Abdominal compartment syndrome successfully treated with neuromuscular blockade

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ABSTRACT
A 48 year old male admitted to the intensive care unit after a cardiac arrest complicated by a stroke intra-operatively during automatic implantable cardioverter defibrillator placement. He post-operatively developed a rigid abdomen, elevated peak and plateau pressures, hypoxia and renal insufficiency. He was diagnosed with abdominal compartment syndrome with an intra-abdominal compartment pressure of 40mmHg. The patient was administered 10 mg of intravenous cisatracuriumbesylate in preparation for bedside surgical abdominal decompression. Cisatracurium eliminated the patients need for surgical intervention by reducing his abdominal compartment pressures to normal and improving his hypoxia and renal function. This case illustrates that neuromuscular blockade should be attempted in patients with abdominal compartment syndrome prior to surgical intervention.

Key words: Abdominal compartment syndrome, intra-abdominal hypertension, medical management of abdominal compartment syndrome, neuromuscular blockade

INTRODUCTION
Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are increasingly common in critically ill patients following massive fluid resuscitation, severe abdominal trauma and extensive abdominal surgery.\(^\text{[1-3]}\) These conditions left untreated have been associated with significant morbidity and mortality.\(^\text{[4,5]}\) Increased intra-abdominal pressure (IAP) monitoring and consensus definitions and recommendations have lead to greater recognition of this condition and patient survival. ACS is defined as a sustained IAP of >20 mmHg that is associated with new organ dysfunction or failure.\(^\text{[6]}\) The mainstay of therapy is immediate surgical decompression of the abdomen. There have been some reports of temporary improvement in IAP following neuromuscular blockade (NMB) in patients with IAH, but all eventually required surgical management.\(^\text{[7-9]}\) In this report, we describe a patient who was definitively treated with only NMB, with complete resolution of symptoms.

CASE REPORT
A 48-year-old morbidly obese African-American male with a medical history of congestive heart failure, hypertension, obstructive sleep apnea on 2 l of home oxygen and non-complaint with continuous positive airway pressure at night presented to the emergency department (ED) with worsening shortness of breath and lower extremity oedema over one week. Vital signs in the ED were temperature, 98.7 degrees Fahrenheit; blood pressure, 116/66 mmHg; heart rate, 130 beats per minute; respiratory rate, 30; oxygen saturation, 85% on 2 l nasal cannula; weight, 162 kg and body mass index, 47. Physical examination revealed a morbidly obese male in acute respiratory distress with an irregularly irregular pulse and one plus lower extremity pitting oedema. Laboratory values in the ED include white blood cell count, 9 800/µl; haemoglobin, 14.2 g/dl; platelet count, 230 000/µl; sodium, 140 mEq/l; potassium, 4.5 mEq/l; chloride, 104 mEq/l; bicarbonate, 29 mEq/l; blood urea nitrogen (BUN), 38 mg/dl; creatinine, 1.4 mg/dl; glucose, 105 mg/dl; aspartate aminotransferase (AST), 51 U/l; alanine aminotransferase (ALT), 40 U/l; alkaline phosphatase,
68 U/l; total bilirubin, 1.5 mg/dl; albumin, 3.4 g/dl; total protein, 7.1 g/dl; international normalised ratio (INR), 1.2; myoglobin, 97 μg/l; troponin, 0.07 ng/ml and B-type natriuretic peptide, 934 pg/ml. Urine toxicology screen was positive for cocaine. Arterial blood gas showed a pH 7.33, PaCO$_2$ 68.5 and PaO$_2$ 88.6 on FiO$_2$ of 40%. Electrocardiogram confirmed atrial fibrillation with a rapid ventricular response. Echocardiogram revealed a myopathic severely enlarged left ventricular chamber with moderate concentric left ventricular hypertrophy and severe global hypokinesis with an ejection fraction of 8 to 10%. The patient’s work of breathing and oxygenation improved with Bipap. He was started on diltiazem and diuresis was initiated. The patient was admitted to the step-down unit with a new diagnosis of atrial fibrillation.

