Intraperitoneal wound in abdominal surgery

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Received: August 12, 2011 Revised: July 14, 2012
Accepted: December 5, 2012 Published online: February 4, 2013

Abstract
The intraperitoneal wound is often forgotten after transperitoneal surgery. This review is a on the peritoneum and the implications of peritoneal injury after surgery. This review will focus on the intraperitoneal wound response after surgical injury.

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Key words: Peritoneum; Vagus nerve; Abdominal surgery; Cytokine; Laparoscopy; Inflammation

Kahokehr AA. Intraperitoneal wound in abdominal surgery. World J Crit Care Med 2013; 2(1): 1-3 Available from: URL: http://www.wjgnet.com/2220-3141/full/v2/i1/1.htm DOI: http://dx.doi.org/10.5492/wjccm.v2.i1.1

INTRODUCTION
An abdominal operation combines a somatic abdominal wall wound with a second wound to the peritoneal cavity and viscera. Little attention has been paid the peritoneal wound that communicates directly to the brain by the vagus nerve. Traditionally most interventions have focused on producing relief from the physiological burden created by the somatic abdominal wall wound. However recent research indicates that the “forgotten” intraperitoneal wound may be clinically important especially in those who undergo extensive transperitoneal injury.

PERITONEUM
The peritoneum is a serous membrane that lines the abdominal cavity and the intra-abdominal viscera. This dynamic cellular membrane has important functions. It provides a frictionless environment for movement of abdominal organs and is a metabolically active sheet of tissue that envelops the majority of the abdominal viscer as and is protected by macrophage scavengers. These scavengers play a key role in the local immune response by producing local mediators such as interleukin-6 (IL-6), tumor necrosis factor (TNF-α) and oxygen radicals. Furthermore the peritoneum is unique compared to surrounding organs in that it carries a lower level of anti-inflammatory pathways resulting to a greater adhesion forming pathways after injury compared to the regeneration that occurs in other organs. The peritoneum is highly metabolically involved and active, enveloping the majority of the abdominal viscera.

Because the entire peritoneal cavity is linked via transcocloic spread of immuno-humoral factors in the peritoneal fluid, it exhibits a coordinated response to injury which is generalised and not limited to the localised area of insult. This is supported by the fact that there are much higher cytokine concentrations in peritoneal fluid than in plasma after gastrointestinal surgery suggesting that cytokine production occurs in a compartmentalised fashion within the abdominal cavity. The nerve supply to the peritoneum is conveyed along the autonomic nervous system from the parasympathetic and sympathetic system. It can convey sensory fibres via the cranial nervous system, namely the sub-diaphragmatic vagus afferents. The sub-diaphragmatic vagal afferents, about 50000 in number are almost all made from low threshold unmyelinated (C) fibres. They convey background sensations such as mechanical stretch, satiety, fullness, nausea and vomiting sensations.
behaviour in humans\textsuperscript{10,11}. Furthermore, 90\% of the subdiaphragmatic vagus is entirely afferent in nature, indicating a critically important role in direct peritoneal to central nervous system signal transmission and modulation of inflammatory processes arising from the peritoneum\textsuperscript{10,12,13}.

Spinal afferent also supply the parietal peritoneal lining and mesentery of the gastrointestinal tract, and have cell bodies located in the dorsal root ganglion projecting to the dorsal horn of the spinal cord. They follow the paths of the sympathetic (splanchnic) and parasympathetic (pelvic) efferents to the gut wall\textsuperscript{14}. All of the visceral afferents combined make no more than 7%-10\% of all afferent inflow to the spinal cord\textsuperscript{15,14}.

**SURGICAL TRAUMA TO THE PERITONEUM**

Comparative studies of cellular immunity after laparoscopic and conventional trans-peritoneal surgery have demonstrated immunologic advantages conferred by reducing the somatic abdominal wall wound by performing minimal access laparoscopy\textsuperscript{19}. In these animal models there appears to be a biological peritoneal advantage after laparoscopy when compared to open surgery. Authors have hence argued, that the peritoneal immune response after laparoscopic surgery is better preserved\textsuperscript{13}. This mechanism is uncertain but has been thought to arise from smaller peritoneal incisions minimizing peritoneal stress, reduced exposure of peritoneum to solubilized pathogens in air, and minimal manipulation and handling of the organs.

A large component of the intra-abdominal afferent system (40%-45\% in the colon and bladder) are by fibres normally unresponsive to stimuli that become activated only in the presence of inflammation and injury\textsuperscript{16}. These “silent nociceptors” are different in that they are mainly concerned with tissue injury rather than mechanical stimuli such as stretch. One theory is that these class of nociceptors lead to abnormal autonomic regulations by insult which produces dramatic changes in the environment that surrounds the nerve endings with potential to excite distant nociceptors not affected by the initial insult\textsuperscript{17}.

