CASE REPORTS

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Abdominal Aortic Compression to Treat Circulatory Collapse Caused by Severe Pulmonary Hypertension during Liver Transplantation

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PULMONARY hypertension associated with end-stage liver disease and portal hypertension can pose a number of serious management problems. One such problem during surgery for liver transplantation is sudden circulatory collapse.[1–3] We report the use of manual compression of the abdominal aorta to increase arterial pressure and coronary perfusion after severe hypotension in a patient with pulmonary hypertension undergoing orthotopic liver transplantation.

Case Report

A 29-year-old, 77-kg woman with autoimmune hepatitis and cirrhosis was scheduled for orthotopic liver transplantation. Relevant medical history included portal hypertension, with several episodes of upper gastrointestinal bleeding and worsening ascites. Physical examination was remarkable only for a grade 3/6 systolic murmur, loudest at the base of the heart and radiating to the neck, and a palpable spleen. Preoperative pulmonary function testing revealed a diffusion capacity 69% of predicted, normal vital capacity and FEV_1. Chest x-ray and electrocardiogram were normal. Transthoracic echocardiogram performed 3 months before transplantation showed normal right and left ventricular size and function and trace tricuspid regurgitation. Peak systolic pressure estimated from tricuspid maximal jet velocity and inferior vena cava size was 35–40 mmHg, interpreted as mild pulmonary hypertension. Arterial blood gases were a pH of 7.42, partial pressure of carbon dioxide (Pa_{CO^2}) of 29 mmHg, and arterial oxygen pressure (Pa_{O_2}) of 87 mmHg with the patient breathing room air.

Anesthesia was induced with a rapid sequence technique, using thiopental, succinylcholine, fentanyl, and oxygen, and maintained with fentanyl, isoflurane, and pancuronium. Monitoring access included bilateral radial artery catheters and an oxygen saturation pulmonary artery catheter. Initial pulmonary artery pressure (PAP) was 52/27 mmHg (mean 35 mmHg), and pulmonary vascular resistance (PVR) was 304 dyne·s·cm^{-5}. Central venous pressure was 11 mmHg, pulmonary artery occlusion pressure 6 mmHg, cardiac output 7.7 l/min, and systemic vascular resistance 675 dyne·s·cm^{-5}. Arterial blood gases were: pH 7.26, Pa_{CO2} 40 mmHg, and Pa_{O2} 178 mmHg, with a fraction of inspired oxygen level of 1.0.

Before surgical incision, we attempted to decrease the PVR with infusions of 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ nitroglycerin and 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ dobutamine, resulting in a PVR of 130 dyne·s·cm^{-5}, a PAP of 36/17 mmHg (mean 23 mmHg), systemic vascular resistance of 520 dyne·s·cm^{-5}, cardiac output of 10.2 l/min, central venous pressure of 3 mmHg, and pulmonary artery occlusion pressure of 8 mmHg. Because the pulmonary pressures and resistance were responsive to vasodilator therapy, the decision was made to proceed with the liver transplant.

Initially, the surgery proceeded uneventfully, PVR fluctuated between 130 and 210 dyne·s·cm^{-5}, and the mean PAP ranged between 23 and 44 mmHg. The liver was isolated from the circulation approximately 3.25 h after induction of anesthesia. During the anhepatic phase, venovenous bypass from the femoral and portal veins to the left axillary vein at an average flow of 1.5 l/min was used. Blood gases obtained just before the anhepatic phase were pH 7.40, Pa_{CO2} 36 mmHg, Pa_{O2} 377 mmHg, and bicarbonate 22 mEq/l. Blood gases obtained 12 min after the start of the anhepatic phase were pH 7.38, Pa_{CO2} 32 mmHg, Pa_{O2} 123 mmHg, and bicarbonate 19 mEq/l, revealing an increasing metabolic acidosis. After approximately 14 min of the anhepatic phase, the arterial pressure slowly decreased, from 91/49 mmHg (mean 66 mmHg) to 66/36 mmHg (mean 49 mmHg) (fig. 1, arterial diastolic pressure charted). At 22 min into the anhepatic phase, the PAP increased rapidly, from 26/19 mmHg to 93/73 mmHg. Systemic hypotension responded transiently to volume infusion and phenylephrine infusion, but the electrocardiogram demonstrated ST changes, with depression in the inferior and lateral leads. This ischemia was treated with 100% O_2, hyperventilation, infusion of red cells and crystalloid, nitroglycerin, isoproterenol, and multiple 5–10 μg boluses of phenylephrine and epinephrine. Approximately 30 min after the start of the anhepatic phase, both systemic and pulmonary arterial pressures decreased precipitously, associated with complete heart block at a rate of 30 beats per minute. In response to this circulatory collapse, the aorta was manually, subtotally compressed, just below the diaphragm, to increase proximal arterial pressure; during the next 1–2 min, proximal arterial pressure increased to 107/62 mmHg (mean 75 mmHg), accompanied by an almost immediate return to sinus rhythm, with resolution of the ST changes. The PAPs decreased to 36/28 mmHg (mean 31 mmHg; fig.

