Raising Awareness of Abdominal Compartment Syndrome after Gynecologic-Oncology Surgery

Jinekolojik-Onkoloji Cerrahisi Sonrası Abdominal Kompartman Sendromu Farkındalığını Artırma

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ABSTRACT

A 56 years old female, body mass index 44 kg/m^2, postmenopausal patient who had a solid mass of approximately 6-cm in diameter at the left adnexal area underwent laparotomy with vertical incision. Staging surgery was performed because of the frozen section result that was reported as a malignant ovarian cancer. On the postoperative 2nd day, the patient had dyspnea, cold sweats, tachycardia, tachypnea and bowel sounds were detected as hypoactive. SO_2 was measured as 88% during follow-up and right costophrenic angle was appearing blunt in her chest X-ray. Arterial blood gas analysis results were as follows; ph: 7.41, PCO_2: 29.2 mmHg, SO_2: 86%, HCO_3: 18.2 mmol/L, and base excess (BE): -4.9 mmol/L. There was no additional feature in the blood cell counts and biochemical tests. The diagnosis of pulmonary emboli was ruled out with thoracic spiral tomography. In the sequel, the patient began vomiting, while her bowel sounds were remained hypoactive with addition of abdominal distension. After the initial intravesical pressure (IVP) was measured as 16 mmHg (22 cmH_2O) manually, the patient was observed with endoluminal decompression methods and medical management. The clinical signs and physical examination findings did not improve; IVP measured two hours later was 21 mmHg (28 cmH_2O). Because of these clinical findings and the rise of sequential IVP measurements, the patient was diagnosed with abdominal compartment syndrome. Decompressive laparotomy was performed and completed by closing the skin without suturing the fascia. Eight months later, her overall condition was good and follow-up has continued. Monitoring intraabdominal pressure with intermittent indirect IVP measurements in intensive care patients with high risk for ACS has great significance for early diagnosis, increasing the awareness for this condition. Early decompressive surgery in ACS is the life-saving step.

Key Words: Abdominal compartment syndrome, intraabdominal hypertension, intravesical pressure measurement

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ÖZET

Elli altı yaşında, vücut kitle indeksi 44 kg/m^2 olan sol adneksiyel alanda yaklaşık 6 cm solid kitleye sahip postmenopozal hastaya vertikal insizyon ile yapılan laparotomide intraoperatif patoloji sonucunun malign over tümörü olarak rapor edilmesi üzerine hastaya evrelendirici cerrahi uygulandı. Postoperatif 2. Günde, hastada dispne, soğuk terleme, taşikardi, taşipne gelişmiş ve barsak sesleri hipoaktif olarak saptandı. Takipte SO_2 %88'di ve akciğer direk grafide sağ kostafrenik alan künttü. Arteriyel kan gazı sonucunda ph: 7.41, PCO_2: 29.2 mmHg, SO_2: % 86, HCO_3: 18.2 mmol/L, ve baz açığı: -4.9 mmol/L idi. Spiral tomografi ile pulmoner emboli tanısı dışlandı. Hastanın mevcut bulgularına kusma ve abdominal distansiyon eklendi. Başlangıç intravezikal basıncın (İVB) manuel olarak 16 mmHg (22 cmH_2O) saptanması üzerine hasta endolüminal dekompresyon metodları ve medikal yönetim ile takip edildi. Klinik bulgularda ve fizik muayenede gerileme olmaması üzerine 2 saat sonra ölçülen İVB 21 mmHg (28 cmH_2O) idi. Hastaya abdominal kompartman sendromu tanısıyla dekompresif laparatomi uygulandı ve fasya açık bırakılıp sadece cilt onarımı ile operasyon tamamlandı. Hasta postoperatif 8. ayda hastalıksız olarak yaşamına devam etmekteydi. AKS açısından yüksek riske sahip yoğun bakım hastalarında aralıklı indirekt İVB ölçümü ile intraabdominal basınç monitorizasyonu AKS farkındalığını arttırma ve erken tanı açısından büyük öneme sahiptir. AKS'de erken dekompresif cerrahi hayat kurtarıcı basamaktır.

