REVIEW

CAN INTRA-ABDOMINAL HYPERTENSION BE PREDICTED AFTER INCISIONAL HERNIA CORRECTION?

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Abstract

Intra-abdominal hypertension (IAH) leading to abdominal compartment syndrome (ACS- a condition that can impair the function of nearly every organ system) is a high mortality complication of incisional hernia repair. This review aims to present the current evidence on the relation between incisional hernia repair and the risk of developing IAH, and the impact of IAH on surgical care. IAH is defined by a sustained or repeated pathological elevation in intra-abdominal pressure (IAP) greater 12 mmHg. ACS has a high mortality rate, ranging from 40-100%, so it is important to: recognize the risk factors, use preventive techniques, monitor the intraabdominal pressure, and act early to decrease the pressure before it leads to organ dysfunctions. Hernia dependent risk factors are an incisional hernia volume/peritoneal volume calculated using volumetric CT calculation > 20% and a maximum transverse hernia diameter > 10cm. The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25mL of sterile saline. Recommend measures to reduce IAP both invasive and non-invasive: decompressive laparotomy in case of ACS, sedation and analgesia, neuromuscular blockade, body positioning, nasogastric/colonic decompression, promotility agents, diuretics and continuous renal replacement therapies, fluid resuscitation strategies, percutaneous catheter drainage (PCD), and different temporary abdominal closure (TAC) techniques among those requiring an open abdomen.

Keywords: *incisional hernia correction, intra-abdominal hypertension, abdominal compartment*

Introduction

The management of incisional hernias has changed a lot over the past decade, with considerably better results both in avoiding relapse and postoperative complications. At the same time, there is an increased interest in the study of a relatively recently defined pathology, which is Intraabdominal Hypertension (IAH) and its complication Abdominal Compartment Syndrome (ACS). The purpose of this article is to review the current evidence on the relation between incisional hernia repair and the risk of developing IAH and the impact of IAH on surgical care.

Materials and method

Source data were obtained from PubMed and Web of Knowledge searches of the medical literature, with an emphasis on the time period after 2000, using the following terms and their combinations: "intraabdominal", "intra-abdominal", "pressure", "hypertension", "incisional hernia", "ventral hernia", "repair", "reconstruction", "abdominal compartment syndrome", "predicted", "screening". Additional information was derived from the articles referenced by those resulted from the initial search, from the Web site of the World Society of the Abdominal Compartment Syndrome (http://www.wsacs.org) and from General Surgery textbooks.

Results

Incisional hernia

By definition a hernia is represented by the protrusion of viscera from the abdominal cavity through a preexisting, natural route. In an incisional hernia the protrusion route of viscera from the abdominal cavity is formed by trauma induced by cutting (surgical incision, laparoscopic trocar puncture wounds, and stab wounds) [1].

Incisional hernias can develop after any type of abdominal wall incision, vertical incisions being more susceptible compared with transverse, and upper abdominal more susceptible than lower abdominal incisions [2]–[6].

The repair of an incisional hernia is recommended for patients without significant comorbidities that can undergo surgery and present either: (1) a symptomatic hernia, (2) an asymptomatic hernia with a high risk of incarceration, or (3) an asymptomatic hernia that interferes with the patient's quality of life [7]. The repair can be a simple suture, a mesh repair positioned either above the fascia (onlay) or below the fascia (sublay), or component separation repair with or without mesh reinforcement [8].

IAH (intra-abdominal hypertension) and ACS (abdominal compartment syndrome)

Definitions

Massive incisional hernia repair is considered a risk factor for developing IAH. IAH is defined by a sustained or repeated pathological elevation in intra-abdominal pressure (IAP) ≥ 12 mmHg [9]. It must be noted that in patients with increased abdominal girth that developed slowly, for example the morbidly obese and pregnant women, a chronic IAP (up to 10-15 mmHg) is considered physiological [10].

IAH is graded as presented in Table 1 [9].

Grade	IAP (mmHg)
I	12-15
II	15-20
III	21-25
IV	>25

Table 1- IAH grades

While elevated IAP is an independent risk factor statistically associated with increased morbidity and mortality, there is no accepted "critical value" that causes end-organ dysfunction, being depended on preexisting comorbidities. Abdominal perfusion pressure (APP) was found to be better than resuscitation endpoints such as arterial pH, base deficit, arterial lactate, and hourly urinary output, for predicting outcomes [11]. To improve survival rates form IAH and ACS a APP of at least 60 mmHg should be obtained [11,12].

IAH can progress in time to ACS, a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure.

