Ruptured Liver Hematoma Complicated by Increased Intra-abdominal Pressure: Critical Care Considerations

Avishai Ziser MD[1,2], Ludmila Guralnik MD[3], Robert Markovits MD[1], Yousif Matanis MD[2] and Genia Mahamid MD[2]

Departments of [1]Anesthesiology, [2]Critical Care and [3]Diagnostic Radiology, Rambam Medical Center and Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

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We report a 24-year-old man with acute bleeding from a liver hematoma, complicated by increased intraabdominal pressure, abdominal compartment syndrome, severe respiratory insufficiency, and adult respiratory distress syndrome. The patient was treated medically and recovered. Clinicians should be alert to complications associated with abdominal compartment syndrome, as well as to the options of medical versus surgical treatment.

Case Description

This 24-year-old 120 kg male was evaluated for complaints of dyspnea, cough and right pleuritic pain after a few days of flu-like symptoms. Chest radiography demonstrated right lower lobe pneumonia and pleural fluid; ultrasound showed right popliteal venous thrombosis; and spiral computerized tomography scan depicted filling defect in the right pulmonary artery compatible with pulmonary thromboembolism. Heparin and warfarin were administered. The pleural fluid was aspirated and cultured for *Staphylococcus* coagulase positive. The procedure was technically difficult to perform, and after multiple attempts a right pleural chest tube was inserted and 300 ml of pus aspirated. CT scan demonstrated a right lower lobe consolidation and a hematoma in the right lobe of the liver, near its capsule. The patient's clinical status gradually improved; his temperature fell and dyspnea and tachypnea ceased.

Five days after admission the patient suddenly complained of severe abdominal pain. Heart rate was 130 beats/min and blood pressure 90/60 mmHg. Intraabdominal bleeding was suspected. Packed red blood cells and blood products were administered. A repeat CT scan [Figure] showed that the liver hematoma had greatly increased in size and had ruptured into the abdominal cavity, with free blood in the peritoneum, around the spleen and in the pelvis. Selective right hepatic artery angiogram was performed, and the branches supplying the area of the hematoma were embolized. A Greenfield filter was placed in the inferior vena cava and anticoagulant therapy was discontinued. The patient's abdomen was tense and distended and respiratory failure ensued soon after, characterized by dyspnea, tachypnea of 60 breaths/min, hypercapnia up to 52 mmHg and a PaO_2/FiO_2 ratio[1] of 100. Endotracheal intubation was performed and mechanical ventilation started, and sedatives and muscle relaxants were given. Nitric oxide was added, with a temporary improvement in oxygenation.

Chest radiography showed bilateral infiltrates compatible with adult respiratory distress syndrome. Airway pressure-release ventilation was begun, with I:E ratio of 3:1, peak inspiratory pressure of 30–35 cm H_2O and positive end-expiratory pressure of 8. The patient was turned prone, which resulted in an increase in intraabdominal pressure from 32 mmHg in the supine position to 39 mmHg. Hemodynamic parameters demonstrated high cardiac output and low systemic vascular resistance, appropriate cardiac filling pressures and elevated pulmonary artery pressure. The patient was turned supine after 24 h and intraabdominal pressure gradually decreased. A repeated CT scan 3 days later revealed a marked decrease in the amount of intraabdominal blood, mainly around the spleen. Administration of sedatives and muscle relaxants was discontinued, the patient was extubated and 2 days later was discharged from the Intensive Care Unit.

Comment

In this patient a ruptured liver hematoma led to intraabdominal bleeding, increased intraabdominal pressure and ACS[2]. We believe that the liver hematoma was inadvertently caused during attempts to perform pleural puncture. Combined with the anticoagulation therapy for pulmonary thromboembolism, bleeding in the liver continued until the hematoma ruptured the liver

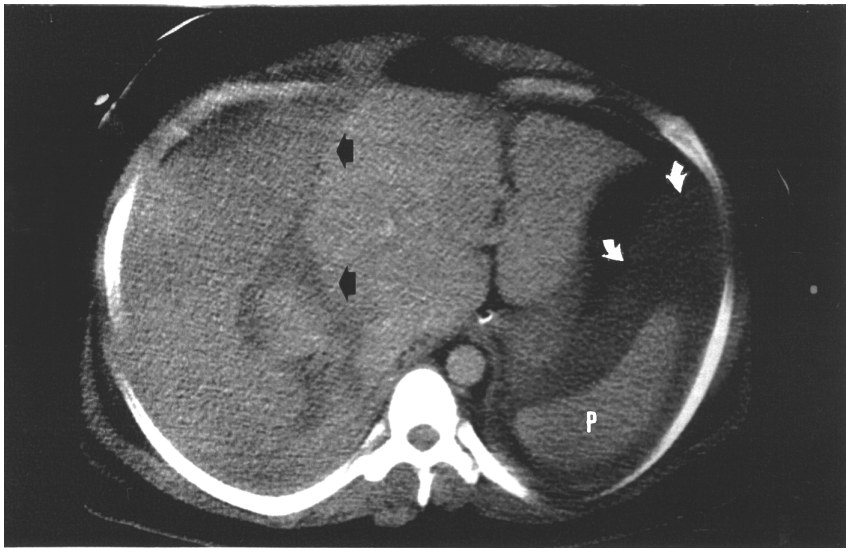
[1] PaO_2/FiO_2 = partial pressure of O_2/fraction of expired O_2

