

## INTRA-ABDOMINAL INFECTION AND ACUTE ABDOMEN-EPIDEMIOLOGY, DIAGNOSIS AND GENERAL PRINCIPLES OF SURGICAL MANAGEMENT

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**Abstract:** Intra-abdominal infections are multifactorial and present a complex inflammatory response of the peritoneum to microorganisms followed by exudation in the abdominal cavity and systemic response. Despite advances in management and critical care of patients with acute generalized peritonitis due to hollow viscus perforation, prognosis is still very poor, with a high mortality rate. Early detection and adequate treatment is essential to minimize complications in the patient with acute abdomen. Prognostic evaluation of complicated IAI by modern scoring systems is important to assess the severity and the prognosis of the disease. Control of the septic source can be achieved either by nonoperative or operative means. Nonoperative interventional procedures include percutaneous drainages of abscesses. The management of primary peritonitis is non-surgical and antibiotic treatment. The management of secondary peritonitis includes surgery to control the source of infection, removal of toxins, bacteria, and necrotic tissue, antibiotic therapy, supportive therapy and nutrition. "Source control" is *sine qua non* of success and adequate surgical procedure involves closure or resection of any openings into the gastrointestinal tract, resection of inflamed tissue and drainage of all abdominal and pelvic collections.

**Key words:** Intra-abdominal infection, secondary peritonitis, source control, surgery.

### INTRODUCTION

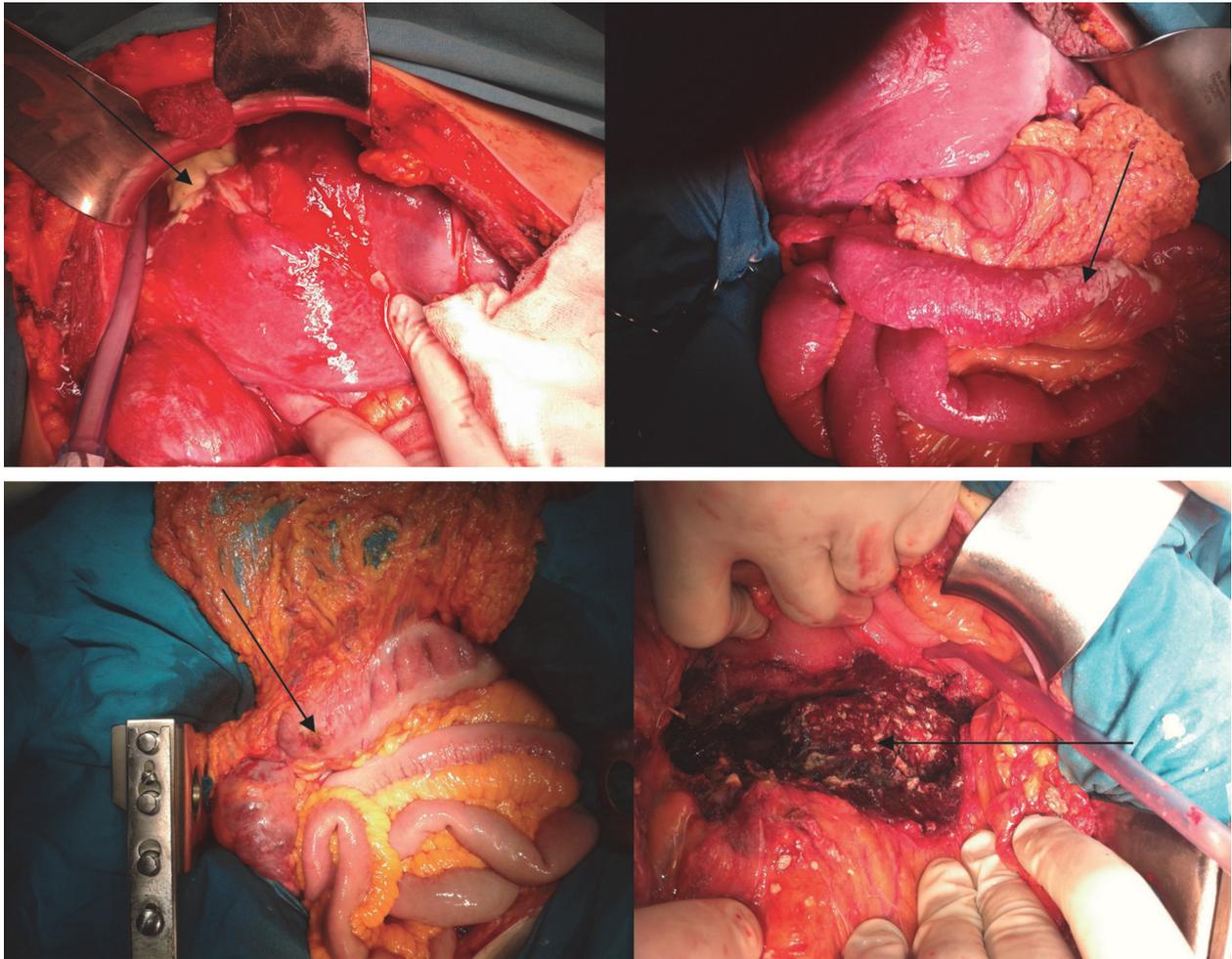
Intra-abdominal infection (IAI) presents a complex inflammatory response of the peritoneum to microorganisms followed by exudation in the abdominal cavity and systemic response (1). Intra-abdominal infection presents a primary, secondary and tertiary peritonitis; in uncomplicated and complicated forms (1). Acute generalized sec-

