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CLINICAL INFORMATION

Management of abdominal compartment syndrome after transurethral resection of the prostate

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KEYWORDS
Transurethral resection of the prostate;
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Abstract Acute abdominal compartment syndrome is most commonly associated with blunt abdominal trauma, although it has been seen after ruptured abdominal aortic aneurysm, liver transplantation, pancreatitis, and massive volume resuscitation. Acute abdominal compartment syndrome develops once the intra-abdominal pressure increases to 20–25 mm Hg and is characterized by an increase in airway pressures, inadequate ventilation and oxygenation, altered renal function, and hemodynamic instability. This case report details the development of acute abdominal compartment syndrome during transurethral resection of the prostate with extra- and intraperitoneal bladder rupture under general anesthesia. The first signs of acute abdominal compartment syndrome in this patient were high peak airway pressures and difficulty delivering tidal volumes. Management of the compartment syndrome included re-intubation, emergent exploratory laparotomy, and drainage of irrigation fluid. Difficulty with ventilation should alert the anesthesiologist to consider abdominal compartment syndrome high in the list of differential diagnoses during any endoscopic bladder or bowel case.

PALAVRAS-CHAVE
Ressecção transuretral de próstata;
Ruptura de bexiga;
Síndrome compartimental abdominal

Manejo da síndrome do compartimento abdominal pós-ressecção transuretral de próstata

Resumo A síndrome compartimental abdominal aguda é mais comumente associada a trauma abdominal fechado, embora tenha sido observada após ruptura de aneurisma da aorta abdominal, transplante de fígado, pancreatite e reanimação com volume maciço. A síndrome compartimental abdominal aguda surge quando a pressão intra-abdominal aumenta para 20-25 mm Hg e é caracterizada pelo aumento das pressões das vias aéreas, ventilação e oxigenação inadequadas, função renal alterada e instabilidade hemodinâmica. Este relato de caso descreve o desenvolvimento da síndrome compartimental abdominal aguda durante a ressecção transuretral de próstata com ruptura da bexiga extra e intraperitoneal sob anestesia geral. Os primeiros sinais da síndrome compartimental abdominal aguda nesse paciente eram

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pressões de pico elevadas das vias aéreas e dificuldade para fornecer volumes correntes. O manejo da síndrome de compartimento inclui reintubação, laparotomia exploratória de emergência e drenagem de líquidos de irrigação. A dificuldade na ventilação deve alertar o anestesiologista para que considere a síndrome compartimental abdominal em primeiro lugar na lista de diagnósticos diferenciais durante qualquer caso de endoscopia de bexiga ou intestino.

Introduction

Intraperitoneal bladder rupture is a rare complication of transurethral resection of the prostate (TURP). Bladder perforation, both extra- and intraperitoneal, occurs in only 1.3% of all patients receiving this procedure. Only 17% of all perforations are intraperitoneal.[1] Intraperitoneal bladder perforation is a severe complication which requires immediate treatment to prevent serious consequences such as peritonitis, uremia, acidosis, and compartment syndrome.[2] Abdominal compartment syndrome (ACS) develops once the intra-abdominal pressure (IAP) increases to 20–25 mm Hg.[3,4] The initial insult from increased IAP is decreased venous return, leading to hypovolemic shock. A study on dogs showed a decrease in cardiac output and stroke volume of 36% after IAP increased to 40 mm Hg.[5] During hypovolemic shock, the increased sympathetic outflow causes a decrease in splanchnic perfusion. Hypoxia of the abdominal tissues initiates a release of inflammatory cytokines that increase capillary permeability and tissue edema, further increasing the IAP. A study on rats[6] showed that along with decreased mean arterial pressure and pH, increasing IAP leads to increased levels of TNF-α, IL-1, and IL-6, i.e. pro-inflammatory cytokines which may serve as a second insult for the induction of multi-organ failure. Unless diagnosed and treated rapidly, up to 36% of cases of ACS lead to multiorgan failure.[7] The increase in thoracic pressure results in a decrease in all lung volumes except residual volume.[5] If a patient is on volume controlled ventilation, a precipitous rise in peak airway pressures may be seen. This case report details the development of ACS during TURP with extra- and intraperitoneal bladder rupture under general anesthesia.

Case description

A 79-year-old male was scheduled to undergo palliative TURP for prostate cancer and obstructive uropathy. His medical comorbidities included hypertension and type II diabetes. Physical examination was significant for a well-appearing elderly male with an indwelling Foley catheter, heart rate 105 beats/min, blood pressure 168/93 mm Hg, body mass index 26 kg/m^2, Mallampati class III airway, and a 2–3 cm oral aperture. Pertinent laboratory tests included a hematocrit of 43.8% and creatinine 1.1 mg/dL. An electrocardiogram revealed sinus tachycardia with left anterior fascicular block. He was deemed a low cardiac risk for a low risk procedure.

