treated with IV epoprostenol before, during, and after undergoing cesarean section.

CASE REPORT

A 35-year-old, gravida (G2,P0) patient with a history of hypothyroidism presented at 26 weeks' gestation with progressive exertional dyspnea and fatigue of several weeks duration. She also reported several recent syncopal episodes. Her first pregnancy was uncomplicated, and she denied prior cardiopulmonary disease, illicit drug use, or ingestion of anorexigens. On physical

examination, her vital signs were as follows: BP, 90/60 mm Hg; heart rate, 105 beats/min; respiratory rate, 20 breaths/min; and oxygen saturation as measured by pulse oximetry, 92%. Jugular venous distension was present. Cardiac auscultation revealed a loud S₂ and a grade 3/6 systolic murmur over the left lower sternal border that was accentuated on inspiration. Lung fields were clear to auscultation bilaterally. Extremities were without clubbing, and 1+ edema was present. An ECG was interpreted as representing normal sinus rhythm. A chest radiograph was unremarkable for parenchymal infiltrates, and a ventilation-perfusion scan was interpreted as revealing a low probability for a pulmonary embolism. Arterial blood gas measurements revealed the following: pH, 7.45; PCO₂, 29 mm Hg; PO₂, 79 mm Hg; and bicarbonate level, 20 mEq/L. An echocardiogram displayed a dilated right ventricle, paradoxical septal wall motion, and normal left ventricular wall motion.

The patient was admitted to labor and delivery and was prescribed bed rest, oxygen, diuretics, and heparin. Fetal heart tones were noted at 150 beats/min, and IM corticosteroids were administered to accelerate fetal lung development. Despite this therapy, the patient continued to report progressive dyspnea, and at 32 weeks' gestation the placement of a pulmonary artery catheter (PAC) demonstrated moderate pulmonary hypertension (Table 1). IV epoprostenol therapy was initiated at 4 ng/kg/min, producing an improvement in the hemodynamic profile (Table 1). At 36 weeks' gestation, while receiving IV epoprostenol, the premature rupture of membranes occurred followed by active labor. However, the progression of labor was inadequate, and a cesarean section was scheduled. Preoperatively, a PAC was placed and epidural anesthesia was administered. Subsequently, the cardiac output declined from 7.4 to 4.1 L/min and the epoprostenol infusion was increased to 10 ng/kg/min. The patient remained hemodynamically stable throughout the cesarean section and delivered a healthy male infant weighing 7 lbs with Apgar scores of 5 and 9, respectively, at 1 and 5 min. A bilateral tubal ligation was performed with patient consent. Following extubation, the PAC was maintained for 48 h to assist with IV fluid administration, and the hemodynamic profile remained stable with the patient receiving 10 ng/kg/min epoprostenol. On postoperative day 2, heparin therapy was resumed.

Three weeks later, the patient underwent a vasodilator trial with calcium-channel blockers but did not have a favorable response, hence, she was continued on epoprostenol therapy. Presently, she has resumed an active lifestyle as a housewife and mother. Furthermore, her 2-year-old son is in good health without any developmental delays.

DISCUSSION

An early case series reported a 50% mortality rate associated with pregnancy and PPH.¹ A more recent account noted a 30% mortality rate² and partly attributed the decline in the mortality rate to earlier recognition, better understanding of the pathophysiology of pulmonary hypertension, along with improvements in medical therapy and critical-care obstetrics. Recognition of the elevated maternal-fetal mortality rate has led physicians to recommend effective contraception and, in the event of a pregnancy, early fetal termination.⁴ The maternal mortality rate is related principally to the increased demands on the cardiopulmonary system during pregnancy. Under normal circumstances, increases in cardiac output in the range of 30 to 50%, blood volume in the range of 40 to 50%, and oxygen consumption of 20% are observed during pregnancy.^{5,6} Other physiologic changes include an increase in cardiac output during labor in patients receiving local

*From the Department of Internal Medicine (Drs. Stewart, Tuazon, and Duarte), Division of Pulmonary and Critical Care Medicine, and the Department of Obstetrics and Gynecology (Dr. Olson), University of Texas Medical Branch, Galveston, TX. Manuscript received May 18, 2000; revision accepted August 28, 2000.

Correspondence to: Alexander G. Duarte, MD, Division of Pulmonary and Critical Care Medicine, John Sealy Annex, 301 University Blvd, Galveston, TX 77555-0561; e-mail: aduarte@utmb.edu

Table 1—Hemodynamic Measurements*

Hospital Course	Dose, ng/kg/min	BP	PAP, mm Hg	PVR, dyne · s · cm ⁻⁵	CO, L/min	CVP, mm Hg	PAOP, mm Hg
Baseline	0	91/49	75/35	660	4.0	13	15
Epoprostenol infusion	4	106/59	63/30	270	7.4	10	16
Cesarean section	10	118/65	63/28	250	7.2	12	17
Postpartum	10	120/73	69/32	350	6.5	9	16

*PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance; CO = cardiac output; CVP = central venous pressure; PAOP = pulmonary artery opening pressure.

anesthesia (pudendal block) and postpartum intravascular volume shifts resulting from blood loss or diuresis. These physiologic events place a great demand on the cardiovascular system, with the greatest incidence of mortality occurring during the first several postoperative days. This is likely related to changes in pulmonary vascular tone due to intravascular volume shifts, hypoxemia, elevated catecholamine levels, or thromboembolism. Identification of the hemodynamic changes has led to the use of anticoagulants, oxygen, and vasodilators in the management of these patients.