ondary peritonitis from gastrointestinal hollow viscus perforation is a potentially life-threatening condition. Despite major advances in diagnosis, management and critical care of patients with secondary peritonitis, prognosis is still very poor, with a high mortality rate (1, 2, 3). Source control, resuscitation and early antibiotic administration are crucial (1–4). Intra-abdominal infections are also classified into community-acquired intra-abdominal infections (CAIAIs) acquired in the community and healthcare-acquired intra-abdominal infections (HA-IAIs), develop in hospitalized patients (1–4). They are characterized by increased mortality because of both underlying patient health status and infection caused by multi-drug resistant organisms (4). Uncomplicated IAI involves a single organ and does not proceed to the whole peritoneum, such infections can be successfully treated by surgical resection alone, or with antibiotics alone (5). In complicated IAI the infection spreads from localized peritonitis to the diffuse peritonitis (1, 2). The treatment of patients with complicated intra-abdominal infections necessarily involves both: source control and antibiotic therapy (1, 2, 6). Both, the anatomic source of infection, and to a greater degree the physiological compromise, affect the outcome. The outcome of IAI depends on the severity of the patient's systemic response and his pre-morbid physiological reserves, estimated best using the (7).

This review comments on epidemiology, diagnosis and general principles of surgical management in patients with acute abdomen.

### DEFINITION, CLASSIFICATION, SCORING

The accepted classification of intra-abdominal infection is a division into primary, secondary and tertiary peritonitis (Table 1) (6).



**Figure 1.** Secondary peritonitis. *a.* Perforated liver abscess. *b.* Fibrin on small bowel loops. *c.* Colon perforation. *d.* Infected pancreatic necrosis.  
(Image source: Clinic for Emergency Surgery, Clinical Center of Serbia, Belgrade, Serbia)

Primary peritonitis present the result of the hematogenous bacterial spread to the peritoneum without loss of integrity of the gastrointestinal tract, secondary peritonitis follows a perforation of a hollow organs or cystic structure and tertiary peritonitis is a recurrent infection of the peritoneal cavity that follows either primary or secondary peritonitis (2). Primary peritonitis is rare, and it mainly occurs in early childhood and in cirrhotic adult patients (2). Secondary peritonitis is an acute peritoneal infection resulting from loss of integrity of the gastrointestinal tract due to spontaneous or traumatic organ rupture (Figure 1) (6). It is the most common form of peritonitis. Most frequently encountered in clinical practice as a result of perforation of the duodenal ulcer, or by direct invasion from infected gangrenous appendicitis. Anastomotic dehiscences are common causes of peritonitis in the postoperative period. Secondary peritonitis with severe sepsis or septic shock have mortality rates of approximately 30% (4, 6, 8).