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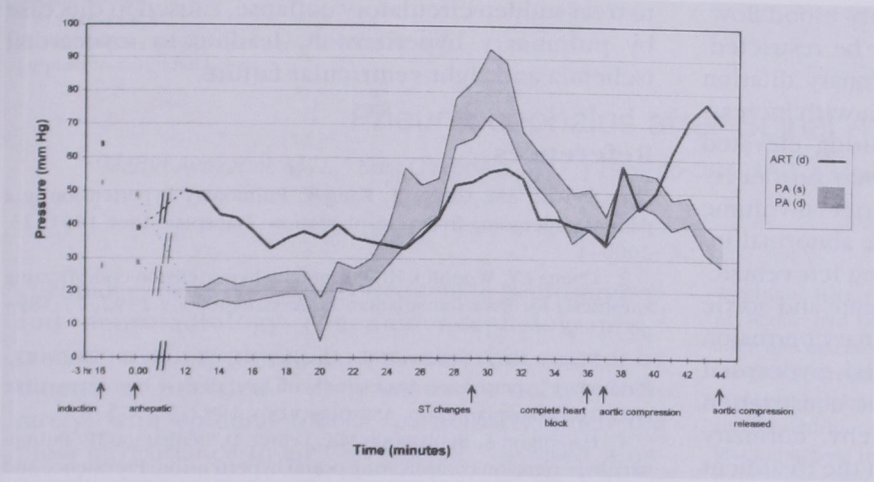

Fig. 1. Comparison of pulmonary arterial pressures (systolic and diastolic) and arterial diastolic pressure during the anhepatic phase. The pulmonary pressure range is represented by the gray filled area. The arterial diastolic pressure is displayed as a black line. The onset of electrocardiographic changes and complete heart block are noted, subsequent to the elevated pulmonary artery pressures. The reduction in pressures at the 30th min are due to ventricular failure and hemodynamic collapse, with complete resolution after aortic compression. The rise in arterial pressure is biphasic because of immediate release of aortic compression and then reapplication.

1). Two subsequent episodes of systemic hypotension and increased pulmonary pressures, followed by near circulatory collapse responding to manual compression of the aorta, occurred during the anhepatic phase.

The patient's blood pressure and electrocardiograph wave forms and rhythm were stable during venous reperfusion. During the remainder of the procedure, the mean PAP and PVR progressively increased, whereas systemic pressures were stable. At the end of surgery, the PAP was 52/41 mmHg (mean 46 mmHg), and the PVR was 400 dyne·s·cm^{-5}. Arterial blood gases were pH 7.4, Pa_{CO2} 34 mmHg, and Pa_{O2} 106 mmHg at a fraction of inspired oxygen level of 1.0. Postoperatively, the PAP ranged between 70–80/40–50 mmHg, with PVR in the range of 350–400 dyne·s·cm^{-5}. Arterial pressures were stable, in the range of 105–135/55–80 mmHg, resulting in a systemic vascular resistance range of 950–1,200 dyne·s·cm^{-5}.