Epidemiology and mortality

One of the first prospective studies on IAP after hernia repair found a 92% incidence of IAH grade I or higher [13]

Mortality for patients that progressed to ACS is high, ranging from 40-100% [14-16]. This high mortality gives clinicians the incentive to recognize early IAH, before its development to ACS.

While a study from 2003 found that despite early recognition the outcome of ACS remained unchanged [17], advances in management in the following years resulted in an improved patient survival to hospital discharge from 50% to 72% [18].

IAH and ACS physiology

The IAP is dependent on the abdominal compliance and the volume that needs to be accommodated. Blaser has described the three phase response of the abdominal cavity to increased pressure and/or volume: (1) reshaping- constant pressure maintained by an increase in volume; (2) stretching- near linear relationship of pressure and volume; (3) rapid pressurization- exponential rise in pressure in a near inextensible compartment [19].

After an insult the body reacts with a systemic inflammatory response, leading by a series of steps to an increase in IAP by defeating the compensatory mechanisms. This increased IAP acts like an insult, creating a vicious cycle. The continuous increase of pressure begins to affect the function of different organs (either by direct compression or by indirect effects from other systems, like the decrease in cardiac output), this point marking the onset of ACS. Some of the principal effects are presented in Figure 1. The end result is multiple organ failure (MOF) and death [1].

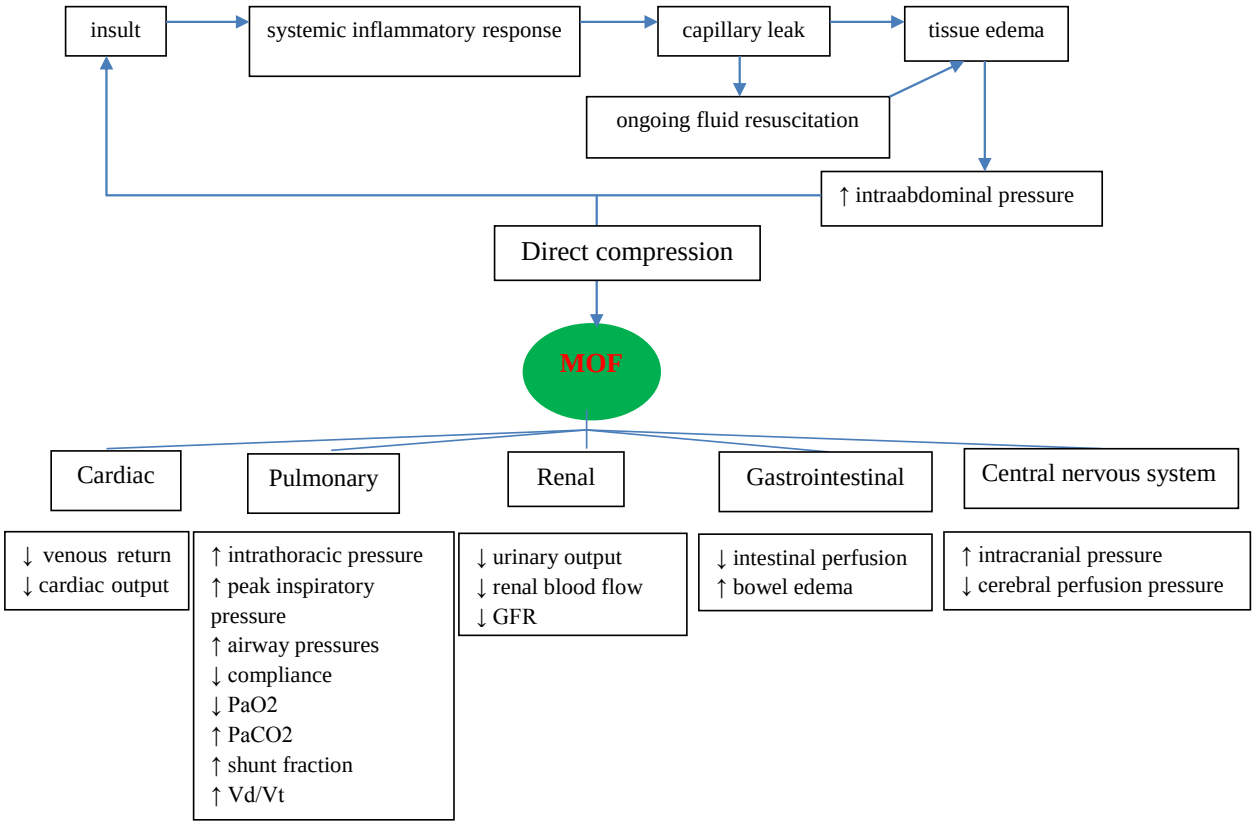

Figure 1 - Physiologic consequences of IAH and ACS.