Several reports have demonstrated an improvement in hemodynamics and outcome in nonpregnant patients receiving vasodilator and anticoagulation therapy for the treatment of PPH.^{3,7} Subsequently, several reports have described the use of vasodilator therapy during parturition and postpartum with good outcomes.^{8,9} The majority of patients in these series had improvements in hemodynamics similar to our patient, leading one to speculate that a favorable short-term response to vasodilator therapy may be predictive of a favorable maternal outcome. Accordingly, we elected to use epoprostenol and heparin for several weeks prior to the expected date of delivery in order to maximize the benefits of this therapy. Furthermore, this therapy was continued in the postpartum period given the increased incidence of complications following labor and delivery. Another important point concerns the safety of the fetus following exposure to IV epoprostenol. Although there is little information on this subject, this report indicates that during the last trimester of pregnancy epoprostenol therapy does not result in any fetal deformities or growth retardation.

Several factors have been implicated as potential risk factors for maternal death, including mode of delivery, type and technique of anesthesia, and manner of maternal monitoring. A recent case series described successful outcomes in seven women with pulmonary hypertension following vaginal delivery.¹⁰ In contrast, greater morbidity and mortality has been associated with the performance of a cesarean section.² To our knowledge, this is one of a small number of reports noting a successful maternal-fetal outcome following cesarean section in a patient with PPH.^{4,11,12} An explanation for this observation has been offered and may be related to the selection of the anesthetic technique (epidural or general anesthesia), although it is not clear whether one technique is superior in the setting of pulmonary hypertension and parturition.¹¹ Another explanation may lie in the fact that cesarean section is more likely to be performed in patients unable to deliver by the vaginal route who subsequently develop hemody-

amic instability. While some authors have disputed the need for a PAC, we advocate its use intraoperatively and during the postpartum period.⁹ In this report, the epoprostenol infusion was titrated using pulmonary artery pressure and cardiac output measurements obtained with a PAC. An important component in the successful management of these patients involves a multidisciplinary team approach with an obstetrician, pulmonary or cardiology specialist, anesthesiologist, and experienced nursing staff.

In summary, PPH is likely to worsen during labor and delivery, resulting in a high maternal mortality rate. Early recognition and treatment with vasodilator and anticoagulation therapy may reduce the likelihood of complications. Elective cesarean section may be performed with intraoperative vasodilator administration. The IV epoprostenol dose not give rise to physical deformities or fetal growth retardation. A multidisciplinary approach to the management of patients with PPH during pregnancy is of great importance for a successful maternal-fetal outcome.

REFERENCES

- McCaffrey RN, Dunn LJ. Primary pulmonary hypertension in pregnancy. *Obstet Gynecol Surv* 1964; 19:567-591
- Weiss BM, Zemp L, Seifert B, et al. Outcome of pulmonary vascular disease in pregnancy: a systematic overview from 1978 through 1996. *J Am Coll Cardiol* 1998; 31:1650-1657
- McLaughlin VV, Genthner DE, Panella MM, et al. Reduction in pulmonary vascular resistance with long term epoprostenol (prostacyclin) therapy in primary pulmonary hypertension. *N Engl J Med* 1998; 338:273-277
- Elkayam U, Dave R, Bokhari SWH. Primary pulmonary hypertension and pregnancy. In: Elkayam U, Gleicher N, eds. *Cardiac problems in pregnancy*. New York, NY: Wiley-Liss, 1998; 183-190
- Ueland K. Pregnancy and cardiovascular disease. *Med Clin North Am* 1977; 61:17-41
- Weiss BM, Hess OM. Pulmonary vascular disease and pregnancy: current controversies, management strategies, and perspectives. *Eur Heart J* 2000; 21:104-115
- Rich S, Kaufmann E, Levy PS. The effect of high doses of calcium-channel blockers on survival in primary pulmonary hypertension. *N Engl J Med* 1992; 327:76-81
- Noontens M, Rich S. Successful management of labor and delivery in primary pulmonary hypertension. *Am J Cardiol* 1993; 71:1124-1125
- Easterling TR, Ralph DD, Schmucker BC. Pulmonary hypertension in pregnancy: treatment with pulmonary vasodilators. *Obstet Gynecol* 1999; 93:494-498

- 10 Smedstad KG, Cramb R, Morison DH. Pulmonary hypertension and pregnancy: a series of eight cases. *Can J Anaesth* 1994; 41:502–512
- 11 O'Hare R, McLoughlin C, Milligan K, et al. Anesthesia for caesarean section in the presence of severe primary pulmonary hypertension. *Br J Anaesth* 1998; 81:790–792
- 12 Badalian SS, Silverman RK, Aubry RH, et al. Twin pregnancy in a woman on long term epoprostenol therapy for primary pulmonary hypertension. *J Reprod Med* 2000; 45:149–152

