J Anesth (2010) 24:280–283
DOI 10.1007/s00540-010-0882-9

CLINICAL REPORT

Abdominal compartment syndrome occurring due to uterine perforation during a hysteroscopy procedure

Kyu Chang Lee · Hye Young Kim · Myeong Jong Lee · Jai Won Koo · Jeong Ae Lim · Seong Hyop Kim

Received: 1 September 2009 / Accepted: 6 December 2009 / Published online: 3 March 2010

Abstract We report a case of abdominal compartment syndrome occurring due to uterine perforation while the patient was undergoing hysteroscopic uterine adhesiolysis for uterine synechia. The cause of the patient's abdominal compartment syndrome was irrigation fluid moving from the uterine cavity into the peritoneal space via defects in the uterus. Anesthesiologists must be alert for these complications during hysteroscopy procedures.

Keywords Abdominal compartment syndrome · Hysteroscopy · Uterine perforation

Introduction

Operative hysteroscopy is a safe and minimally invasive procedure with few major complications reported. Fluid volume overload, uterine perforation, hemorrhage, bowel and urinary tract injuries, and gas embolization have occurred in fewer than 1% of procedures according to one survey [1, 2]. The most frequent reported complication was uterine perforation, but this did not always cause significant problems [3]. However, we present a life-threatening case of abdominal compartment syndrome resulting from uterine perforation.

K. C. Lee · H. Y. Kim (✉) · M. J. Lee · J. W. Koo
Department of Anesthesiology and Pain Medicine, Konkuk University Chungju-Hospital, Kyohyun 2-dong, Chungju-si, Chungbuk-do 380-704, Republic of Korea
e-mail: hae1127@kku.ac.kr

J. A. Lim · S. H. Kim
Department of Anesthesiology and Pain Medicine, Konkuk University Seoul-Hospital, Seoul, Republic of Korea

Case report

A 36-year-old woman weighing 54 kg and standing 162 cm tall, with a history of infertility and uterine synechia, planned to have a hysteroscopic procedure. Her past medical history included anaphylaxis due to nonsteroidal anti-inflammatory drugs (NSAIDs). A preoperative investigation was unremarkable.

Anesthesia was induced with propofol, succinylcholine, and rocuronium, and a laryngeal mask airway (LMA; size 4) was inserted. Anesthesia was done with nitrous oxide (50%) and desflurane (4%–6%). The patient was placed in the lithotomy position with a slight Trendelenburg tilt. Fifty minutes after the induction of general anesthesia, the procedure was started. The patient was hemodynamically stable.

Fifty minutes after the surgery began, peak inspiratory airway pressure had increased from 17 to 24 cmH_2O. End-tidal CO_2 had increased from 33 to 40 mmHg and the peripheral oxygen saturation (SpO_2) had decreased to 95%. On auscultation, breath sounds were diminished over the entire chest. The tidal volume and respiratory rate was reset in an attempt to improve the progressively worsening oxygenation and increasing peak inspiratory pressure. The fractional inspired oxygen (FiO_2)was increased to 1.0, and the SpO_2 to 99%. Over the next few minutes, the peak inspiratory pressure increased to more than 35 cmH_2O, up from a baseline of 17 cmH_2O. An examination of accessible skin revealed flushing of the neck and upper torso. While the FiO_2 was 1.0, the SpO_2 decreased to 96%. An anaphylactic or anaphylactoid reaction was suspected, and epinephrine 100 μg and dexamethasone 5 mg were given intravenously.

The patient was intubated with a tracheal tube (internal diameter [ID], 7.0) immediately after the LMA was

removed. However, the patient was not ventilated by manual bagging. At the same time, high airway pressure and absence of chest wall movement were noted, and end-tidal CO_2 was not checked. Despite visual confirmation of correct placement of the tracheal tube, oxygen desaturation developed rapidly. On auscultation, breathing sounds were not present over the entire chest. The peripheral blood oxygen saturation fell to 20%. The patient had cyanosis and petechia. The surgeons were informed that ventilation was becoming progressively more difficult, but a cause of the problem could not be provided. The patient's blood pressure decreased from 120/80 to 70/30 mmHg. A catheter was placed in the radial artery for continuous measurement of arterial pressure and an additional venous catheter was inserted. Intravenous fluid flow was opened to gravity and epinephrine (100 μg) was given intravenously. Epinephrine (700 μg) was then repeatedly given, which increased her blood pressure only slightly, to 90/50 mmHg. Dopamine infusion was started, and titrated to maintain systolic blood pressure at greater than 90 mmHg. The patient's blood pressure was maintained at 100–140/50–90 mmHg and heart rate at 100–120 beats min^{-1}. Urinary outflow decreased gradually, leading to total anuria. Additionally, furosemide (20 mg) was given intravenously, but this was without effect. Furosemide (40 mg) was then given. Dexamethasone 8 mg, methylprednisolone 250 mg, and 4 mg chlorpheniramine were given intravenously.

Shortly after these intravenous injections, an extremely tense and distended abdomen was detected. The surgeons reported a probable perforation in the uterus. By then, nobody was aware of the in–out imbalance of the irrigation fluid. A decision was made to perform a laparoscopy. The laparoscopy was started and 8000 ml of clear fluid was drained from the peritoneal cavity.