What is also concerning about intraperitoneal nociceptors is that as they are not normally active, discharge after inflammation and injury may be greater in magnitude and duration than the discharges produced by acute injurious stimuli, which potentially makes the central effects even greater than the initial insult\textsuperscript{18}. Transceolomic spread of pro-inflammatory cytokines may activate areas of the peritoneum distant from the site on intraperitoneal injury. Thus downstream effects may persist for a long duration even after the initial injury is near or complete resolution.

The abdominal wall wound can be reduced significantly in size, by minimally access techniques, such as laparoscopy. When one considers procedures where the incision is the cause of the predominant metabolic insult to the patient, the benefits would appear to be obvious and of a significant clinical magnitude. For example in cholecystectomy the metabolic response is thought to be from the abdominal wall wound itself\textsuperscript{39,31}. Therefore it can be postulated that this should translate into a lesser magnified sickness response and hence quicker recovery. The benefits of laparoscopic cholecystectomy are evident when compared to classic open colectomy\textsuperscript{25} but not as obvious when compared to the small or “mini” laparotomy version of the same operation\textsuperscript{21}. Therefore there may be a threshold size effect where further benefits are not seen.

However what is interesting is that in humans the peritoneal cytokine response is similar in laparoscopic and open colonic surgery\textsuperscript{22,23,24}. Also the systemic pro-inflammatory concentrations after both surgical approaches represent only a small fraction of what is generated from the peritoneum. This suggests that the two intra-abdominal approaches are locally equally traumatic to the peritoneal cavity\textsuperscript{22,24}. Thus it seems plausible that laparoscopic surgery does not confer an additional clinical advantage if we concentrate on peritoneal wound disruption. The intraperitoneal disruption is still the same no matter how access to the cavity is gained. A recent review on this topic in an optimized recovery setting has clinically confirmed that similar clinical outcomes can be reached between modalities\textsuperscript{25}. Hence what seems to be rather important in abdominal surgery is whether the peritoneum as an entity is entered, dissected, and manipulated. This is demonstrated in clinical studies of aorta aneurysm repair, with trans-peritoneal aneurysmal repair resulting in significantly higher inflammatory response corresponding to slower clinical recovery compared to the extra-peritoneal approach where this is possible\textsuperscript{26}.

Operating on many organs such as colonic resection necessitates intraperitoneal injury and hence extra-peritoneal approach is not an option. In Part II of this series we will focus on possible new methods to manipulate the intraperitoneal wound in order to reach improved clinical endpoints.

**REFERENCES**

1. Nachtsheim R, Dudley B, McNeil PL, Howdeshell TR. The peritoneal cavity is a distinct compartment of angiogenic molecular mediators. J Surg Res 2006; 134: 28-35 [PMID: 16650862 DOI: 10.1016/j.jss.2006.03.008]

2. Jackson PG, Evans SR. Intraperitoneal macrophages and tumor immunity: A review. J Surg Oncol 2008; 75: 146-154 [PMID: 11063907]

3. Fegan KS, Rae MT, Critchley HO, Hillier SG. Anti-inflammatory steroid signalling in the human peritoneum. J Endocrinol 2008; 196: 369-376 [PMID: 18252960 DOI: 10.1677/JOE-07-0419]

4. Sammour T, Kahokehr A, Soop M, Hill AG. Peritoneal damage: the inflammatory response and clinical implications of the neuro-immuno-humoral axis. World J Surg 2010; 34: 704-720 [PMID: 20049452 DOI: 10.1007/s00268-009-0382-x]

5. Coffey JC, Smith MJ, Wang H, Boucher-Hayes D, Cotter TG, Redmond HP. Cancer surgery: risks and opportunities. Biossage 2006; 28: 433-437 [PMID: 16587986 DOI: 10.1002/bies.20081]

6. van den Tol PM, van Rossen EE, van Eijk CH, Bonhuis F, Marquet RL, Jeekel H. Reduction of peritoneal trauma by using nonsurgical gauze leads to less implantation metastasis
Kahokehr AA. Intraperitoneal wound in abdominal surgery

of spilled tumor cells. Ann Surg 1998; 227: 242-248 [PMID: 9488523 DOI: 10.1097/00000658-199802200-00014]

7 Chuang D, Paddison JS, Booth RJ, Hill AG. Differential production of cytokines following colorectal surgery. ANZ J Surg 2006; 76: 821-824 [PMID: 16922906 DOI: 10.1111/j.1445-2197.2006.03877.x]

8 Baigrie RJ, Lamont PM, Kwiatkowski D, Dallman MJ, Morris PJ. Systemic cytokine response after major surgery. Br J Surg 1999; 79: 799-802 [PMID: 1040367 DOI: 10.1046/j.1365-2109.1999.00837.x]

