Two Case Studies of Cardiopulmonary Effects of Intra-abdominal Hypertension

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KEYWORDS
- Cardiopulmonary effect
- Intra-abdominal hypertension
- Pulmonary hypertension
- Abdominal compartment syndrome

KEY POINTS
- Intra-abdominal hypertension (IAH) falsely elevates the pulmonary artery occlusion pressure (PAOP). Volumetric pulmonary artery catheter monitoring may more accurately reflect preload in this clinical condition.
- Treatment of IAH and abdominal compartment syndrome (ACS) begins with medical interventions but requires a decompressive laparotomy for definitive management.
- Pulmonary hypertension effects cardiac function. Hypoxia may be improved with increased \( F_{io2} \), increased positive end-expiratory pressure (PEEP) and use of inotropic agents that simultaneously reduce pulmonary artery pressure.
- Ventricular dysfunction can be managed with reduction in preload by mechanical means when pharmacologic means fail.

CASE 1: ABDOMINAL COMPARTMENT SYNDROME

A 24-year-old woman had a liver transplant 2 days ago and required 15 L of fluid resuscitation. Her heart rate (HR) is 130 and blood pressure (BP) 80/50. She has a peak airway pressure of 45 with a tidal volume set at 400 mL at a rate of 14, \( F_{io2} \) 100%, and PEEP of 10 cm H\(_2\)O and arterial blood gas (ABG) 7.1, \( P_{co2} \) 52, \( P_{o2} \) 66, bicarbonate (HCO3) 16, and lactate 6. Her urine output has been 5 mL/h for the past 3 hours and her bladder pressure is 28 mm Hg. Her cardiac index (CI) is 2 L/min, pulmonary artery pressure (PAP) 52/26, and central venous pressure (CVP) 14.

This physiologic derangement is that of ACS. In general, normal adult intra-abdominal pressure (IAP) is considered 5 mm Hg to 7 mm Hg. IAH has been defined by the World Society of the Abdominal Compartment Syndrome (www.wsacs.org) as sustained increased IAP greater than or equal to 12 mm Hg and ACS as IAP greater than or equal to 20 mm Hg with new organ dysfunction or failure. IAP in excess of...
25 mm Hg, a level of IAH commonly associated with significant organ dysfunction, is generally accepted as suggesting the need for abdominal decompression.\textsuperscript{1,2}

**CARDIAC EFFECTS OF IAH**

Even modest elevations of IAP can cause a reduction in inferior vena cava blood flow and cardiac preload due to reduced venous return, which results in a drop in cardiac output. Furthermore, elevation of the diaphragm and elevated intrathoracic pressures transmitted from the IAH cause compression of the lungs, which increase the pulmonary vascular resistance. This leads to overdistension of the right ventricle and right ventricular failure, which reduces left ventricular preload. The case (described previously) demonstrates the altered pulmonary artery catheter pressure measurements observed in the setting of ACS. The elevated PAPs may lead to an assumption that preload is adequate, or even excessive, but the actual intravascular volume may be inadequate due to the reduced compliance of the thoracic cavity from the IAH. Administration of fluids may provide a temporary benefit but, without decompression of the abdomen, excessive fluid leads to overdistension of the right ventricle, worsening cardiac output.\textsuperscript{2}

Traditionally, PAOP and CVP are used as surrogates for left ventricular end-diastolic volume. Although likely valid in normal healthy individuals, the multiple assumptions necessary to use PAOP and CVP as estimates of left ventricular preload status and right ventricular preload status, respectively, are not necessarily true in critically ill patients with IAH/ACS. This inaccuracy exists because these pressures reflect the sum of the intrathoracic pressure and the intravascular pressure. Because the intrathoracic pressure is elevated, the correlation between PAOP and CVP to left ventricular end-diastolic volume is abolished. As a rule of thumb, a quick estimate of transmural (tm) filling pressures can be obtained by subtracting half the IAP from the measured filling pressure (PAOP\textsubscript{tm} = PAOP - IAP/2).\textsuperscript{1–3}

Additionally, the compliance of the ventricle is affected by the underlying disease, causing increased ventricular edema as well as elevated intrathoracic pressures. In general, the presence of IAH causes a flattening and rightward shift of the ventricular compliance curve. Therefore, a given PAOP does not correlate with left ventricular end-diastolic volume.