It is important to act in the early stages of the disease. This prevents the progression to the self-sustaining process from the later stages. Early detection is also important because it leads to a reduction in fluid administration, thus preventing the increase of the tissue edema [17], [20].

It has been observed that patients that have undergone abdominal hernia repair surgery tolerate increased IAP much better than those suffering from major trauma and inflammation, and do not enter the vicious cycle previously explained. This is especially true in modest hernia repairs reshaping and/or stretching are sufficient to normalize the IAP. Rapid pressurization can occur in massive hernia repairs. These differences have lead Kirkpatrick to propose a separate classification for this subset of patients, that of the quaternary IAH and ACS [21].

Factors that influence IAP

As it has already been stated, massive incisional hernia repair is a risk factor for developing IAH. This risk is dependent on the hernia characteristics and the operative technique used. Hernia dependent factors described in the literature are: an incisional hernia volume/peritoneal volume calculated using volumetric CT calculation > 20% [22] and a maximum transverse hernia diameter > 10 cm

[23]. Surgeons have several options to reduce the IAP by: (1) reducing the volume that needs to be accommodated by the abdominal cavity using an elective bowel resection; (2) increasing the abdominal compliance by a neuromuscular blockage and Botulinum Toxin A; (3) enlarging the container by: transverse division of abdominal musculature, phrenectomy, progressive pre-operative pneumoperitoneum or musculofascial flaps [21].

Other conditions that can further increase the risk of developing IAH are: prone positioning [24-26], ileus [27], volvulus, hemoperitoneum/pneumoperitoneum or intra-peritoneal fluid collections [28] , intra-abdominal infection/abscess [29] , ascites [30], acidosis [31,32], hypothermia [17], massive fluid resuscitation or positive fluid balance [17], polytransfusion [30,33], obesity [34], PEEP (positive end-expiratory pressure) >10 cmH2O [35], age [33], mechanical ventilation [27], shock or hypotension [36], sepsis [29].

Diagnosis

Clinical and imagistic findings in IAH and ACS are nonspecific and depend, in the case of ACS, on the organs affected. They are insufficient for a positive diagnosis. For example a study on physical examination of the abdomen found it to have a sensitivity of 56%, specificity of 87%, positive predictive value of 35%, negative predictive value of 94%, and accuracy of 84%, in identifying IAH [37].

Definitive diagnosis of ACS requires measurement of the intra-abdominal pressure. The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25mL of sterile saline. IAP should be expressed in mmHg and measured at end-expiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line [9].

There are situations in which the free movement of the bladder wall is impeded - intraperitoneal adhesions, pelvic hematomas, pelvic fractures, abdominal packs, or a neurogenic bladder - and other techniques must be used [38].

Considering the abdomen as a relatively non-compressive and primary fluid in character, subject to Pascal's law, the IAP can be measured in any part of the abdomen. Measurement techniques recommended for screening by a 2012 review are [38]: Cheatham's bladder technique [39], Malbrain's manometry technique [40] and Collee's stomach technique [41].

Management

Different measures to reduce IAP are recommended by the 2013 Consensus [9] both invasive and non-invasive: decompressive laparotomy in case of ACS, sedation and analgesia, neuromuscular blockade, body positioning, nasogastric/colonic decompression, promotility agents, diuretics and continuous renal replacement therapies, fluid resuscitation strategies, percutaneous catheter drainage (PCD), and different temporary abdominal closure (TAC) techniques among those requiring an open abdomen.

The steps taken by clinicians are depended on a constant and accurate assessment of IAP.

Conclusions

IAH leading to ACS (a condition that can impair the function of nearly every organ system) is a high mortality complication of incisional hernia repair. Its development is dependent on the size of the repaired hernia, the techniques used and the patients preoperative condition.

The understating of the pathophysiology of IAH/ACS is important when trying to apply patient-tailored treatments.

Diagnosis of ACS requires that intra-abdominal pressure be measured; symptoms, physical signs, and imaging findings are insufficient to diagnose ACS.

Early diagnosis of ACS is critical for a proper management and an increase of survival rates.

References

[1]V. Karimyan, Hernia Surgery. 2004.

[2]N. Fassiadis, M. Roidl, M. Hennig, L. M. South, and S. M. Andrews, "Randomized clinical trial of vertical or transverse laparotomy for abdominal aortic aneurysm repair," Br. J. Surg., vol. 92, no. 10, pp. 1208–1211, Oct. 2005.