Anahtar Sözcükler: Abdominal kompartman sendromu, intraabdominal hipertansiyon, intravezikal basınç ölçümü

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INTRODUCTION

Abdominal compartment syndrome (ACS) is a progressive clinical condition characterized by the persistent increase in intraabdominal pressure (IAP) that may result in sepsis and even multiple organ failure. The increased intraabdominal pressure causes reduced venous return and cardiac output resulting in end-organ hypoperfusion and ischemia, and intestinal ischemia leads to translocation of microorganisms, giving rise to sepsis (1). The World Society of the Abdominal Compartment Syndrome (WSACS) defines intraabdominal hypertension (IAH) as sustained or repeated pathologic elevation of IAP ≥12 mmHg, and ACS as a sustained IAP >20 mmHg that is associated with new organ dysfunction (2). IAH is categorized as follows: Grade I: IAP 12-15 mmHg; Grade II: IAP 16-20 mmHg; Grade III: IAP 21- 25 mmHg; and Grade IV: IAP >25 mmHg (3). Diagnosis is not made upon a single measurement and successive measurements are required. In contrast to IAH, ACS is not graded.

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Clinical manifestations of ACS include abdominal distention, abdominal rigidity, tachypnea or hyperpnea, loss of diaphragm movements, hypotension, tachycardia, weak or thin pulse, oliguria or anuria, cold extremities, flat neck veins, and altered consciousness (1).

The incidences of IAH and ACS among general intensive care patients are 25% (range: 10-32%) and 4%, respectively (4, 5). The incidence of IAH among surgical intensive care patients reaches 65% (6). In this article, we present a case with abdominal compartment syndrome that developed after abdominal surgery performed for staging ovarian carcinoma.

CASE REPORT

A 56 years old postmenopausal patient presenting with abdominal pain had goiter and diabetes mellitus in her medical history. Her body weight was 120 kg, yielding a body mass index (BMI) of 44 kg/m^2. Upon physical examination and imaging studies, a solid mass of approximately 6-cm in diameter was detected at the left adnexal area. Tumor markers were elevated (CA125: 497 IU/ml, CA19-9: 162 IU/ml). Due to an initial diagnosis of pelvic mass, laparotomy was performed with a vertical incision. Intraoperative inspection revealed diffuse tumor dissemination and frozen section examination showed an ovarian malignant epithelial tumor. The operation was completed with type 2 hysterectomy, bilateral salpingo-oophorectomy, retroperitoneal lymph node dissection, rectosigmoid resection, low rectal anastomosis, pelvic peritonectomy, total omentectomy, splenectomy, diaphragm stripping and loop colostomy, and placement of two soft drains to the abdominal cavity. Prophylaxis for venous thromboemboli was provided with enoxaparin and compression stockings. The patient had stable hemodynamics [heart rate (HR) 86 bpm, blood pressure (BP) 120/70 mmHg, body temperature 36.7°C, oxygen saturation with pulse oximetry (SO_2) 97%, respiratory rate (RR) 16/min, urinary output (UO):100cc/hr] and was mobilized on postoperative 1st day. There was gas passage through the colostomy once. On postoperative 2nd day, the patient had dyspnea and cold sweats. In a physical examination, she had tachycardia and tachypnea, and bowel sounds were detected as hypoactive (HR: 120 bpm, BP: 120/80 mmHg, RR: 22/min, respiratory sounds normal, normoglycemic). The patient was admitted to intensive care unit. SO_2 was measured 88% during follow-up and right costophrenic angle was appearing blunt in her chest X-ray. Arterial blood gas analysis results were as follows; ph: 7.41, PCO_2: 29.2 mmHg, SO_2: 86%, HCO_3: 18.2 mmol/L, and base excess (BE): -4.9 mmol/L. There was no additional feature in the blood cell counts and clinical biochemical tests. The diagnosis of pulmonary emboli was ruled out with thoracic spiral tomography. Adequate intravenous fluid resuscitation and oxygen support was provided to the patient. In the sequel, the patient began vomiting, while her bowel sounds remained hypoactive with the addition of abdominal distension. Her oral intake was stopped, and a urinary catheter was placed (>100cc/hr). The fluid coming out of the abdominal drains was serous in appearance. Colostomy tract was open and abdominal x-ray was normal. In order to measure intravesical pressure (IVP) manually,(1) i.v. infusion set was filled with physiological serum completely. Zero point of the ruler (30 cm) was placed on the level of superior iliac crest, while the patient was in the supine position (central venous catheter manometer can also be used). Then, the bladder was filled with maximum 25 cc physiological serum (SF), and the urinary catheter was clamped. Next, the needle of the i.v. infusion set was inserted into the urinary catheter above the clamp. During late expiration, the infusion set was cut close to the pouch, and the fluid was allowed to drain off through gravity. The point it stabilized was noted as cmH_2O (1 mmHg= 1.36 cmH_2O). The initial IVP was measured as 16 mmHg (22 cmH_2O) and the nasogastric catheter was placed. Metoclopramide was administered intravenously with six hour intervals. The colostomy opening and rectum were touched at intervals to provide endoluminal decompression. Although there was no change in control arterial blood gas and laboratory analysis; the patient's dyspnea, tachypnea, tachycardia, and vomiting continued, and physical examination findings did not improve; IVP that measured two hours later was 21 mmHg (28 cmH_2O). The patient was diagnosed with abdominal compartment syndrome, and decompressive laparotomy was performed. During the operation, there was minimal clear-appearing fluid in the abdominal cavity. The fluid was sampled for culture. All intestinal loops were dilated and filled the abdominal cavity completely. No obstruction, fistula or leakage of anastomosis was detected. The colostomy tract was open. The abdominal drains were replaced with new ones and decompression laparotomy was completed by closing the skin with mattress sutures without suturing the fascia. Antibiotic therapy was started intra-operatively. Endoluminal decompression was applied via the nasogastric catheter and by touch of colostomy opening and rectum. Adequate intravenous fluid resuscitation was provided, metoclopramide was continued as a prokinetic agent and total parenteral nutrition was administered.