On hospital day number eight, the overnight nurse found the patient unresponsive. A code blue was called. His initial rhythm was ventricular fibrillation and he was shocked with 200 J twice before regaining pulses. When stabilised, he was transferred to the intensive care unit (ICU) where he regained consciousness and full neurologic function within one hour of the event. The following day, an automatic implantable cardioverter defibrillator (AICD) was placed. Intraoperatively, the patient had runs of ventricular tachycardia with hypotension to 80 mmHg systolic. Postoperatively, the patient was found to have a right fixed mid-dilated pupil, flaccid paralysis of the left arm and leg with hyperreflexia throughout the left side. Computed tomography (CT) of the head revealed a right midbrain and thalamic stroke thought to be secondary to transient arrhythmia-induced hypoperfusion, intraoperatively.

On postoperative day number three, after AICD placement and stroke, the patient acutely developed a rigid distended board-like abdomen with dullness to percussion. Vitals at that time were temperature, 98.4 degrees Fahrenheit; blood pressure, 112/67 mmHg; heart rate range, 55 beats per minute, and respiratory rate 26 on ventilator settings of assist control volume control with a rate of 26; tidal volume, 500 ml; FiO$_2$, 40% and positive end-expiratory pressure of 5. His peak inspiratory pressure on the ventilator had risen from 26 to 72 mmHg; his plateau had risen from 15 to 48 mmHg with oxygen desaturation from 93 to 85%. The patient had received 1.5 l in and urinated 4.5 l over the 24 hours, leading up to this event. His laboratory values from that morning were unchanged from previous (white blood count, 10 400/μl; haemoglobin, 12.6 g/dl; platelets, 200 000/μl; sodium, 139 mEq/l; potassium, 4.7 mEq/l; chloride, 100 mEq/l; bicarbonate, 34 mEq/l; BUN, 36 mg/dl; creatinine, 1.41 mg/dl; glucose, 78 mg/dl; calcium, 8.3 mg/dl; magnesium, 2.0 mEq/l; phosphorus, 3.0 mg/dl; AST, 77 U/l; ALT, 94 U/l; alkaline phosphatase, 186 U/l; total bilirubin, 1.3 mg/dl). This constellation of findings prompted the team to measure an abdominal compartment pressure which was elevated to 53 cm H$_2$O (40 mmHg) with an abdominal perfusion pressure (APP) of 42 mmHg. A CT scan of the abdomen and pelvis was performed which showed no dilated loops of bowel, no ileus, no evidence of free air, abscess or obstruction and minimal free fluid [Figure 1]. Pertinent labs from the event revealed a lactate of 1.0 mg/dl; lipase, 159 U/l; amylase, 76 U/l; creatinine, 1.53 mg/dl. Surgery was consulted and the FiO$_2$ was increased to 100%, which improved his oxygen saturation to 96% despite his elevated peak inspiratory pressures.

Figure 1: Computed tomography of the abdomen and pelvis showing no dilated loops of bowel, ileus, free air, abscess or obstruction.
Surgical recommendations were for an emergent bedside surgical abdominal decompression. The patient was administered 10 mg of intravenous cisatracurium besylate by anaesthesia in preparation for surgical management. Shortly following paralytic administration, his peak inspiratory pressure corrected from 72 to 23 mmHg, his plateau pressure from 48 to 16 mmHg and his oxygenation status tolerated a decrease in FiO$_2$ down to 40% with his oxygen saturation returning to baseline. Measurement of his abdominal compartment pressure revealed a drop from 53 (40 mmHg) to 23 cm H$_2$O (17 mmHg) and then normalised to 15 cm H$_2$O (11 mmHg) over a three-hour period. During this entire event, the patient averaged 100 to 200 cc/hr of urine output and laboratory values from the following day showed a drop in creatinine from 1.53 to 1.29 mg/dl. Given this correction, surgical management was postponed and the patient was monitored. The patient remained hospitalised for an additional 73 days. He was ultimately transferred to a skilled nursing facility without return of symptoms.