Ten minutes after the laparoscopic drainage, the patient was ventilated gradually by manual bagging. SpO_2 increased to 80% and end-tidal CO_2 was 5–20 mmHg. The duration that the patient was hypoxic was about 20 min.

During the drainage, arterial blood gas analysis showed a pH of 7.130, a carbon dioxide tension ($PaCO_2$) of 37.7 mmHg, and an oxygen tension (PaO_2) of 83.9 mmHg. Serum electrolytes showed Na^+ of 123 mmol L^{-1}, K^+ of 2.7 mmol L^{-1}, and Cl^- of 93 mmol L^{-1}; 3% NaCl and 40 mmol KCl were infused to correct the electrolyte imbalance.

A total of 18000 ml of irrigation fluid [Urosol (CJ Jeiljedang, Hwasung, Korea); D-sorbitol, 2.7 g 100 ml^{-1}; D-mannitol, 0.54 g 100 ml^{-1}] was used for irrigation, and 8000 ml was recovered in the peritoneal space. The operation lasted for 2.2 h. The estimated blood loss was 100–150 ml. At the end of the surgery, blood pressure was 130/80 mmHg; heart rate, 100 beats min^{-1}; and SpO_2, 95%–96% ($FiO_2 = 1.0$). Peak inspiratory pressure decreased to 25–32 cmH_2O, and urine began to flow. The patient had a pupillary light reflex and showed response to pain.

On arrival in the intensive care unit, her SpO_2 was 95% ($FiO_2 = 1.0$). Lung ventilation was performed with biphasic positive airway pressure (high/low positive end-expiratory pressure [PEEP], 19/13 mmHg; respiratory rate, 18 beats min^{-1}; pressure support, 12 mmHg). A chest and abdomen radiograph showed a pleural effusion and multiple air-fluid levels.

Two days after the surgery, arterial blood gas analysis showed a pH of 7.388, a $PaCO_2$ of 39.3 mmHg, and a PaO_2 of 105.3 mmHg while the patient was breathing oxygen at 3 L min^{-1} from a nasal cannula, and serum electrolytes were within the normal range.

The patient was discharged from the hospital 7 days after the surgery without postoperative complications.

Discussion

A hysteroscopy requires the insertion of a hysteroscope into the uterine cavity and the installation of suitable distension media for visualization of the endometrium. The irrigation fluid is thought to be absorbed via open myometrial vesicles or through transtubal loss with rapid peritoneal absorption. Uterine perforation can provide another route by which the distension media can gain intraperitoneal access [4]. Our present report describes an unusual and serious complication, of abdominal compartment syndrome (ACS), due to uterine perforation.

Compartment syndrome is a condition in which there is increased pressure in a confined anatomical space. Compartment syndrome is well recognized in the fascial spaces of the extremities, the orbital globe (glaucoma), the intracranial cavity (epidural or subdural hematoma), the pleura, pericardium, and the kidney. Because the contents of the abdomen are contained within the confined space of the abdominal cavity, any increase in the volume of the abdominal contents will result in an increase in intraabdominal pressure (IAP).

Abdominal compartment syndrome causes significant dysfunction of almost all organ systems. An increase in IAP causes a gradual derangement in the physiology of many organs. This may adversely affect pulmonary, cardiovascular, renal, splanchnic, and neurological function. The increased IAP in ACS decreases lung compliance and raises peak airway pressure during mechanical ventilation, resulting in hypoxemia and hypercapnia. It also decreases venous return, reducing cardiac output and the perfusion of abdominal organs, causing oliguria and splanchnic ischemia. Decreased venous return may cause raised intracranial pressure, leading to brain death [5, 6].

Several means of measuring IAP have been reported, such as through the inferior vena cava, stomach, rectum, vagina, or urinary bladder, but measurement of IAP using the bladder is the current mainstay of diagnosis [6]. Normal IAP is less than 0 mmHg. Although an IAP of more than 20 mmHg is clinically significant, recent studies demonstrate that even at a relatively low IAP of 10–15 mmHg significant alterations in organ function can be seen [7, 8]. We did not monitor IAP, but it is suggested that an IAP of more than 30 mmHg results in markers of physiologic deterioration such as an extremely tense and distended abdomen, respiratory failure, and anuria [9]. In a therapeutic approach according to the level of IAP, abdominal decompression is recommended for IAP of more than 25 mmHg [10, 11]. In the case presented above, laparoscopic drainage (decompressive laparoscopy) was performed and this resulted in immediate improvement.

At an early stage of the surgery in the present patient, we suspected that the cause of the increased airway pressure and hypoxia was airway obstruction arising from an anaphylactic or anaphylactoid reaction. Tracheal intubation was performed after LMA removal for safety and intravenous drugs were administered. But the patient was not ventilated and her SpO_2 decreased. Before decompressive laparoscopy was started, lung ventilation showed decreasing tidal volume and increasing respiratory rate, and applying PEEP to reset these parameters was done in an attempt to improve progressively the patient's worsening oxygenation and increasing peak inspiratory pressure. The diagnosis was delayed because of the patient's history of NSAID anaphylaxis and the lack of close communication between anesthesiologists and surgeons. During decompression, slow attenuation of the hypoxia was observed.