The patient had spontaneous movement of all four extremities and eye opening approximately 4 h postoperatively. Liver function was satisfactory, with good bile production. The patient began to have seizures and started receiving phenytoin. Over the next 2 days, there was minimal improvement, and the trachea remained intubated for airway protection and mild hypoxemia. On the fourth postoperative day, there was worsening hypoxemia, coinciding with increasing PVR. The patient had a sudden increase in PAP, leading to complete heart block, which deteriorated rapidly to asystole refractory to pharmacologic resuscitation. Autopsy revealed chronic scattered thromboemboli in arterioles, pulmonary arteriopathy, intimal fibrosis, medial thickening, and reduplication of the elastic lamina, consistent with pulmonary hypertension. Gross examination of the heart was normal. Microscopic observation showed myocyte hypertrophy.

Discussion

Intraabdominal compression of the aorta successfully reversed the severe episode of hypotension and bradycardia that occurred during the anhepatic phase of liver transplant surgery in a patient who had concurrent severe pulmonary hypertension. We believe this sudden circulatory collapse was triggered by pulmonary hypertension, resulting in right ventricular ischemia and then failure. Intraabdominal aortic compression increased arterial diastolic pressure and coronary perfusion to a level sufficient to relieve the myocardial ischemia. Although such a maneuver may be lifesaving acutely, it cannot be expected to alter the course of postoperative pulmonary hypertension.

Although pulmonary hypertension is not common in patients with end-stage liver disease and portal hypertension, a reported prevalence of up to 2% is 2–3 times greater than in the absence of portal hypertension.[4] When pulmonary hypertension is present in patients undergoing liver transplantation, morbidity and mortality are increased.[1,2] An initial period of hypotension, as shown in figure 1, is fairly common during liver transplantation; the most likely cause is blood loss combined with fluid shifts, but other factors, such as acidosis, hypoxemia, or release of vasoactive substances, may play a role. It is unclear whether vasoactive substances acting on a susceptible pulmonary vascular bed also contributed to the sudden rise in PAP in a patient exhibiting moderate pulmonary hypertension at the outset.

Studies have shown that acute increases in PAP can trigger right heart failure, and that impairment of myocardial blood flow may lead to right ventricular ischemia.[5,6] The major determinants of myocardial blood flow are coronary perfusion pressure, coronary vasomotor tone, and intraventricular pressure (wall tension). In contrast to the left ventricle, blood flow to the right ventricle normally occurs throughout the cardiac cycle. When right ventricular systolic pressure is

elevated above arterial pressure, coronary blood flow, especially to the subendocardium, may be restricted, occurring mainly during diastole.[5] Coronary dilation may temporarily improve blood flow, but with increasing pulmonary hypertension, the resulting elevated right ventricular intracavitary pressure may further restrict blood flow.[5,7,8] Increased right ventricular volume and intracavitary pressure also can cause abnormal interventricular septal movement, reducing left ventricular cavity size and filling.[5,9] Stroke volume and aortic pressure may fall, further reducing coronary perfusion pressure, which may lead to generalized myocardial ischemia and acute heart failure.[10] Aortic constriction to maintain aortic pressure, and, thereby, coronary perfusion pressure, has been effective in the treatment of cardiac failure consequent to acute right ventricular hypertension.[5]

Typical therapeutic measures for the treatment of pulmonary hypertension involve the use of pulmonary vasodilators; in general, these drugs have similar effects on the systemic vasculature, leading to hypotension and, possibly, myocardial ischemia. Pharmacologic therapy with phenylephrine was shown to improve ventricular failure secondary to right ventricular hypertension by a mechanism similar to mechanical aortic compression.[9] Inhaled nitric oxide was recently shown to be of benefit in the intraoperative management of severe pulmonary hypertension during liver transplantation.[11] This agent was unavailable for this patient, so aortic compression was the only available method, in addition to those already used, for treatment of right ventricular ischemia.

In summary, manual aortic compression to increase systemic arterial pressure and coronary perfusion pressure may be a rapid and effective intraoperative measure to treat sudden circulatory collapse, caused in this case by pulmonary hypertension, leading to myocardial ischemia and right ventricular failure.

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