During follow-up, the severity of dyspnea, tachypnea, tachycardia, and abdominal distention signs declined. IVP monitorization was started at postoperative 2nd hour and continued with four hour intervals during the first 24 hours. IVP declined to 12 mmHg at postoperative 18th hour. Gas passage through colostomy started postoperative on the 1st day and stool passage started on the 3rd day. There was no bacterial growth in the culture of intraabdominal fluid. All of her abnormal signs improved on the 5th day and enteral feeding was initiated. She made a full recovery on the postoperative 8th day. The patient completed six cycles of platinum-based chemotherapy during the next four months. After three months from completion of the chemotherapy, the loop colostomy was closed. Her overall condition was good, and follow-up has continued.

DISCUSSION

The mortality rates of IAH and ACS are 50-75%, which can be reduced down to 34-37% with early diagnosis and the appropriate management (7, 8). IAH/ACS may develop both in medical and surgical intensive care patients. Most common causes among adults include intraabdominal trauma, bleeding, and major abdominal surgery (9). Nevertheless, there are numerous etiologies and risk factors that include non-surgical and even iatrogenic causes (Table 1) (2, 6). Although clinical suspicion is important for diagnosis, symptoms in ACS do not have typical features. Therefore, sensitivity of clinical evaluation alone is quite low (40-56%) (10). The simple gold-standard method for diagnosis is intravesical pressure (IVP) measurement that recommended by WSACS, on condition that bladder tumors and other conditions which create bladder compression are ruled out (11). The gastric and rectal pressure measurements can also be used for diagnosis, but they are less reliable than IVP (12). Although indications for measurement of IAP vary, current guidelines recommend IVP measurement for all critically ill or injured patients who have one or more risk factors (Table 1) for the development of IAH. Measurements should be performed every 4-6 hours until risk factors are resolved and IAP remains normal for 24-48 hours (2, 3). Silva et al. emphasized that IAP should be measured routinely in intensive care units (13).