Early prognostic evaluation of complicated IAI is important to assess the severity and the prognosis of

the disease. Scoring systems can be divided into: disease-independent scores for evaluation of serious patients requiring care in the intensive care unit (ICU) such as Acute Physiology and Chronic Health Evaluation II (APACHE II) scoring system and Simplified Acute Physiology Score (SAPS II) and peritonitis-specific scores such as Mannheim Peritonitis Index (MPI) (7, 9). APACHE-II is applied within 24 hours of admission of a patient to an ICU: an integer score from 0 to 71 is based on several measurements; higher scores correspond to more severe disease and a higher risk of death (7). The APACHE-II score has been validated prospectively in a large number of patients and has been adopted by the Surgical Infection Society as the best available method of risk stratification in IAI (7). Although APACHE II is considered as a golden standard, value of this scoring system in peritonitis has been questioned because of the APACHE II impossibility to evaluate interventions, despite the fact that interventions might significantly alter many of the physiological variables (10, 11, 12).

**Table 1.** Classification of intraabdominal infections

PRIMARY PERITONITIS	Diffuse bacterial peritonitis in the absence of disruption of intraabdominal hollow viscera	A. Spontaneous peritonitis in children B. Spontaneous peritonitis in adults C. Peritonitis in patients with CAPD D. Tuberculous and other granulomatous peritonitis
SECONDARY PERITONITIS	Localized (abscess) or diffuse peritonitis originating from a defect in abdominal viscus	A. Acute perforation peritonitis 1. Gastrointestinal perforation 2. Intestinal ischemia 3. Pelviperitonitis and other forms B. Postoperative peritonitis 1. Anastomotic leak 2. Accidental perforation and devascularization C. Post-traumatic peritonitis 1. After blunt abdominal trauma 2. After penetrating abdominal trauma
TERTIARY PERITONITIS	Peritonitislike syndrome occurring late due to disturbance in the host's immune response	A. Peritonitis without evidence for pathogens B. Peritonitis with fungi C. Peritonitis with low-grade pathogenic bacteria

The Mannheim Peritonitis Index (MPI) is a specific score, which provides an easy way to handle with clinical parameters, allowing the outcome prediction. Long-term survivors have an MPI score of about 20; non-survivors have a score of 33. The MPI is specific for peritonitis and easy to calculate, even during surgery (11, 12, 13). Billing et al. demonstrated the reliability of MPI in 2003 patients from 7 centres in Europe (14). For patients with a score less than 21 the mean mortality rate was 2.3%, for score 21–29 mortality was 22.5% and for score greater than 29 mortality was 59.1%; the sensitivity was 86%, specificity 74% and accuracy 83% in predicting mortality (14). Panhofer et al. proposed the use of both MPI and APACHE II in patients who developed tertiary peritonitis, concluding that combination of prognostic scores was very useful to detect tertiary peritonitis (15). Inui et al. investigated the utility of Charlson Comorbidity Index and multiple organ dysfunction (MOD) (16). Among patients who failed initial therapy, a non-appendiceal source of infection and a Charlson score  $> \text{ or } = 2$  were determined to be independent risk factors. Non-appendiceal source of infection and MOD score  $> \text{ or } = 4$  on postoperative day 7 were independent predictors for re-intervention (16).

