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Case Report

Abdominal Compartment Syndrome and Acute Kidney Injury Due to Excessive Auto–Positive End-Expiratory Pressure

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Abdominal compartment syndrome is an under-recognized cause of acute kidney injury in critically ill patients. We report a case of a patient with severe obstructive lung disease who, while intubated for respiratory failure, developed abdominal compartment syndrome and oliguric acute kidney injury due to air-trapping and excessive auto–positive end-expiratory pressure (auto-PEEP; also known as intrinsic PEEP). When chemical paralysis was initiated and the auto-PEEP resolved, the patient's intra-abdominal hypertension rapidly improved and kidney function recovered immediately. Abdominal compartment syndrome secondary to excessive auto-PEEP appears to be unreported in the literature; however, any process that significantly increases intrathoracic pressure conceivably could cause increased pressure to be transmitted to the abdominal compartment, resulting in organ failure. Patients undergoing mechanical ventilation, which puts them at risk of airflow obstruction and the development of intra-abdominal hypertension, should be evaluated for air-trapping and excessive auto-PEEP.
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INDEX WORDS: Intra-abdominal hypertension; intra-abdominal pressure; abdominal compartment syndrome; acute kidney injury; auto–positive end-expiratory pressure (auto-PEEP).

The past few decades have seen an overwhelming increase in the understanding of intra-abdominal hypertension (IAH) and abdominal compartment syndrome. Despite initial reports more than a century ago that IAH can have detrimental effects on the kidney, there has been a relative dearth of scientific literature supporting this connection, especially from the nephrology community. However, recent evidence has implicated IAH as an independent risk factor for acute kidney injury (AKI). In intensive care populations, the incidence of IAH and abdominal compartment syndrome may be high as 54% and 12%, respectively.[1] Therefore, nephrologists must maintain a high index of suspicion when confronted with AKI in a critically ill patient.

CASE REPORT

A 57-year-old man with severe chronic obstructive pulmonary disease due to α_1-antitrypsin deficiency was admitted with a 2-day history of progressive shortness of breath. The patient was being treated with home oxygen therapy as well as nocturnal bilevel positive airway pressure for sleep apnea. He reported a dry cough but denied fevers, sputum production, chest pain, or abdominal pain. He had been hospitalized 6 times in the previous year for chronic obstructive pulmonary disease exacerbations. Pulmonary function testing from 16 months prior to admission showed a forced expiratory volume in the first second of expiration of 0.59 L (26% of predicted) with a significant bronchodilator response and elevated residual volume to total lung capacity ratio of 211% of predicted, suggestive of significant air trapping. Outpatient medications included fluticasone/salmeterol and albuterol inhalers.

On presentation, the patient was afebrile, with heart rate of 101 beats/min, blood pressure of 142/90 mm Hg, and respiratory rate of 24 breaths/min with blood oxygen saturation of 98% on 2 L/min of oxygen through a nasal cannula. Physical examination showed an obese man in no acute respiratory distress. Lung examination was notable for decreased air movement bilaterally with scattered expiratory wheezes. On cardiac auscultation, the patient had regular tachycardia without murmurs. Physical examination findings were otherwise unremarkable. Laboratory data included the following values: white blood cell count, 10.8 $\times 10^3/\mu$L; serum creatinine, 0.73 mg/dL (corresponding to estimated glomerular filtration rate of >60 mL/min/1.73 m^2 using the IDMS-traceable 4-variable MDRD [Modification of Diet in Renal Disease] Study equation); and serum urea nitrogen, 7 mg/dL. Arterial blood gas analysis showed pH 7.34, $Paco_2$ of 55 mm Hg, Pao_2 of 80 mm Hg, bicarbonate level of 29 mEq/L, and arterial oxygen saturation of 93%. A chest radiograph showed bilateral lung hyperexpansion, but no infiltrates or effusions. His most recent echocardiogram showed ejection fraction ≥75%.

The patient was admitted to a general medical-surgical floor and started on treatment with steroids, albuterol/ipratropium nebulizers, and levofloxacin. Five hours after admission, he developed worsening dyspnea with increased work of breathing manifested by diaphoresis and use of accessory muscles of respiration. He was intubated with a size 7.0 endotracheal tube and transferred to the intensive care unit.