The main principle of its management is the determination of the underlying cause. Medical management is started when IAP is greater than 12 mmHg. In its medical management; enteral nutrition should be minimized or stopped, excessive fluid resuscitation should be avoided, pain should be controlled with analgesia and sedation, and patient's supine position should not exceed an angle of 30° (2). Implementation of nasogastric or rectal tubes are not routinely recommended in all surgical patients; but if there is dilatation of stomach or intestines, those should be used for enteral decompression (2, 14). Use of promotility agents such as erythromycin and metoclopramide, is beneficial in the presence of paralytic ileus (3). Neuromuscular blocking agents such as neostigmine may be effective in treatment, because they induce colonic decompression and reduce the abdominal muscular tone; therefore, they decrease the abdominal compliance (2). Paracentesis and percutaneous drainage should be considered in the presence of intraabdominal fluid (1). Decompressive laparotomy (DL) is required in the presence of IAH that are refractory to medical therapy or ACS (3). DL reduces IAP immediately and improves the organ functions; however, the possibility of reperfusion damage should be kept in mind (15). Management after DL is very important. Planned incisional hernia that involves leaving the fascial defect open and closure of the skin only, is usually the safest solution if it is applicable without leading to an increase in IAP (16). Open abdomen techniques (OAT) can be used if it is thought that IAP would not be reduced (2). Bogota bag, Wittman patch, absorbable fascial meshes and modified vacuum pack sandwich technique are the most common OAT's (17). If not managed properly, serious complications can develop after OAT's, such as hemodynamic instability and hypothermia due to fluid evaporation, entero-atmospheric fistulae, sepsis due to contamination, erosion at intestinal wall, bleeding and loss of intestinal functions (2, 17).

The present case had three risk factors associated with ACS; obesity, history of major surgery and an elderly age. In our case, ACS was managed with DL which was terminated by closing the skin and leaving the fascia open that was defined as planned incisional hernia in literature. After secondary closure; medical management included adequate fluid resuscitation with avoiding positive balance, use of promotility agents such as metoclopramide, nasogastric tube insertion, intermittent rectal/colonic decompression, and percutaneous drain, while monitoring IVP every four hours.

Table 1: Risk factors for intra-abdominal hypertension and abdominal compartment syndrome

Diminished abdominal wall compliance
• Abdominal surgery [a]
• Major trauma [a]
• Major burns
• Prone positioning [a]
Increased intra-luminal contents
• Gastroparesis, gastric distention [a]
• İleus (mechanic or paralytic) [a]
• Colonic pseudo-obstruction
• Volvulus
Increased intraabdominal contents
• Acute pancreatitis [a]
• Distended abdomen
• Hemoperitoneum/pneumoperitoneum or intra-peritoneal fluid collection [a]
• Intraabdominal or retroperitoneal tumors
• Intraabdominal infection/abscess [a]
• Laparoscopy with excessive insufflation pressures
• Liver dysfunction/cirrhosis with ascites
• Peritoneal dialysis
Capillary leak/fluid resuscitation
• Acidosis [a]
• Damage control laparotomy
• Hypothermia [a]
• Increased APACHE-II or SOFA score [a]
• Massive fluid resuscitation or positive fluid balance [a]
• Polytransfusion [a]
Others
• Age[a]
• Sepsis/Bacteremia[a]
• Coagulopathy
• Increases head of bed angle (higher than 30^o) [a]
• Massive incisional hernia repair
• Mechanical ventilation [a]
• Obesity or increased body mass index [a]
• PEEP>10 cmH_2O [a]
• Peritonitis
• Pneumonia
• Shock or hypotension [a]

APACHE-II; acute physiology and chronic health evaluation-II
SOFA; sequential organ failure assessment
PEEP; positive end expiratory pressure
[a] indicates primary literature support

CONCLUSION

Early diagnosis helps to reduce morbidity and mortality significantly in IAH and ACS. Since sensitivity of clinical evaluation is low, it is thought that monitoring IAP with intermittent indirect IVP measurements in intensive care patients with high risk for ACS has great significance for early diagnosis, increasing the awareness for this condition. Early decompressive surgery in ACS is the life-saving step. Due to high complications rates, morbidity and mortality observed with open abdomen techniques; it is a convenient option to use the method involving closure of the skin only with leaving fascia open. After this implementation, close and attentive post-surgery medical management should be performed and IVP should be measured once in every four hours or continuously.

Conflict of interest
No conflict of interest was declared by the authors

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