Resuscitation to absolute PAOP and CVP in patients with IAH/ACS should be avoided because such a practice can lead to underresuscitation, inappropriate administration of diuretics, and inappropriate end-organ perfusion. Instead, volumetric monitoring with newer-generation pulmonary artery catheters that provide a measure of the right ventricular end-diastolic volume have shown a better correlation with preload recruitable increases in cardiac output. Right ventricular end-diastolic volume goal-directed fluid resuscitation leads to a reduction of hard clinical endpoints, such as multiple organ failure and death.\textsuperscript{4} Many studies report that at high levels of PEEP, right ventricular end-diastolic volume consistently maintained a highly significant correlation with cardiac output, whereas PAOP and CVP frequently exhibited inverse correlations with cardiac output.\textsuperscript{4}

IAH falsely elevates the PAOP. Volumetric pulmonary artery catheter monitoring is a better option for monitoring preload in this clinical condition.

**PULMONARY EFFECTS OF IAH**

On average, 50% of the increased IAP is transmitted to the intrathoracic compartment. IAH causes an increase in alveolar pressures, dead space, and shunt fraction. IAH causes a decrease in transpulmonary pressures, functional residual capacity, and
static compliance of the chest wall. The combined effect of these changes is hypoxemia and hypercapnia. The elevated abdominal pressures are compromising pulmonary function in these patients, evidenced by the elevated peak airway pressures in the setting of small tidal volumes compromising ventilation. Furthermore, significant hypoxia is present despite modest PEEP and maximum inspired oxygen content.\textsuperscript{2,3,5}

To reverse this process, the abdominal cavity needs to be decompressed immediately.\textsuperscript{2–5} To improve perfusion while measures are taken to open the abdomen, a volume challenge may be warranted. Increasing the respiratory rate and tidal volume to improve minute ventilation reduces respiratory acidosis. This may require use of sedation and chemical paralysis, which is complicated by a patient’s hypotension. Unfortunately, this intervention often elevates peak airway pressures even when sedation and chemical paralysis are used. The concern about elevated airway pressures producing barotrauma is not warranted in the setting of ACS because the transalveolar pressure is the important factor. In this setting, the elevated airway pressures are not a result of overdistension of alveoli from the ventilator but are due to elevated pressures from the ACS pushing against the lung. The transmural plateau pressure takes into account the IAP and is calculated by subtracting the half the IAP from the plateau pressure. This is the value that should be kept below 30 cm H$_2$O to 35 cm H$_2$O if lung-protective strategies are used.\textsuperscript{3–5}

The acidotic pH affects cardiac function and the heart’s ability to respond to pressors. Given the patient’s poor cardiac function, the administration of HCO$_3$ to elevate the pH may benefit cardiac function. Furthermore, on opening the abdomen, there is often a washout of lactate that further drops the pH, which could lead to further myocardial depression that may be fatal. The addition of epinephrine to improve cardiac contractility is necessary given the low cardiac output and hypotension. Dobutamine may contribute to reflex hypotension, which may be detrimental in this setting. Although it is tempting to use dobutamine, because of the elevated PAPs, the observed elevated pressure is not because of intrinsic pulmonary artery hypertension but is a reflection of elevated intrathoracic pressure. Dobutamine or milrinone can reduce intrinsic pulmonary vascular resistance but would have no effect on this patient’s elevated intrathoracic pressure.

Treatment of IAH and the ACS begins with medical interventions but requires a decompressive laparotomy for definitive management.

In summary, pressure-directed resuscitation and ventilator management must take into account the effect of transmitted IAH to the thoracic cavity. Volume-based monitoring should be used in terms of cardiac function and resuscitation because this approach correlates better with cardiac function. Treatment aims to improve tissue perfusion by improving cardiopulmonary function. When medical interventions fail, surgical intervention is immediate decompressive laparotomy.

