TJTES
TURKISH JOURNAL OF TRAUMA
& EMERGENCY SURGERY

Ulusal Travma Dergisi (2002) 8: 133 - 136

ABDOMINAL COMPARTMENT SYNDROME

KARIN KOMPARTMAN SENDROMU

Dr. Recep GÜLOĞLU

ÖZET

***Amaç** : Karın boşluğunda basınç yükselmesi sonucu oluşan kampartman sendromundaki değişiklikler ve tedavi yöntemlerinin incelenmesi.*

***Gereç ve Yöntem** : Yazarın karın kompartman sendromu ile ilgili deneyimleri ve görüşleri literatur eşliğinde gözden geçirilmiştir.*

***Bulgular ve Sonuç** : Karın kompartman sendromu en sık ciddi travma, peritonit, ileus ve yanıklılarda görülmektedir. Yüksek basınçlı olgularda acil laparotomi ile basınç düşürülmelidir. Dekompresyon yapılan hastalarda karın duvarı primer kapatılmadan sentetik materyellar kullanılarak karın içi hacmi genişletilmelidir. Bu işlem mortalite ve morbidite oranlarını düşürecektir.*

***Anahtar Kelimeler:** Karın kompartman sendromu*

ABSTRACT

***Objective** : To invstigte the physiologic effects and therapeutic management of the abdominal kompartman sendrome (ACS)*

***Methods** : A review of the recent literature, experiences and opinions of the author are expressed in the paper.*

***Results and Conclusion** : Intraabdominal bleeding, peritonitis, ileus, shock-reperfusion syndrome, intestinal edema, laparoscopic procedures with gas, closure of the abdomen in spite of increased pressure and burns are some of the factors that take place in etiology. The critical intrabdominal pressure value that requires decompression is debatable. In patients with high abdominal pressures, the pressure should be decreased with laparotomy immediately and the abdomen should not be closed primarily. These measures will help us to lower the rates of mortality and morbidity and improve the quality of health services.*

***Key Words:** Abdominal Compartment Syndrome.*

DEFINITIONS

Compartment syndrome is the consequence of increased contents (edema, blood, mass etc) in spaces such as the legs, cranium, eyes and abdomen (Fig.1). The abdomen is the largest cavity in the body, however organ dysfunction may appear in many clinical scenarios associated with increased abdominal pressure. Although the interest on this issue has recently aroused, there exists reports that attract attention to the abdominal compartment syndrome (ACS) for 150 years (1,2,3).

ACS is encountered worldwide especially in surgical intensive care units. Intraabdominal bleeding (trauma, ruptured aortic aneurysm, postoperative bleeding etc), peritonitis, ileus, shock-reperfusion syndrome, intestinal edema, laparoscopic procedures with gas, closure of the abdomen in spite of increased pressure and burns are some of the etiological factors (Table-1) (4). Morbid obesity and cirrhosis of the liver complicated with ascites are other clinical situations, which present a chronic setting for ACS as well as in pregnancy.

PATHOPHYSIOLOGIC CHANGES IN ACS

Pressure within the abdomen, under normal conditions is about zero (0±5 cm H2O) and any increase in the intraabdominal pressure has an adverse effect on the organism. Free oxygen radicals are released as a consequence of edema in the intestinal mucosa due to the splanchnic hypoperfusion and then systemic dysfunction occurs especially in the cardiovascular, respiratory and urogenital systems. Intraabdominal hypertension causes a decrease in cardiac output despite normal ejection fraction. Preload is decreased due to decreased venous return; whereas afterload is increased. Peripheral vascular resistance and inspiratory pressures are also increased.

Lung compliance falls since the thoracic cavities are compressed by the increased abdominal distention. The result is increased pulmonary vascular resistance and increased pulmonary artery pressures as well as increased airway pressures (6). Blood gas analysis reveals decreased pO2 and increased pCO_2. Tonometric studies determine severe acidosis.

Fig.1: The appearance of intestines in a patient with ACS.