[3]T. Inaba et al., "Prospective randomized study of two laparotomy incisions for gastrectomy: Midline incision versus transverse incision," Gastric Cancer, vol. 7, no. 3, pp. 167–171, Sep. 2004.
[4]S. Levrant, E. Bieber, and R. Barnes, "Risk of Anterior Abdominal Wall Adhesions Increases with Number and Type of Previous Laparotomy," J. Am. Assoc. Gynecol. Laparosc., vol. 1, no. 4, Part 2, p. S19, Aug. 1994.
[5]K. A. Bickenbach et al., "Up and down or side to side? A systematic review and meta-analysis examining the impact of incision on outcomes after abdominal surgery," Am. J. Surg., vol. 206, no. 3, pp. 400–409, Sep. 2013.
[6]S. R. Brown and P. B. Goodfellow, "Transverse verses midline incisions for abdominal surgery.," in Cochrane database of systematic reviews (Online), no. 4, S. R. Brown, Ed. Chichester, UK: John Wiley & Sons, Ltd, 2005, p. CD005199.
[7]R. J. Fitzgibbons et al., "Watchful waiting vs repair of inguinal hernia in minimally symptomatic men: a randomized clinical trial.," Jama, vol. 295, no. 3, pp. 285–92, Jan. 2006.
[8]D. L. Sanders and A. N. Kingsnorth, "The modern management of incisional hernias," Bmj, vol. 344, no. may09 1, pp. e2843–e2843, May 2012.
[9]A. W. Kirkpatrick et al., "CONFERENCE REPORTS AND EXPERT PANEL," Intensive Care Med, vol. 39, pp. 1190–1206, 2013.
[10]E. E. Frezza, K. O. Shebani, J. Robertson, and M. S. Wachtel, "Morbid obesity causes chronic increase of intraabdominal pressure," Dig. Dis. Sci., vol. 52, no. 4, pp. 1038–1041, Mar. 2007.
[11]A. F. K. Moore, R. Hargest, M. Martin, and R. J. Delicata, "Intra-abdominal hypertension and the abdominal compartment syndrome," Br. J. Surg., vol. 91, no. 9, pp. 1102–1110, Aug. 2004.
[12]J. J. De Waele, I. De Laet, A. W. Kirkpatrick, and E. Hoste, "Intra-abdominal hypertension and abdominal compartment syndrome," Am. J. Kidney Dis., vol. 57, no. 1, pp. 159–169, Aug. 2011.
[13]G. Habib et al., 2015 ESC Guidelines for the management of infective endocarditis, vol. 36, no. 44. 2015.
[14]M. Sugrue, "Abdominal compartment syndrome.," Curr. Opin. Crit. Care, vol. 11, no. 4, pp. 333–338, Aug. 2005.
[15]M. L. N. G. Malbrain et al., "Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study.," Crit. Care Med., vol. 33, no. 2, pp. 315–322, Feb. 2005.
[16]G. An and M. A. West, "Abdominal compartment syndrome: a concise clinical review.," Crit. Care Med., vol. 36, no. 4, pp. 1304–1310, Apr. 2008.
[17]Z. Balogh et al., "Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure.," J. Trauma, vol. 54, no. 5, pp. 848-59-61, May 2003.
[18]M. L. Cheatham and K. Safcsak, "Is the evolving management of intra-abdominal hypertension and abdominal compartment syndrome improving survival?," Crit. Care Med., vol. 38, no. 2, pp. 402–407, Feb. 2010.
[19]A. R. Blaser, M. Björck, B. De Keulenaer, and A. Regli, "Abdominal compliance," J. Trauma Acute Care Surg., vol. 78, no. 5, pp. 1044–1053, May 2015.
[20]E. L. Daugherty, D. Taichman, J. Hansen-flaschen, and B. D. Fuchs, "Abdominal Compartment Syndrome Is Common in Medical Intensive Care Unit Patients Receiving Large-Volume Resuscitation," Physiology, vol. 22, no. 5, pp. 294–299, Sep. 2008.
[21]A. W. Kirkpatrick et al., "Intra-Abdominal Hypertension and Abdominal Compartment Syndrome after Abdominal Wall Reconstruction: Quaternary Syndromes?," Scand. J. Surg., 2016.
[22]C. Sabbagh, F. Dumont, B. Robert, R. Badaoui, P. Verhaeghe, and J. M. Regimbeau, "Peritoneal volume is predictive of tension-free fascia closure of large incisional hernias with loss of domain: A prospective study," Hernia, vol. 15, no. 5, pp. 559–565, 2011.
[23]C. I. Mavrodin, G. Pariza, D. Ion, and V. I. Antoniac, "Abdominal compartment syndrome -- a major complication of large incisional hernia surgery.," Chirurgia (Bucur)., vol. 108, no. 3, pp. 414–7, 2013.
[24]A. W. Kirkpatrick et al., "Clinical review: Intra-abdominal hypertension: does it influence the physiology of prone ventilation?," Crit. Care, vol. 14, no. 4, p. 232, 2010.
[25]R. Hering et al., "Prone positioning, systemic hemodynamics, hepatic indocyanine green kinetics, and gastric intramucosal energy balance in patients with acute lung injury," Intensive Care Med., vol. 28, no. 1, pp. 53–58, Jan. 2002.
[26]R. Hering et al., "The effects of prone positioning on intraabdominal pressure and cardiovascular and renal function in patients with acute lung injury.," Anesth. Analg., vol. 92, no. 5, pp. 1226–31, May 2001.
[27]M. G. Vidal et al., "Incidence and clinical effects of intra-abdominal hypertension in critically ill patients," Crit Care Med, vol. 36, no. 6, pp. 1823–1831, Jun. 2008.
[28]L. Ke et al., "Risk factors and outcome of intra-abdominal hypertension in patients with severe acute pancreatitis.," World J. Surg., vol. 36, no. 1, pp. 171–8, Jan. 2012.