CASE 2: PULMONARY HYPERTENSION

A 70-year-old man with a history of pulmonary hypertension was in a motor vehicle crash 24 hours ago. He is intubated for hypoxia and tachypnea with multiple rib fractures. His HR is 120, BP 90/60, and urine output is 10–15 mL/h to 15 mL/h. Chest radiograph shows bilateral patchy infiltrates, PAP is 68/40, CI 1.8 is L/min, and CVP is 18. Transesophageal echocardiogram shows a distended right ventricle with global hypokinesia worse in the right ventricle, indicative of cardiac contusion. His ventilator is set at a tidal volume of 600 mL, respiratory rate 16, FiO$_2$ 60%, and PEEP 14 with an ABG pH 7.25, PCO$_2$ 50, PO$_2$ 90, HCO$_3$ 20, and lactate 3. He is started on ECMO and his lungs eventually recover, but when attempts are made to decannulate, his CI drops
to 1.8 L/min, HR increases to 120 bpm, BP is 150/100, mixed venous oxygen saturation (SvO2) drops to 50%, CVP is 5 cm H2O, pulmonary wedge pressure increases to 24 cm H2O, and systemic vascular resistance (SVR) is 4440 dyn*s/cm5.

This case illustrates the complexities of managing patients with underlying cardiac disease—in this case pulmonary hypertension—complicated by pulmonary contusion and tissue hypoperfusion. The goals of management for this patient are to (1) improve tissue perfusion, (2) minimize further lung injury, and (3) minimize the difference between myocardial oxygen demands and contractility.

**ACUTE MANAGEMENT OF PULMONARY HYPERTENSION**

Patients who have evidence of pulmonary hypertension, defined as a pulmonary artery diastolic pressure greater than 15 mm Hg and a pulmonary systolic pressure greater than 25 mm Hg in the setting of chest trauma, pose unique challenges. To optimize perfusion without increasing the difference between myocardial oxygen demands and cardiac contractility, the right ventricular end-diastolic pressure must be reduced. Overdistension of the right ventricle must be avoided to optimize right ventricular function and minimize the effects of right ventricular distension on left ventricular function. These 2 goals are accomplished if pulmonary vascular resistance is reduced.

Minimizing hypoxia and acidosis helps reduce pulmonary vascular resistance, so FIO2 may need to be increased to minimize pulmonary vasoconstriction that reflexively occurs with hypoxia. The net effect of high PEEP, however, may reduce tissue perfusion due to increasing pulmonary vascular resistance, which worsens right ventricular function. Therefore, accepting higher FIO2 and minimizing PEEP if the elevated PEEP leads to a reduced CI may be necessary.

Ventilator strategies that minimize intrathoracic pressure are necessary to facilitate right ventricular function by minimizing pulmonary vascular resistance. Keeping intrathoracic pressures low also avoids barotrauma to the lung, reducing the inflammatory response within the lung. Reducing tidal volumes and accepting a higher PCO2 (permissive hypercapnea) achieve this goal. Care must be taken that the acidosis does not diminish cardiac function. At times, the use of an HCO3 solution to combat the acidosis may be necessary during the acute resuscitation. Finally, pharmacologic reduction of the pulmonary pressures may be achieved with inotropes, such as dobutamine or milrinone, while simultaneously augmenting contractility.

Inhaled nitric oxide (iNO) therapy also provides a pharmacologically rational approach in that it reduces ventilation-perfusion mismatch due to preferential vasodilation of the pulmonary capillaries that perfuse alveoli that are sufficiently distended to receive the inhaled nitric oxide. This improves oxygenation while reducing pulmonary vascular resistance and, hence, offloads the right ventricle. Dosing of iNO, according to the guidelines of the European Society of Cardiology, involves iNO at 10 ppm to 20 ppm for 5 minutes. They defined vasoreactivity as a reduction of mean PAP greater than or equal to 10 mm Hg to reach an absolute value of mean PAP of less than 40 mm Hg with an increased or unchanged cardiac output. Despite these physiologic benefits, there are insufficient data to support the routine use of inhaled nitric oxide in this clinical setting because there are no large prospective studies showing that this affects mortality. Thereafter, oral agents, such as phosphodiesterase inhibitors (sildenafil), can be used for long-term management and should be overlapped with iNO.