Intraoperative monitoring for this patient included standard American Society of Anesthesiologists (ASA) monitors. An 18-gauge peripheral intravenous catheter was placed prior to induction. Anesthesia induction and tracheal intubation were uneventfully performed and the surgery was begun. Volume controlled ventilation was started with settings of: tidal volume 500 mL, respiratory rate 10/min, positive end-expiratory pressure 5 cm H_2O. Two hours into the surgery, the peak airway pressures increased from 20 to 37 cm H_2O, tidal volumes decreased from 450 to 100 mL, and an audible inspiratory leak around the endotracheal tube cuff was heard. After checking the cuff for adequate pressure, suctioning the endotracheal tube, checking the circuit, hand ventilating, and confirming decreased breath sounds bilaterally, albuterol and dexamethasone were given with mild improvement in ventilation.

Shortly after this event, the surgical procedure was completed. The patient resumed spontaneous respirations with a rate of 20 breaths/min and tidal volumes of 120 mL. Neuromuscular blockade was reversed, and he awakened and followed commands. When he demonstrated head lift for five seconds, the patient was extubated. However, he had minimal respiratory effort right after extubation, and the drapes were removed to examine for chest rise. The abdomen was noted to be severely distended and tympanic to percussion. A rapid-sequence induction was performed, but placement of the endotracheal tube was more difficult due to swelling around the vocal cords. After endotracheal intubation, the patient became hemodynamically unstable with arterial blood pressure decreasing to the 70s/40s. A radial arterial line was placed and phenylephrine and ephedrine boluses were given to maintain a mean arterial blood pressure above 60 mm Hg. The continuous bladder irrigation, which is customary after TURP procedures, was discontinued. Upon stopping the irrigation, tidal volumes improved to 350 mL.

Due to the lack of space in the cystoscopy suite, the patient was transported next door to the trauma operating room for exploratory laparotomy. An arterial blood gas at that time showed a combined metabolic and respiratory acidosis (pH 7.08, pCO_2 66, HCO_3^- 19, sodium 126, base excess −10). Upon incision, 3 L of clear irrigation fluid were suctioned out of the peritoneum. Laparotomy revealed a bladder neck rupture with extraperitoneal fluid collection, with a second tear in the peritoneum leading to the massive intraperitoneal fluid collection. Immediately after the abdominal fluid was removed, airway peak pressures returned to baseline. However, the patient developed hypotension and required a norepinephrine infusion

to maintain hemodynamic stability. After decompression of the abdomen, the pH normalized within 1 h. The surgeons performed a repair of the bladder rupture, open suprapubic prostatectomy, and placement of a suprapubic catheter.

The patient remained intubated after the procedure and was transported to the surgical intensive care unit. He was extubated the following day and was discharged home on postoperative day 5 with an indwelling Foley catheter. At his follow-up visit one month after surgery, the Foley catheter was removed and the patient continued with a good recovery.

Discussion

Our patient developed abdominal compartment syndrome (ACS) caused by extra- and intraperitoneal bladder rupture during TURP. ACS after TURP is a rare occurrence previously described just once in the literature.[7]

An increase in peak airway pressures was noted 2 h into the procedure, which we initially attributed to reactive airway disease. Of note, this patient's prostate was approximately 120 g, and our urology team does not routinely perform TURP for prostate size greater than 80 g. However, the purpose of this patient's surgery was to offer relief from obstructive uropathy and to avoid major abdominal surgery. Due to the abnormally large prostate and prolonged duration of the resection (greater than 2½ h), as well as the pressurized bladder irrigation used during and immediately after the procedure, our patient quickly developed a distended abdomen which lead to ACS and respiratory distress immediately after extubation. His treatment included discontinuation of the irrigation solution and rapid exploratory laparotomy to evacuate the fluid and repair the bladder injury.

Opening the abdomen is the most effective method to reduce IAP and is the treatment of choice for abdominal compartment syndrome when IAP is constantly higher than 30 mm Hg with ongoing organ failure refractory to medical therapy.[8] Due to our patient's hemodynamic instability and quickly worsening clinical picture, his IAP was never measured but the relief after laparotomy incision was instant. His pre-incision pH was 7.08 and it quickly normalized by the end of the surgery.

An additional challenge our team faced was that the cystoscopy suite is small and not suited for an urgent open abdominal exploration. Therefore, our patient had to be moved next door to the trauma operating room. We were fortunate that this complication occurred in a trauma hospital with an immediately available operating room and in-house trauma surgeons. TURP commonly takes place in ambulatory surgical centers where emergency assistance may be limited. Although bladder rupture is a rare complication, appropriate measures for its management should be considered.

Since this event, the operating room team in the cystoscopy suite has made it a point to monitor the abdomen for distention during prolonged cystoscopy procedures, and to assess the abdomen visually with the operating room lights on before extubation. The irrigation fluid input and output are also collected and measured. Another option would be to do an ultrasound examination of the abdomen in the cystoscopy suite in cases where symptoms suspicious for bladder rupture present. This would avoid doing an unnecessary laparotomy in patients where the suspicion is there, but who remain hemodynamically stable. This case has reminded us to have bladder rupture high on the list of differential diagnoses when a case of unexpected increased airway pressures occurs during cystoscopy.

In the presented case, our patient developed acute abdominal compartment syndrome after extraperitoneal bladder rupture and subsequent intraperitoneal leak during TURP. His hemodynamic instability and difficulty with ventilation led to the decision to perform emergent exploratory laparotomy in an adjacent operating room. Quick recognition and intervention after this complication prevented long term morbidity for this patient.

Conflicts of interest

The author declares no conflicts of interest.

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