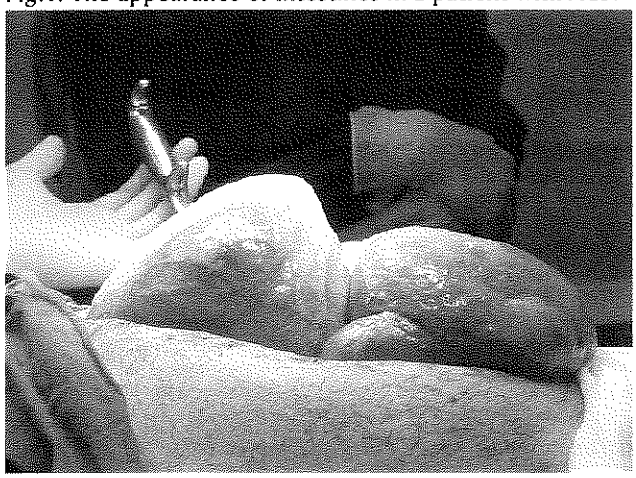
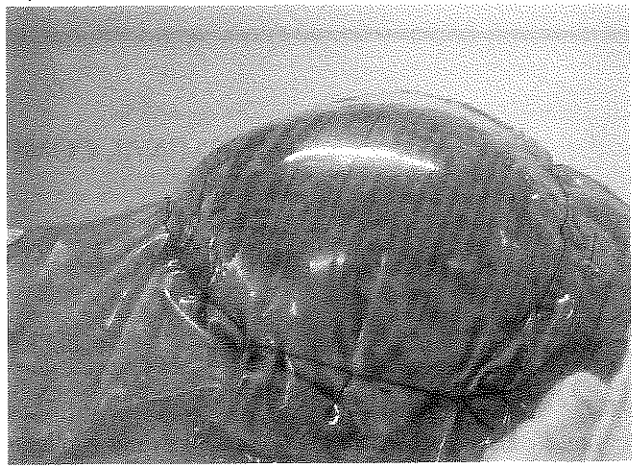


Fig. 2: Closure of the abdominal wall with sterile nylon



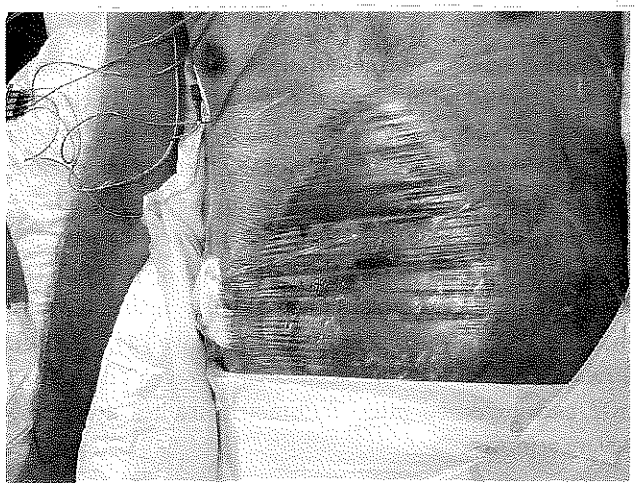
Decrease in renal perfusion and glomerular filtration rate result in oliguria. Oliguria is a milestone in ACS. It presents itself at intraabdominal pressure of 15-20 mmHg. A pressure of more than 20 mmHg will cause anuria. Some studies express that blood circulation in intraabdominal organs except the adrenals and the abdominal wall is decreased. Intracranial and intrathoracic pressures are increased as well (1,5,6,7,8).

Morris et al. have defined ACS in a distended abdomen associated with lack of ventilation due to increased inspiratory pressure, hypercarbia, oliguria and increased abdominal pressure (9). Deterioration of respiratory functions and oliguria had been regarded as the first clinical findings of ACS. The mortality rate is as high as % 40 in ACS and early diagnosis and treatment is life saving (10).