9 Knowles CH, Aziz Q. Basic and clinical aspects of gastrointestinal pain. Pain 2009; 141: 191-209 [PMID: 19155134 DOI: 10.1016/j.pain.2008.12.011]

10 van der Zanden EP, Snoek SA, Heinsbroek SE, Stanisor OI, Verseijden C, Boeckxstaens GE, Peppelenbosch MP, Greaves DR, Gordon S, De Jonge WJ. Vagus nerve activity augments intestinal macrophage phagocytosis via nicotinic acetylcholine receptor alpha4beta2. Gastroenterology 2009; 137: 1029-1039, e1-1039.e1 [PMID: 19427310 DOI: 10.1053/j.gastro.2009.04.057]

11 Zagon A. Does the vagus nerve mediate the sixth sense? Trends Neurosci 2001; 24: 671-673 [PMID: 11672813 DOI: 10.1016/S0166-2236(00)01929-9]

12 Berthoud HR, Neuhuber WL. Functional and chemical anatomy of the afferent vagal system. Auton Neurosci 2000; 85: 1-17 [PMID: 11189015 DOI: 10.1016/S1566-6736(00)00215-0]

13 Maier SF, Goehler LE, Fleschner M, Watkins LR. The role of the vagus nerve in cytokine-to-brain communication. Ann N Y Acad Sci 1998; 840: 289-300 [PMID: 9629257 DOI: 10.1111/j.1749-6632.1998.tb09569.x]

14 Grundy D, Al-Chaer ED, Aziz Q, Collins SM, Ke M, Taché Y, Wood JD. Fundamentals of neurogastroenterology: basic science. Gastroenterology 2006; 130: 1391-1411 [PMID: 16678554 DOI: 10.1053/j.gastro.2005.11.060]

15 Novitsky YW, Litvin DE, Callery MP. The net immunologic advantage of laparoscopic surgery. Surg Endosc 2004; 18: 1411-1419 [PMID: 15791361 DOI: 10.1007/s00464-005-8275-x]

16 Cervero F, Jänig W. Visceral nociceptors: a new world order? Trends Neurosci 1992; 15: 374-378 [PMID: 1279857 DOI: 10.1016/0166-2236(92)90082-8]

17 Laird JM, Roza C, Cervero F. Effects of artificial calculus on rat ureter motility: peripheral contribution to the pain of ureteric colic. Am J Physiol 1997; 272: R1409-R1416 [PMID: 9176331]

18 Cervero F, Laird JM. Visceral pain. Lancet 1999; 353: 2145-2148 [PMID: 10382712 DOI: 10.1016/S0140-6736(99)01306-9]

19 Hill AG, Connolly AB. Minimal access colonic surgery: is it truly minimally invasive? ANZ J Surg 2006; 76: 282-284 [PMID: 16768679 DOI: 10.1111/j.1445-2197.2006.03711.x]

20 Keus F, de Jong JA, Gooszen HG, van Laarhoven CJ. Laparoscopic versus open cholecystectomy for patients with symptomatic cholecystolithiasis. Cochrane Database Syst Rev 2006; CD006231 [PMID: 17052685]

21 Keus F, de Jong JA, Gooszen HG, van Laarhoven CJ. Laparoscopic versus small-incision cholecystectomy for patients with symptomatic cholecystolithiasis. Cochrane Database Syst Rev 2006; CD006229 [PMID: 17052484]

22 Wu FP, Sietses C, von Blomberg BM, van Leeuwen PA, Meijer S, Cuesta MA. Systemic and peritoneal inflammatory response after laparoscopic or conventional colon resection in cancer patients: a prospective, randomized trial. Dis Colon Rectum 2003; 46: 147-155 [PMID: 12576886 DOI: 10.1053/dcol.2003.4661-2]

23 Sammour T, Kahokehr A, Chan S, Booth RJ, Hill AG. The humoral response after laparoscopic versus open colorectal surgery: a meta-analysis. J Surg Res 2010; 164: 28-37 [PMID: 20828745 DOI: 10.1016/j.jss.2010.05.046]

24 Hill AG, Connolly AB. Minimal access colorectal surgery: is it truly minimally invasive? Dis Colon Rectum 2006; 49: 144-145 [PMID: 16273331 DOI: 10.1010/s10350-005-0208-4]

25 Khan S, Gatt M, MacFie J. Enhanced recovery programmes and colorectal surgery: does the laparoscope confer additional advantages? Colorectal Dis 2009; 11: 902-908 [PMID: 19183327 DOI: 10.1111/j.1463-1318.2009.01781.x]

26 Shindo S, Kubota K, Kojima A, Matsumoto M. A comparison of the inflammatory response and the recovery of bowel function between trans- and extraperitoneal approaches of abdominal aortic aneurysmectomy. Int Angiol 2005; 24: 355-358 [PMID: 16359095]

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