During the procedure, our patient showed hemodynamic values that were stably maintained, but compensatory tachycardia was observed in response to the decrease in stroke volume. In ACS, the causes of hemodynamic compromise are alterations in preload, afterload, and intrathoracic pressure [6, 12]. These changes, which are caused by increased IAP, are exacerbated by hypovolemia [7]. Therefore, volume resuscitation is useful to improve cardiac output and stroke volume.

In our patient, anuria and hyponatremia occurred. Hypotonic, electrolyte-free irrigation fluid for distension of the uterine cavity has the potential to be absorbed in volumes large enough to cause hyponatremia [13, 14]. Decreases in serum sodium were reported to be proportional to the amount of fluid absorbed [15]. Also, renin activity and aldosterone levels increased when the IAP was more than 30 mmHg. These changes were associated with anuria, considerably reduced renal blood flow, and increased renal vascular resistance [16, 17]. Treatment was directed at maintenance of urine output and correction of the electrolyte imbalance. Furosemide, dopamine, and hypertonic saline were given.

We have described herein ACS as a complication of hysteroscopic surgery in a healthy woman. Patients with ACS typically present with a tense distended abdomen, increased peak inspiratory airway pressures, severe hypercapnia, hypotension, and oliguria [18]. Anesthesiologists should ascertain any new developments in the patient's condition. Fortunately, our patient's recovery was uneventful with standard management. A favorable outcome for patients with established ACS requires timely recognition and treatment, as many of the adverse effects of ACS are reversible if the IAP is promptly decreased.

During a hysteroscopy procedure, the anesthesiologist and surgeons should be aware of ACS that may have occurred due to uterine perforation that was diagnosed late.

References

1. Jansen FW, Vredevoogd CB, van Ulzen K, Hermans J, Trimbos JB, Trimbos-Kemper TC. Complications of hysteroscopy: a prospective, multicenter study. Obstet Gynecol. 2000;96:266–70.
2. Paschopoulos M, Polyzos NP, Lavasidis LG, Vrekoussis T, Dalkalitsis N, Paraskevaidis E. Safety issues of hysteroscopic surgery. Ann N Y Acad Sci. 2006;1092:229–34.
3. Hulka JF, Peterson HA, Phillips JM, Surrey MW. Operative hysteroscopy: American Association of Gynecologic Laparoscopists' 1993 membership survey. J Am Assoc Gynecol Laparosc. 1995;2:131–2.
4. Gallagher ML, Roberts-Fox M. Respiratory and circulatory compromise associated with acute hydrothorax during operative hysteroscopy. Anesthesiology. 1993;79:1129–31.
5. An G, West MA. Abdominal compartment syndrome: a concise clinical review. Crit Care Med. 2008;36:1304–10.
6. Deenichin GP. Abdominal compartment syndrome. Surg Today. 2008;38:5–19.
7. Ridings PC, Bloomfield GL, Blocher CR, Sugerman HJ. Cardiopulmonary effects of raised intra-abdominal pressure before and after intravascular volume expansion. J Trauma. 1995;39:1071–5.
8. Bongard F, Pianim N, Dubecz S, Klein SR. Adverse consequences of increased intra-abdominal pressure on bowel tissue oxygen. J Trauma. 1995;39:519–24.
9. Nathens AB, Brenneman FD, Boulanger BR. The abdominal compartment syndrome. Can J Surg. 1997;40:254–8.
10. Kron IL, Harman PK, Nolan SP. The measurement of intraabdominal pressure as criterion for abdominal re-exploration. Ann Surg. 1984;199:28–30.
11. Ivatury RR, Porter JM, Simon RJ, Islam S, John R, Stahl WM. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma. 1998;44:1016–21.
12. Malbrain MLNG. Is it wise not to think about intraabdominal hypertension in the ICU? Curr Opin Crit Care. 2004;10:132–45.
13. Olsson J, Hahn RG. Stimulated intraperitoneal absorption of irrigation fluid. Acta Obstet Gynecol Scand. 1995;74:707–13.
14. Hahn RG. Irrigation fluids in endoscopic surgery. Br J Urol. 1997;79:669–80.

15. Baumann R, Magos AL, Kay JD, Turnbull AC. Absorption of glycine irrigating solution during transcervical resection of endometrium. BMJ. 1990;300:304–5.
16. Tal R, Lask DM, Keslin J, Livne PM. Abdominal compartment syndrome: urological aspects. BJU Int. 2004;93:474–7.
17. Gudmundsson FF, Gislason HG, Myking OL, Viste A, Grong K, Svanes K. Hormonal changes related to reduced renal blood flow and low urine output under prolonged increased intra-abdominal pressure in pigs. Eur J Surg. 2002;168:178–86.
18. Hunter JD, Damani Z. Intra-abdominal hypertension and the abdominal compartment syndrome. Anaesthesia. 2004;59:899–907.