DIAGNOSIS AND MEASUREMENT

The first step in the diagnosis is awareness of this syndrome and intermittent measurements of the intraabdominal pressure. Direct or indirect measurement of intraabdominal pressure is available in clinical settings (11,12). Intraabdominal pressure should be detected routinely in high- risk patients in ICUs (13,14). Direct measurement can be accomplished with an intraabdominal drain or laparoscopic catheter and indirect measurement is possible through a catheter in bladder or stomach. The intraabdominal pressure in our department is measured as follows: 100 ml of saline is injected into the empty bladder via a urinary Foley catheter. The pressure is measured while the patient is in the recumbent position and the symphysis pubis is accepted as the landmark or 0 point. This can be done with a saline line or a transducer connected to a monitor.

Fig. 3: Closure of the abdominal wall with drape.



The critical intrabdominal pressure value that requires abdominal decompression is debatable in the literature (1,15). Even though, a level of 10 mmHg during laparoscopic procedures causes temporary organ dysfunction. However, recent reports prefer to use Burch's grading in intraabdominal pressures and an intervention is justified in patients with Grade 3 or higher pressures (Table-2) (1,2,4). In order to assess the indication of decompression, we use the measurement of the abdominal pressure in conjunction with physical findings and laboratory results.

TREATMENT

In patients with ACS, decompression is often achieved via laparotomy with open abdomen or closing abdomen with a synthetic mesh (4). This technique provides an increased space, which is essential in the management of ACS. Although there are recent studies advocating the laparoscopic and percutaneous treatment of ACS, further studies are required to define laparoscopy and percutaneous techniques as effective as laparotomy

Table-1 Frequent causes of increased intraabdominal pressures

- Increased intraabdominal content
- Pancreatitis
- Pneumoperitoneum
- Intraabdominal or retroperitoneal bleeding
- Intraabdomianl infection
- Ileus
- Damage control surgery
- Tight closure of the abdomen

(16,17).

In the last two decades trauma surgeons have had advantages of both damage controlled surgical techniques and abdominal closure techniques (2,4). In order to prevent ACS in high-risk patients, we do not prefer to close the abdominal wall primarily. The gap between the abdominal wound edges is covered with sterile nylon, drape or non-absorbable synthetic graft (Fig. 2 and 3). We do not prefer to close the abdomen temporarily with clamps or sutures, since we believe that these techniques are minimal invasive methods. ACS may develop in patients with ruptured abdominal aortic aneurysm and as mentioned in literature by Rasmussen et al, we suggest using synthetic grafts for the closure of the abdomen in these patients after reconstruction of the aorta (18).

CONCLUSION

ACS is encountered in severe abdominal trauma, ruptured abdominal aortic aneurysm, peritonitis, pancreatitis, pneumoperitoneum and burns at most. A recent review of the literature from 1966 to 2000 by Joynt et al revealed the incidence of ACS in high risk surgery patients as 5-40%(19). Clinical and expermental evidence indicate that increased intraabdominal presure may adversely affect cardiac, respiratory, metabolic and renal functions. In another issue it was demonstrated that elevated intraabdominal pressure delays healing of colonic anastomoses (20).

Educational programs as preventive medicine in trauma by using internet and media, faster transportation of the patients, appropriate resuscitation, advances in diagnosis and treatment together with intensive care supports will decrease the incidence of ACS. Also high pressures should be avoided in laparoscopic procedures. We have to define the patients who are prone to ACS on time and these patients should be treated in ICUs. In patients with high abdominal pressures and clinical findings of ACS, the pressure should be decreased with laparotomy immediately and the abdomen should not be closed primarily. These measures will help us to lower the rates of mortality and morbidity and improve the quality of health services.