## PATHOPHYSIOLOGY AND HOST RESPONSE

The total area of the peritoneum is approximately  $1.8 \text{ m}^2$ , which is covered by the mesothelial cells microvilli that measure to 3.0 m in length (2). Peritoneal fluid has the properties of lymph, and is secreted by the peritoneal serosa. Diaphragmatic lymphatic channels act like valves and suck synchronous with respiration peritoneal fluid and any bacteria and pro inflammatory mediators through the thoracic ducts into the venous circulation. Inspiration decreases intra-thoracic pressure relative to intra-abdominal pressure, creating a pressure gradient favoring fluid movement out of the abdomen. Entry of pro-inflammatory substances into the vascular space produces hemodynamic and respiratory findings of sepsis. Positive-pressure ventilation likely attenuates this process. Perforation of the gastrointestinal tract is the most common cause of acute intra-abdominal infection and their highly infectious content flows into the free peritoneal cavity triggers a strong host response (2). The most common causes of acute abdomen are perforation due to peptic ulcer disease, diverticulitis, appendicitis, malignant lesion, bo-

wel wall necrosis after strangulation or incarcerated hernia. The three major intra-peritoneal defense mechanisms are: mechanical clearance of bacteria via lymphatics, phagocytic killing of bacteria by immune cells, and mechanical sequestration (2). The arm of the peritoneal defense system is to localize bacterial contamination. Hyperemia and exudation of fluid follow activation of immune cells. Several events favor the deposition of fibrin, including activation of mesothelial and macro-phage-mediated procoagulant activity acting on fibrinogen in reactive peritoneal fluid, coupled with loss of plasminogen activator from mesothelial cells. The combined effect is the deposition of fibrinous exudates. Ileus and fibrin formation accentuate the process. Formation of an abscess is one of the beneficial functions of fibrin formation encapsulating the infection and preventing systemic spread.

## DIAGNOSIS

Early detection and adequate treatment is essential to minimize complications in the patient with acute abdomen (17–21). A physical examination combined with abdominal ultrasonography (US) represents the initial investigation in patients with acute abdominal pain. The abdomen is distended, it is quiet to auscultation, and tender to palpation. Abdominal pain is almost always the predominant symptom, unless its perception is masked by the administration of analgesics or the presence of a fresh surgical wound. Rupture of a viscus is associated with sudden on-set pain. When fully developed, pain is steady, unrelenting, burning, and aggravated by any motion. Pain is usually most intense in the region of most advanced peritoneal inflammation. Patients can usually localize pain arising from irritation of the parietal peritoneum -peritoneal signs. It may be associated with tenderness and involuntary muscle spasm -guarding. Rebound tenderness can be elicited by gently depressing an area distant from the area of pathology and letting it bounce back. Direct, percussion tenderness and referred rebound tenderness confirms the presence of peritoneal irritation. Rigidity of the abdominal muscles is produced after involvement of the parietal peritoneum by inflammation but also by reflex muscle spasm and abdominal hypertension. Reflex spasm may become so severe that it produces board like abdominal rigidity. Rectal and vaginal examinations are essential to locate the extent of tenderness and the possible presence of a pelvic mass. Anorexia is always present, nausea is frequent and rarely accompanied by vomiting.

Systemic manifestations in complicated IAI are SIRS manifestations: body temperature  $> 38^{\circ}\text{C}$  or  $< 36^{\circ}\text{C}$ , heart rate  $> 90$  beats per minute, respiratory rate  $> 20$