[29]I. B. Kim, J. Prowle, I. Baldwin, and R. Bellomo, "Incidence, risk factors and outcome associations of intra-abdominal hypertension in critically ill patients," Anaesth. Intensive Care, vol. 40, no. 1, pp. 79–89, Jan. 2012.
[30]Z. J. Balogh, "Mission to Eliminate Postinjury Abdominal Compartment Syndrome," Arch. Surg., vol. 146, no. 8, p. 938, Aug. 2011.
[31]J.-F. Ouellet, A. Leppaniemi, C. G. Ball, M. L. Cheatham, S. D'Amours, and A. W. Kirkpatrick, "Alternatives to formal abdominal decompression.," Am. Surg., vol. 77 Suppl 1, pp. S51-7, Jul. 2011.
[32]R. Jaeschke et al., "Use of GRADE grid to reach decisions on clinical practice guidelines when consensus is elusive.," Bmj, vol. 337, p. a744, Jul. 2008.
[33]L. Dalfino, L. Tullo, I. Donadio, V. Malcangi, and N. Brienza, "Intra-abdominal hypertension and acute renal failure in critically ill patients," Intensive Care Med., vol. 34, no. 4, pp. 707–713, Apr. 2008.
[34]B. L. De Keulenaer, J. J. De Waele, and M. L. N. G. Malbrain, "Nonoperative management of intra-abdominal hypertension and abdominal compartment syndrome: evolving concepts.," Am. Surg., vol. 77 Suppl 1, pp. S34-41, Jul. 2011.
[35]A. R. Blaser, P. Par, R. Kitus, and J. Starkopf, "Risk factors for intra-abdominal hypertension in mechanically ventilated patients," Acta Anaesthesiol. Scand., vol. 55, no. 5, pp. 607–614, May 2011.
[36]M. Malbrain, ML; Cheatham, "Definitions and pathophysiological implications of intra-abdominal hypertension and abdominal compartment syndrome," Am Surg, vol. 77, pp. 6–11, 2011.
[37]A. W. Kirkpatrick, F. D. Brenneman, R. F. McLean, T. Rapanos, and B. R. Boulanger, "Is clinical examination an accurate indicator of raised intra-abdominal pressure in critically injured patients?," Can. J. Surg., vol. 43, no. 3, pp. 207–211, Jun. 2000.
[38]M. L. N. G. Malbrain, "Different techniques to measure intra-abdominal pressure (IAP): Time for a critical re-appraisal," Appl. Physiol. Intensive Care Med. 2 Physiol. Rev. Ed., pp. 13–27, 2012.
[39]M. L. Cheatham and K. Safcsak, "Intraabdominal pressure: A revised method for measurement," J. Am. Coll. Surg., vol. 186, no. 5, pp. 594–595, May 1998.
[40]M. Malbrain, M. Léonard, and D. Delmarcelle, "A novel technique of intra-abdominal pressure measurement:validation of two prototypes," Crit. Care, vol. 6, no. Suppl 1, p. P4, 2002.
[41]G. G. Collee, D. M. Lomax, C. Ferguson, and G. C. Hanson, "Bedside measurement of intra-abdominal pressure (IAP) via an indwelling naso-gastric tube: Clinical validation of the technique," Intensive Care Med., vol. 19, no. 8, pp. 478–480, 1993.