Table -2 Grading System for ACS by Burch

GRADE	BLADDER PRESSURE		MANAGEMENT
	cm H_20	mm Hg	
I	10-15	7-11	Normal pressures
II	15-25	11-18	Frequent measurements
III	25-35	18-26	Consider decompression
IV	>35	>26	Decompression

REFERENCES:

1. *Ivatury RR, Diebel L, Porter JM, et al. Intraabdominal hypertension and the abdominal compartment syndrome. Surg Clin North Am. 1997; 77: 783-800.*

2. *Feliciano DV, Moore EE, Mattox KL. Trauma damage control. In: Mattox KL, Feliciano DV, Moore EE, eds. Trauma. 4th ed. New York: McGraw-Hill; 2000: 907-931.*

3. *Coombs HC. The mechanism of the regulation of intra-abdominal pressure. Am J Physiol. 1920; 61:159.*

4. *Wittmann DH, Iskander GA. The Compartment Syndrome of the abdominal cavity: A state of the art review. J Int Care Med. 2000; 15: 201.*

5. *Schein M, Wittmann DH, Aprahamian CC, et al. The abdominal compartment syndrome: The physiological and clinical consequenses of elevated intra-abdominal pressure. J Am Coll Surg. 1995; 180: 745-753.*

6. *Obeid F, Saba A, Fath J, et al. Increases in intraabdominal pressure affect pulmonary compliance. Arch Surg. 1995; 130: 544-548.*

7. *Maxwell RA, Fabian TC, Croce MA, et al. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. J Trauma. 1999; 47: 995-999.*

8. *Rosin D, Brasesco O, Varela J, et al. Low-pressure laparoscopy may ameliorate intracranial hypertension and renal hypoperfusion. J Laparoendosc Adv Surg Tech A. 2002; 12: 15-19.*

9. *Morris JA, Eddy VA, Blinman TA, et al. The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg. 1993; 217: 575-586.*

10. *Jonathan RH. Abdominal compartment syndrome. In: Naude PG, Bongard FS, Demetriades D, eds. Trauma Secrets. Henley&Belfus Inc. 1999: 103-107.*

11. Yol S, Kartal A, Tavli S, et al. Is Urinary Bladder Pressure a Sensitive Indicator of Intra-Abdominal Pressure? Endoscopy. 1998; 30: 778-780.

12. Akinci OF, Uzunkoy A, Tacyildiz IH, et al. The measurement of infra-vesical pressure as an indicator of abdominal pressure (In Turkish). Turk J Surg. 1997; 13: 329-332.

13. Güloglu R, Berber E, Taviloglu K, et al. Clinical importance of intraabdominal pressure in the surgical intensive care. The Eur J Emerg Surg and Int Care. 1997; 20: 191-194.

14. Chen RJ, Fang JF, Chen MF. -Intra-abdominal pressure monitoring as a guideline in the nonoperative management of blunt hepatic trauma. J Trauma. 2001; 51: 44-50.

15. Burch JM, Moore EE, Moore FA, et al. The abdominal compartment syndrome. Surg Clin North Am. 1996; 76: 833-842.

16. Corcos AC, Sherman HF. Percutaneous treatment of secondary abdominal compartment syndrome. J Trauma. 2001; 51: 1062-1064.

17. Chen RJ, Fang JF, Lin BC, et al. Laparoscopic decompression of abdominal compartment syndrome after blunt hepatic trauma. Surg Endosc. 2000; 14: 966.

18. Rasmussen TE, Hallett JW Jr, Noel AA, et al. Early abdominal closure with mesh reduces multiple organ failure after ruptured abdominal aortic aneurysm repair: guidelines from a 10-year case-control study. J Vasc Surg. 2002; 35: 246-253.

19. Joynt GM, Ramsay SJ, Buckley TA. Intra-abdominal hypertension--implications for the intensive care physician. Ann Acad Med Singapore. 2001 May; 30(3): 310-9

20. Koloğlu M, Sayek İ, Koloğlu LB, Eng C, Önat D.: Effect of persistently elevated intraabdominal pressure on healing of colonic anastomoses. Am J Surg. 1999 Oct; 178(4): 293-7.

*Department of General Surgery, Istanbul Medical Faculty, University of Istanbul, Istanbul, Turkey.
Yazışma Adresi: Recep Güloğlu MD.
İstanbul Üniversitesi, İstanbul Tıp Fakültesi,
Genel Cerrahi A.B.D. G Servisi , 34390, Çapa, İstanbul
e-posta : rguloglu@yahoo.com