Persistent hypercapnea and high airway pressures complicated his course in the intensive care unit. Despite different ventilatory strategies and aggressive sedation with midazolam, propofol, and fentanyl, peak inspiratory pressures often were >50 cm H_2O and

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Table 1. Relevant Laboratory Data During the Course of Illness

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Serum potassium (mEq/L)	3.2	3.5	5.5	5.0	4.0	3.9	3.6	4.2
Serum bicarbonate (mEq/L)	28	24	24	19	21	21	23	25
Serum urea nitrogen (mg/dL)	7	15	33	67	64	56	46	37
Serum creatinine (mg/dL)	0.73	1.08	2.12	3.37	2.87	2.40	1.62	1.30
24-h urine output (mL)	1,400	1,225	500	1,450	1,725	1,770	2,100	2,050
Highest recorded PIP (cm H_2O)	—	35	45	67	45	40	42	40
Bladder pressures (cm H_2O)	—	20	30	35; 5[a]	5	—	—	—

Note: Conversion factors for units: serum urea nitrogen in mg/dL to mmol/L, ×0.357; creatinine in mg/dL to μmol/L, ×88.4.
Abbreviation: PIP, peak inspiratory pressure.
[a]Two measurements were performed.

auto–positive end-expiratory pressure (auto-PEEP) persistently was >25 cm H_2O. Subsequently, he developed oliguria; blood urea nitrogen and serum creatinine levels, which had been increasing since admission, now were 67 mg/dL and 3.37 mg/dL, respectively, corresponding to estimated glomerular filtration rate of 23 mL/min/1.73 m^2 using the MDRD equation (Table 1). Urinalysis results were unremarkable; urine sediment showed few hyaline casts but otherwise was bland. Random urine sodium excretion was <10 mEq/L. Increasing abdominal distention was noted on serial abdominal examinations. Intravesicular pressures were elevated when transduced using an indwelling urinary catheter (Table 1).

Abdominal computed tomographic scan showed no acute intra-abdominal pathology, but cirrhosis without ascites was noted. This finding of liver cirrhosis most likely was due to α_1-antitrypsin deficiency. Kidney ultrasound showed kidneys that were normal in size and appearance without evidence of hydronephrosis or calculi.

During the next few hours, the patient's urine output continued to decrease (no urine for >6 hours), he had worsening metabolic acidosis, and renal replacement therapy was considered. In the meantime, refractory hypercapnea and auto-PEEP led to the decision to chemically paralyze. A 20-mg intravenous bolus of cisatracurium was administered, which led to an immediate decrease in auto-PEEP from 35 to 8 cm H_2O. The patient then was maintained on a continuous infusion of cisatracurium. Within several minutes of the initial administration of the neuromuscular blocking agent, bladder pressure decreased from 35 to 5 cm H_2O. After the first 3 hours of paralysis, urine output totaled 300 mL, followed by several liters over the next 24 hours. Renal replacement therapy was not initiated, and during the next few days, his creatinine level returned to baseline. Hypercapnea improved, and he eventually was extubated and maintained on intermittent bilevel positive airway pressure. A few days later, he was discharged to an inpatient rehabilitation facility.

DISCUSSION

Normal intra-abdominal pressure varies with respiration, but generally is ≤5 mm Hg. IAH is defined as sustained intra-abdominal pressure ≥12 mm Hg, and abdominal compartment syndrome occurs when elevated intra-abdominal pressure leads to organ dysfunction. Abdominal compartment syndrome generally does not occur at pressures <20 mm Hg.[2]

IAH and abdominal compartment syndrome can affect multiple organ systems. Pulmonary compliance is decreased, which decreases total lung capacity and residual volume. This can result in respiratory failure due to hypoventilation or barotrauma due to prolonged exposure to elevated airway pressures. Similarly, IAH can decrease venous return to the heart, which leads to decreased cardiac preload, which in turn may result in decreased cardiac output.[3] The gastrointestinal system may be affected because increased intra-abdominal pressure decreases mesenteric blood flow and intestinal perfusion. Neurologically, sustained elevations in abdominal pressure can elevate intracranial pressure.[4] Common early clinical manifestations associated with IAH and abdominal compartment syndrome in critically ill patients include a tense abdomen, elevated peak inspiratory pressures, oliguria, and difficulty ventilating.[5-7]

The association between intra-abdominal hypertension and oliguria has been observed since the late 19th century. Only more recently has there been an appreciation for the decrease in kidney function at lower levels of IAH. Malbrain et al[8] reported that IAH (defined as intra-abdominal pressure ≥18 mm Hg) was an independent cause of AKI in patients admitted to the intensive care unit after abdominal surgery. Abdominal perfusion pressure, which conceptually is similar to cerebral perfusion pressure, can be defined as the difference between mean arterial pressure and intra-abdominal pressure.[9] As IAH increases, abdominal perfusion pressure decreases, and this in turn causes a decrease in kidney perfusion pressure.

Apart from the decreased kidney perfusion induced by low arterial perfusion, elevated intra-abdominal pressure also can cause renal vein compression, which increases venous resistance, impairs venous drainage, and leads to a progressive decrease in glomerular perfusion.[10] This decrease in glomerular filtration gradient appears to be the key causative factor in IAH-induced AKI. As described