**DISCUSSION**

To our knowledge, this is the first documented case of NMB definitively treating ACS. A few case reports and one series discuss the use of NMB to temporarily ameliorate IAH, but none have shown benefit in patients with ACS.[7-9] De Laet et al. performed the first prospective trial of NMB in the management of IAH, reporting temporary reductions in IAP in nine out of ten patients. The single patient who did not respond had an IAP of 25 mmHg (Grade IV IAH), leading the authors to suggest NMB is not effective in severe IAP or ACS. The World Society of the Abdominal Compartment Syndrome state that NMB may be considered in selected patients with mild to moderate IAH while other interventions are being performed (Grade 2C).

Our patient met criteria for ACS based on IAP of 40 mmHg associated with worsening hypoxia and respiratory distress. His APP (APP = MAP – IAP) was 42 mmHg (82 – 40 mmHg). With this reduction in perfusion pressure, his creatinine increased from 1.41 to 1.53 mg/dl. His peak inspiratory pressures went as high as 72 mmHg, while his oxygen saturation dropped to 85% on a FiO$_2$ of 40%.

Numerous risk factors for the development of IAH and ACS have been suggested in an attempt to predict high-risk patients and explain the pathophysiology of this disorder. Independent risk factors identified by three large prospective trials include large volume fluid resuscitation (>3.5 l/24hours); pancreatitis, pulmonary, renal and liver dysfunction; acidosis, hypothermia; abdominal surgery; coagulopathy and ileus.[2-5] Despite the wide variety of risk factors, the final pathway remains the same; hypoperfusion of the intra-abdominal organs leading to hypoxia and proinflammatory cytokine release. Cytokines in concert with oxygen radicals promote capillary leakage causing oedema and the ever increasing swollen tissue further raises the IAP leading to a feed-forward cascade.[10]

NMB is thought to improve IAH by increasing abdominal wall compliance, thus decreasing the IAP by increasing the total amount of abdominal compartment volume through muscle relaxation.[11] Although there is no consensus on how NMB ameliorates ACS, it may be that early treatment of IAH/ACS stops the cycle of tissue abdominal organ hypoxia and oedema before it spirals out of control.

This case illustrates two important concepts of ACS. One, the pathophysiology of this illness is diverse. ACS is not always due to volume expansion or ileus. This patient had no preceding acute volume resuscitation, ischaemia, acidosis, hypothermia or inflammation. This episode of ACS was likely due to spastic contractions of his abdominal muscles decreasing abdominal wall compliance and increasing abdominal pressure. The etiology of his abdominal muscle spasm is not clear. Numerous causes have been reported, mostly secondary to intra-abdominal inflammatory conditions, which are not relevant to this case.[12] It has also been reported with abdominal cutaneous nerve root entrapments which are more common in obese individuals like our patient.[13,14] Epilepsia partialis continua, secondary to an acute stroke, has also been reported to cause contraction of the abdominal wall musculature, and this is also remotely possible, given our patient’s recent large stroke.[15] Two, medical management of ACS should always be attempted prior to surgical decompression. This includes evacuation of the intraluminal contents by nasogastric tubes, rectal tubes and prokinetics,[16-18] removal of intraperitoneal fluid or ascites by paracentesis and improvement of abdominal wall compliance through adequate sedation, elevation of the head of bed and potentially NMB.
CONCLUSION

IAH and ACS are an increasingly common problem in ICU patients. Therapy is usually considered to be surgical. But, practitioners should always consider medical management first. In addition to removing intraluminal and extraluminal fluid, NMB should be attempted to ameliorate the pressure. In rare cases like ours, it may even be curative.

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