pISSN 2233-7903 · eISSN 2093-0488

### CASE REPORT

# A rare reason of abdominal compartment syndrome: non-Hodgkin lymphoma

Mehmet Abdussamet Bozkurt, Kaplan Baha Temizgönül, Osman Köneş, Halil Alış

Department of General Surgery, Bakırköy Dr.Sadi Konuk Training and Research Hospital, İstanbul, Turkey

Abdominal compartment syndrome (ACS) is characterized by intra-abdominal hypertension (IAH) which affects all body systems. In healthy individuals, normal intra-abdominal pressure (IAP) is <5 to 7 mmHg. The upper limit of IAP is generally accepted to be 12 mmHg. ACS has been classified into primary, secondary, and tertiary subtypes. Non-Hodgkin lymphoma (NHL) is a rare reason for ACS. We report here one case of NHL as a primary retroperitoneal mass in an 80-year-old male patient who presented with IAH.

Key Words: Abdominal hypertension, Non-Hodgkin lymphoma

# INTRODUCTION

Abdominal compartment syndrome (ACS) is characterized by intra-abdominal hypertension (IAH), elevation and splinting of the diaphragm, high pleural pressure, and poor venous return to the heart, producing low cardiac output and shock which, in turn, results in poor venous return across the capillaries setting a vicious cycle. ACS and IAH affect all body systems, most notably the circulatory, respiratory, urinary and nervous [1].

In 2006, The World Society of the Abdominal Compartment Syndrome (WSACS) established consensus definitions for IAH and for ACS: intra-abdominal pressure (IAP) is defined as the steady-state pressure concealed within the abdominal cavity, while ACS is defined as a sus-

tained IAP > 20 mmHg (with or without abdominal perfusion pressure < 60 mmHg) that is associated with a new organ dysfunction/failure. In healthy individuals, normal IAP is < 5 to 7 mmHg [2].

The upper limit of IAP is generally accepted to be 12 mmHg by the WSACS, including the expected increase in normal pressure from clinical conditions that exert external pressure to the peritoneal envelope or diaphragm; this includes obesity and chronic obstructive pulmonary disease. In contrast, IAH is defined as a sustained or repeated pathologic increase in IAP >12 mmHg [3].

Pre-existing comorbidities, such as chronic renal failure, pulmonary disease, or cardiomyopathy, play an important role in aggravating the effects of elevated IAP and may reduce the threshold of IAH that causes clinical mani-

Received May 3, 2012, Revised June 4, 2012, Accepted June 17, 2012

Correspondence to: Mehmet Abdussamet Bozkurt

Department of General Surgery, Bakırköy Dr.Sadi Konuk Training and Research Hospital, Tatlınar Street 8, 5 Zuratbaba, Bakırköy, İstanbul, Turkey

 $\label{eq:tel:eq:tel:energy} \textbf{Tel:} + 905325791673, \textbf{Fax:} + 002212524491, \textbf{E-mail:} \ \textbf{msametbozkurt@yahoo.com}$ 

© Journal of the Korean Surgical Society is an Open Access Journal. All articles are distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0/) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

festations of ACS. Chronically increased IAP can give time for the body to accommodate increased IAP and can be achieve without developing ACS [4].

Increased IAP impairs venous return and arterial inflow. Abdominal decompression is essential in its management, but ischemia-reperfusion injury may occur as a complication of decompression [5].

ACS has been classified into primary, secondary, and tertiary subtypes. Primary ACS is contracted or occurs trauma within the abdominopelvic region such as blunt and penetrating trauma, hemorrhage from ruptured abdominal aortic aneurysm, and closure of the abdomen under tension, postoperatively. Secondary ACS occurs in diseases remote from the abdomen, such as massive fluid resuscitation in burns and severe acute pancreatitis.

Non-Hodgkin lymphomas (NHL) in nearly 70% of cases present with a generally multi-localized, single or multiple lymphadenomegaly without pain, with involvement in almost 30 to 40% of latero-cervical lymph nodes. In approximately 30% of cases, extra-nodal sites are involved including the female genital tract [6]. Retroperitoneal presentation is extremely rare and because of the uncommon anatomic location, the diagnosis and subsequent management of these patients tend to be difficult.