Pulmonary hypertension affects cardiac function. Improving oxygenation with high FIO2, keeping intrathoracic pressure low using low tidal volumes, and vasoactive drugs to lower pulmonary pressures are all treatment options.
EXTRACORPOREAL MEMBRANE OXYGENATION

The benefits of ECMO are 2-fold: ECMO provides the ability to oxygenate the blood in the setting of pulmonary damage and it reduces right ventricular preload, thereby reducing right ventricular end-diastolic volume and pressure. The latter effect is important for patients who have borderline right ventricular function at baseline, which cannot compensate for additional increases in pulmonary vascular resistance and volume resuscitation. If such conditions develop, as in the previously described case, and are temporary or reversible, then the use of ECMO may be warranted. Several case series and a randomized trial describe the use of ECMO in patients with severe acute lung injury with improved survival. The use of ECMO also reduces the amount of ventilator support required and, therefore, ventilator pressures can be reduced, which further augment right ventricular function.

Previous randomized controlled trials of ECMO enrolled patients who had severe hypoxemia (PAO$_2$/FIO$_2$ <0.2). Patients with bleeding disorders, recent cerebrovascular accident or gastrointestinal hemorrhage, evidence of traumatic brain injury, or severe underlying nonreversible systemic disease that greatly limits the likelihood of survival were excluded. Typically, femoral-femoral or femoral-jugular cannulae are inserted for venovenous ECMO and heparinization is required to keep partial thromboplastin time between 45 seconds and 60 seconds. In patients with left ventricular dysfunction, venoarterial ECMO is indicated.

Settings of mechanical ventilation for patients on venovenous ECMO should minimize ventilator-associated lung injury and permit higher degrees of protective lung ventilation. High PEEP levels (>10 cm H$_2$O) should be maintained to prevent further lung collapse after the institution of ECMO. Blood oxygenation and decarboxylation through the ECMO circuit also allow tidal volume reduction to limit plateau pressure (suggested plateau pressure <25 cm H$_2$O). FIO$_2$ on the ventilator should be reduced to the minimal value to keep arterial saturation greater than 85%.

Weaning of venovenous ECMO should be considered when pulmonary function has improved, as indicated by higher lung compliance (increasing tidal volumes on pressure controlled ventilation), resolving lung infiltrates, and improvement in arterial PCO$_2$ and PO$_2$. Mechanical ventilation should be set to lung-protective levels of support (eg, tidal volume 6 mL/kg, plateau pressure <30 cm H$_2$O, PEEP 8–12 cm H$_2$O, and FIO$_2$ <60%). Then the fresh gas flow to the oxygenator can be switched off while maintaining previous blood flow through the circuit. If a patient remains stable and adequately ventilated after a few hours of observation, and if echocardiography reveals no evidence of severe acute cor pulmonale, ECMO cannulae can be simply pulled out. Other methods of weaning include gradual decreasing of the level of ECMO support.

ELEVATED SYSTEMIC VASCULAR RESISTANCE

In the case described, the patient is unable to come off ECMO, demonstrating signs of left ventricular overload with an elevated pulmonary wedge pressure. The management of left ventricular failure requires reduction in preload, a reduction in afterload, and an increase in contractility when preload and afterload manipulation have been optimized. Preload reduction with diuresis may facilitate ECMO discontinuation. Patients with diastolic heart failure are usually difficult to wean from the ventilator due to cardiogenic pulmonary edema because ventilator pressures are reduced, leading to increased flow to the left ventricle. The treatment of these patients aims at reducing pulmonary venous pressure and congestion, and such treatment usually requires diuretic therapy. Aggressive diuresis may result in serious hypotension in patients with diastolic heart failure because of the steepness of the curve of left ventricular diastolic pressure in

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relation to volume. Moreover, tachycardia may cause insufficient time for complete relaxation, resulting in an increase in diastolic pressure that compromises left ventricular filling, leading to elevated left-sided filling pressures. Therefore, afterload reduction should be used when possible to manage the elevated SVR. SVR is a calculated value \([\text{MAP} - \text{CVP}] \times \frac{80}{\text{CO}}\), where \(\text{MAP}\) is mean arterial pressure and \(\text{CO}\) is cardiac output. Therefore, afterload elevation may not be the cause of the increased SVR but instead may be the result of a substantial drop in cardiac output. When SVR increases are due to the latter, contractility agents, such as epinephrine, are necessary. Agents, such as dobutamine or milrinone, have the added advantage of reducing afterload while simultaneously increasing contractility and should be used in the case described.

Ventricular dysfunction can be managed with reduction in preload by mechanical means when pharmacologic means fail.

REFERENCES