[2] ACS = abdominal compartment syndrome

capsule. Bleeding ceased once the coagulation parameters returned to the normal range; and, in addition, the increased IAP[3] functioned as a mechanical tamponade. ACS was diagnosed — as previously described [1] — by the findings of a tense and distended abdomen, elevated peak airway pressure, and hypercarbia. High intra-abdominal pressure supported the diagnosis.

Oliguria is common in ACS, but not always present [1]. Respiratory insufficiency, caused by the elevated diaphragm and increased intrathoracic pressure, is an early sign of the condition, and hypoventilation, hypoxemia and high Paw are common. Cases of PIP[4] above 85–90 cm H_2O have also been reported [2]. We avoided high PIP by using pressure-controlled ventilation and APRV[5]. ARDS, massive transfusion and hypovolemic shock [3] all contributed to the severe hypoxemia.

We chose APRV for several reasons. Since it is a pressure cycled mode, high PAW is avoided. A long inspiratory phase maintains high mean airway pressure, and combined with PEEP[6] this mode of ventilation helps to prevent atelectasis, which is common in ACS due to the elevation of the diaphragm. APRV may induce progressive alveolar recruitment over time, and keeps the alveoli open. Other potential benefits from APRV include less barotrauma, reduction in circulatory compromise, and better matching of pulmonary ventilation and perfusion [4]. A dynamic compliance of 18–26 cm H_2O enabled a tidal volume of about 500 ml, and the $PaCO_2$ was maintained in the range of 45–55 mmHg. Permissive hypercapnia, which is associated with reduced PIP and low tidal volume, is commonly used for patients with ARDS and should be considered for ventilatory failure associated with ACS, even without ARDS. It may prevent barotrauma and volutrauma to the lungs. Turning the patient prone has been shown to improve oxygenation and decrease the alveolar-to-arterial oxygen difference in patients with ARDS. It substantially increased IAP in our patient but did not cause hemodynamic or respiratory deterioration. Nitric oxide seemed a valuable adjunct in our patient. This agent improves oxygenation, particularly in those with increased pulmonary vascular resistance, which is typical for patients with increased IAP. The combination of nitric oxide and a prone position may have additive beneficial effects in patients with ARDS [5]. A few attempts at early weaning from nitric oxide caused an immediate drop in SaO_2 and an increase in systolic pulmonary artery pressure up to 73 mmHg. Intraabdominal pressure of 25–32 cm H_2O is considered a high risk for ACS [1]; yet high pressure by itself is not an indication for decompression laparotomy. Our patient had high cardiac output and oxygen delivery, normal mixed venous oxygen saturation, blood pH and lactate, with minimal inotropic support (dopamine 2.7 μg/kg/min). Maintaining high cardiac output can be achieved by optimizing preload with an addition of inotropic agents. A pulmonary artery catheter might be valuable for monitoring and guiding therapy. A decreasing SvO_2[7] might be an early sign of ACS, and oxymetric pulmonary artery catheter with continuous measurement of SvO_2 can be helpful, although this was not studied in our patient. Renal function and urinary output were maintained by volume infusion to assure high preload pressures, "renal dose" dopamine, and low-dose diuretics.



CT scan showing a huge subcapsular and intrahepatic hematoma (black arrows), with free blood (white arrows) in the abdominal cavity and around the spleen (P).

Signs of worsening ACS include low cardiac and urine output, decreasing SvO_2, and metabolic acidosis. Such cases require decompression laparotomy [1]. Surgical decompression of the abdomen is a major intervention that is associated with high mortality [1], and surgical hemostasis for parenchymal hepatic bleeding is difficult. While our patient had a severe life-threatening respiratory insufficiency, his hemodynamic parameters and renal function remained stable, and we therefore elected not to perform surgical decompression.

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Correspondence: Dr. A. Ziser, Dept. of Anesthesiology, Rambam Medical Center, P.O.Box 9602, Haifa 31096, Israel. Tel: (972-4) 854 2487; Fax: (972-4) 854 2961; email: aziser@rambam.health.gov.il

[3] IAP = intraabdominal pressure
[4] PIP = peak inspiratory pressure
[5] APRV = airway pressure-release ventilation
[6] PEEP = positive end-expiratory pressure
[7] SvO_2 = mixed venous oxygen saturation