Late Mediastinal Shift After Repeated Aspiration of Postpneumonectomy Seroma*

Carey Cullinane, MD; Kevin L. Kovitz, MD, FCCP; and Renee S. Hartz, MD

Development of a postoperative seroma is a frequent complication after muscle-sparing thoracotomy. We describe an unusual case of late mediastinal shift in a patient in whom our original plan to perform a limited muscle-sparing thoracotomy was abandoned. The procedure was converted to a standard posterolateral incision to perform a pneumonectomy for a large central carcinoid tumor with extrabronchial extension. Fluid that accumulated in her pneumonectomy space presumably shifted into the dissected tissues of her chest wall, and was then drained repeatedly by her local physician in the time interval between 2 weeks and 3 months after surgery.

(*CHEST* 2001; 119:975–977)

Key words: bronchial carcinoid; mediastinal shift; postpneumonectomy seroma

Considerable debate surrounds the relative merits of standard posterolateral and muscle-sparing thoracotomy for pulmonary resections. Advocates of the latter contend that the technique allows for more rapid recovery of respiratory function, better arm and shoulder mobility, and a more watertight seal of the chest because incisions in the chest wall are not superimposed. They also claim that the incision is more cosmetic and can be rapidly approximated. As in other procedures, which involve extensive mobilization of muscle and subcutaneous tissue, the development of a postoperative seroma is a relatively common complication.¹ In this report, we describe a patient whose left muscle-sparing thoracotomy was converted to a posterolateral incision, with division of the latissimus dorsi and sparing of the serratus anterior. The patient was returned to the care of her local physician 2 weeks after surgery, after we had performed aspiration of

approximately 200 mL of seroma fluid to relieve a pressure sensation over her incision. When she returned to our medical center 6 months after surgery for follow-up of her carcinoid tumor, chest radiography revealed that the pneumonectomy space was almost empty, and the left heart border was almost at the left chest wall.

CASE REPORT

A 36-year-old woman with an 8-month history of dyspnea was referred to a pulmonologist in our medical center for evaluation. Findings included postobstructive pneumonitis of her left lower lobe and bronchoscopic documentation of a smooth yellow mass completely occluding the orifice of the left lower lobe bronchus. Biopsy revealed typical carcinoid tumor. CT revealed no obvious evidence of involvement of the hilar structures and a postobstructive pneumonia. The patient's medical history was significant only for the occurrence of non-small cell lung cancer in her mother and grandmother. After extensive discussions with the patient concerning endoscopic removal of the mass, the patient requested surgical resection and was referred to the thoracic surgery service.

Because of our impression that the tumor could be removed by bronchotomy or lower lobectomy, we performed a muscle-sparing thoracotomy. However, on exploration of the chest, there was extrabronchial extension, and the mass was firmly adherent to the central hilar structures. We deemed that pneumonectomy was necessary for safe and effective removal and elected to enlarge the incision to avoid vascular injury. The incision was extended posteriorly, and the latissimus muscle was divided. The previously dissected serratus anterior muscle was easily retracted. An uneventful pneumonectomy was performed, and the chest was closed in a standard fashion. Large Jackson Pratt drains were placed behind and in front of the chest wall muscles and kept on suction for 4 days postoperatively. After the drains were removed, she exhibited a seroma, which was aspirated of 200 mL on one occasion. She was discharged from the hospital on postoperative day 9. She returned for routine surveillance on postoperative day 12. Chest radiography at that visit demonstrated normal right lung expansion, essentially midline mediastinal structures, and an appropriate fluid level in her pneumonectomy space (Fig 1). At that visit, a moderate-sized chest wall seroma was aspirated of approximately 200 mL of serosanguinous fluid.

The patient returned to our clinic 6 months later for follow-up of her carcinoid tumor. She had no complaints and was markedly less dyspneic than at her initial presentation. On examination, there were no unusual findings and no chest wall seroma. Her neck veins were flat. However, chest radiography revealed that there was marked mediastinal shift to the left (Fig 2). CT scan did not demonstrate tumor recurrence. On careful questioning, the patient admitted that her local physician had performed repeated aspirations of the seroma (several 30-mL syringes full on each of three or four occasions).

DISCUSSION

Because of the considerable morbidity associated with the standard posterolateral thoracotomy technique, several alternative surgical approaches to lung resection have been developed. The usual muscle-sparing incision involves creating subcutaneous flaps and mobilizing the anterior border of the latissimus from the superior aspect of the axilla toward its inferior insertion at the iliac crest. The serratus anterior can

*From the Departments of Surgery (Drs. Cullinane and Hartz) and Medicine (Dr. Kovitz), Tulane University Medical School, New Orleans, LA.

Manuscript received May 8, 2000; revision accepted August 28, 2000.

Correspondence to: Renee S. Hartz, MD, Department of Surgery, Tulane University Medical School, 1430 Tulane Ave. SL22, New Orleans, LA 70112; e-mail: rshartzmd@aol.com