We report here one case of NHL as a rare primary retroperitoneal mass in an 80-year-old male patient who presented with IAH.

Fig. 1. Multiple masses in retroperitoneal region.

#### CASE REPORT

A patient, 80 years old, was admitted to an emergency surgery clinic with complaints of abdominal distention and not being able to pass gas or stool. He had a medical history of heart failure and was using beta blockers. He was admitted to the emergency clinic for distension a week prior. At physical examination there was abdominal distention, but bowel sounds were normoactive. Digital rectal examination was normal. On admission, laboratory results were normal except for the following; white blood cell, 16,100/mm<sup>3</sup> and creatinine, 1.1 mg/dL. Plain abdomen and chest X-rays were unremarkable (Figs. 1, 2). IAP was 20 mmH<sub>2</sub>O. The patient was placed under observation. Intravenous fluids were started and a urinary catheter was inserted.

Total urine output was only 500 mL in 24 hours, therefore central venous pressure catheterization was performed which measured 15 cm $H_2O$ . An abdominal computed tomography scan revealed multiple masses in the abdomen.

On the 24th hour of follow-up, there was evidence of respiratory and renal failure. IAP was measured at 25 mmH<sub>2</sub>O and later increased to 30 mmH<sub>2</sub>O.

Emergency surgery was planned with IAH prediagnosis. During the surgery, 200 mL of intraperitoneal fluid (caused by ascites aspiration) and multiple retroperitoneal masses were found and stomach, small and large intestine were normal. The greatest palpated mass was  $3\times 4$  cm. Biopsies were taken from the intestinal mesentery. The fas-



Fig. 2. Retroperitoneal-mesenteric masses.

thesurgery.or.kr 243

cia was left opened and the patient's skin was sutured. At postoperative follow-up in intensive care unit (ICU), IAP decreased to 20 cmH<sub>2</sub>O, and urinary flow increased.

On the 24th hour postoperation IAP was again elevated to 25 mmH<sub>2</sub>O. The patient was taken back to surgery for further decompressive laparotomy in order to treat the elevated IAP. During the second operation edema was found in the small intestine and the abdominal wall was left unclosed; this was successful in relieving the IAH. Pathology results confirmed NHL. Patient died of cardiac failure on the 10th postoperative day.

## **DISCUSSION**

Decompressive laparotomy reduces IAP by relieving the tension exerted by the abdominal wall on the inferior vena cava and the portal vein to allow more blood to return to the heart [5]. This explains the sudden normalization of the patient's blood pressure after surgery. Conversely, closure of the abdomen following a laparotomy cannot be done without tension, it is recommended that delayed closure (staged abdominal repair) be undertaken to prevent IAH and ACS, postoperatively.

Surgical abdominal decompression has long been the standard treatment for patients who develop ACS. It represents a life-saving intervention when a patient's IAH becomes refractory to medical treatment options and organ dysfunction and/or failure is evident. Most patients tolerate primary fascial closure within 5 to 7 days if decompressed before significant organ failure develops [7].

Multiple and profound physiologic abnormalities are caused by ACS/IAH, both within and outside the abdomen. Early recognition of increased IAP is primordial in the management. In order for this to occur, monitoring of IAP, either intermittent or continuous, is necessary for all patients presenting with risk factors. Additionally, understanding of the pathophysiology of ACS/IAH is of prime importance when trying to apply patient-tailored treatments. Moreover, surgical intervention should be indicated by IAH and not delayed until ACS is clinically apparent.

Medical interventions aimed at decreasing IAP target

the 3 important contributors to IAH: 1) solid-organ and hollow-viscera volume; 2) space occupying lesions, such as ascites, blood, fluid, or tumors; and 3) conditions that limit expansion of the abdominal wall. When using medical management options to decrease IAP, it is important to always consider individualized pathophysiologic mechanisms leading to IAH because these may differ considerably from one patient to another. Critically, in patients with IAH, small changes in intra-abdominal volume may have a pronounced effect on IAP.

Ileus is a common finding in critically ill patients, especially those with abdominal conditions such as pancreatitis, peritonitis, and abdominal trauma, and postoperative patients. Administration of prokinetic agents is used to overcome abdominal distention and ileus and thus is a treatment option for IAH. When such pharmacologic measures are unsuccessful in decreasing intraluminal volume, endoscopic decompression can be considered.

Ascites and blood are the most common components of space-occupying lesions, but abscesses and free air also can contribute to IAH. When located in the free intraperitoneal space, these collections may be easy targets for percutaneous drainage, which can be performed at the bedside in the ICU under ultrasound guidance. Limited abdominal wall compliance also may be an important contributor to IAH. Increased abdominal muscle tone, most often due to pain or agitation, can be relieved by adequate analgesia and sedation if necessary. Use of restrictive bandages should be avoided. Neuromuscular blockade repeatedly has effectively decreased IAP in patients with IAH [8]. A trial with neuromuscular blocking agents could be considered when simpler measures are not sufficient or are ineffective, and continuous infusion of these agents could be considered when a clinically relevant effect is shown.

The WSACS recently proposed a medical treatment algorithm based largely on expert opinion that is aimed at both decreasing IAP and optimizing fluid resuscitation and systemic perfusion. The medical treatment options discussed may be applied in a stepwise fashion; critically, the present level of evidence supporting these and other elements of this algorithm is limited, and the separate elements are not supported by clinical outcome data.

244 thesurgery.or.kr

However, this algorithm was part of an integrated approach that Cheatham et al. [9] found to improve outcome and decrease hospital costs.

NHL rarely presents primarily with a pelvic or retroperitoneal mass; during post-mortem studies of NHL patients less than 1% incidence was demonstrated [10]. In our case study, the uncommon primary retroperitoneal NHL made the diagnosis more difficult, and even after the NHL was identified in the retroperitoneal space, IAP presentation did not occur in other cases. Our case study is the first with such clinical presentation submitted for publication.

Our case study is a reminder to include the differential diagnosis of NHL when a patient admits to emergency with increased IAP, at which time decompressive laparatomy might be considered as a first line treatment.

### CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

# **REFERENCES**

1. Sanda RB. Abdominal compartment syndrome. Ann Saudi

- Med 2007;27:183-90.
- 2. Malbrain ML, Cheatham ML, Kirkpatrick A, Sugrue M, Parr M, De Waele J, et al. Results from the International Conference of Experts on Intra-abdominal Hypertension and Abdominal Compartment Syndrome. I. Definitions. Intensive Care Med 2006;32:1722-32.
- 3. Carlotti AP, Carvalho WB. Abdominal compartment syndrome: a review. Pediatr Crit Care Med 2009;10:115-20.
- Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000;49:621-6.
- 5. Kim BS, Kwon JW, Kim MJ, Ahn SE, Park HC, Lee BH. Abdominal compartment syndrome caused by a bulimic attack in a bulimia nervosa patient. J Korean Surg Soc 2011;81 Suppl 1:S1-5.
- 6. Allen GW, Forouzannia A, Bailey HH, Howard SP. Non-Hodgkin's lymphoma presenting as a pelvic mass with elevated CA-125. Gynecol Oncol 2004;94:811-3.
- 7. Hadjis NS, McAuley GJ, Ruo L, Kwauk S, Olatunbosun F. Acute abdominal pain and urgency to defecate in the young and the old: a useful symptom-complex? J Emerg Med 1999;17:239-42.
- 8. De Laet I, Hoste E, Verholen E, De Waele JJ. The effect of neuromuscular blockers in patients with intra-abdominal hypertension. Intensive Care Med 2007;33:1811-4.
- Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF. Abdominal perfusion pressure: A superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000;49:621-6.
- Fulignati C, Pantaleo P, Cipriani G, Turrini M, Nicastro R, Mazzanti R, et al. An uncommon clinical presentation of retroperitoneal non-Hodgkin lymphoma successfully treated with chemotherapy: a case report. World J Gastroenterol 2005;11:3151-5.

thesurgery.or.kr 245