breaths per minute (not ventilated) or  $\text{PaCO}_2 < 32$  mm Hg (ventilated),  $\text{WBC} > 12,000$ ,  $< 4,000$  or  $> 10\%$  immature forms (bands) (18). Bone RC Temperature usually ranges between  $38^{\circ}\text{C}$  and  $40^{\circ}\text{C}$ ; the fever is more spiking in character in younger and healthier patients, whereas older or debilitated patients may exhibit only a modest febrile response. Tachycardia and a diminished palpable peripheral pulse volume are indicative of hypovolemia, hypovolemic shock and sepsis. Respirations are typically rapid and shallow. Hypotension and hypoperfusion signs such as lactic acidosis, oliguria, and acute alteration of mental status are indicative of evolution to severe sepsis (21). A leukocyte count of more than 25,000 or leukopenia of fewer than 4000/mL<sup>3</sup> are both associated with higher mortality. The differential count showing relative lymphopenia and moderate to marked leftward shift, even if the leukocyte count is normal or subnormal. Procalcitonin (PCT) appeared to be a parameter for early detection of progressing sepsis and valuable aid in deciding if further relaparotomies were necessary after initial operative treatment of an intra-abdominal septic focus (19, 22).

Computerized tomography (CT) is the imaging of choice for most intra-abdominal processes in hemodynamically stable patient (17, 20, 21). Diagnostic laparoscopy should be considered in patients without a specific diagnosis after appropriate imaging and as an alternative to active clinical observation which is the current practice in patients with non-specific abdominal pain (17). Plain radiographs of the abdomen may reveal free air on an upright abdominal or lateral decubitus film, a uniform indicator of visceral perforation in the absence of prior intervention (17, 21). The radiological picture of intra-abdominal infection otherwise mimics that of paralytic ileus. Radiographs of the chest may show air beneath the diaphragm if the patient remains in an upright position for 5 min or more before the film (21).

## GENERAL MANAGEMENT

Early treatment of generalized secondary peritonitis may result in a better outcome and any delay may correlate with exponentially increasing mortality (21). Control of the septic source can be achieved either by nonoperative or operative means (21, 23, 24). Nonoperative interventional procedures include percutaneous drainages of abscesses. The management of primary peritonitis, an essentially “non-surgical”, is antibiotic-treatment. The management of secondary peritonitis include surgery to control the source of infection, antibiotic therapy, supportive therapy and nutrition (21, 23, 24).

## SOURCE CONTROL

“Source control” is *sine qua non* of success and adequate surgical procedure involves closure or resection of any openings into the gastrointestinal tract, resection of inflamed tissue and drainage of all fluid collections (25). Laparotomy is usually performed through a midline incision. Timing and adequacy of source control are the most important issues in the management of intra-abdominal infections, because inadequate and late operation may have a negative effect on the outcome (26, 27, 28). The latter aspect of surgical management is controversial, with recent recommendations focused only on the source of infection as opposed to complete peritoneal debridement. Intensive care measures to support tissue oxygenation and maintain organ function remain important, while awaiting recovery brought upon through surgical and antibiotic therapy. Antibiotic therapy should be started as early as possible after diagnosis (26, 27, 28). The trend to continue administration of antibiotics for fixed periods is no longer justified (28). An important rule towards limiting the currently prevailing practices of excessive antibiotic prescription is the recommendation of the Surgical Infection Society that ‘simple’ intra-abdominal infection do not require therapeutic postoperative antibiotics. Antimicrobial regimens effective against common gram-negative and anaerobic enteric pathogens are the mainstay of therapy (26, 27, 28). For patients with community-acquired intra-abdominal infections, narrower-spectrum antimicrobial agents with a low potential for iatrogenic complications are appropriate. Patients with nosocomially-acquired, intra-abdominal infections are more likely to harbor resistant pathogens. Inadequate empiric antimicrobial therapy is associated with treatment failure and death. Therefore, broader spectrum antimicrobial regimens are recommended for these patients, and to coverage of more resistant gram-negative bacilli and anaerobes, use of agents effective against enterococci, resistant staphylococci and *Candida* should be considered (26, 27, 28). Conditions without such peritoneal inflammatory response, in which contamination has occurred but infection is not established, or in which the infectious process remains contained within a diseased, but resectable organ, represent ‘simple’ forms of peritonitis like in appendicitis or cholecystitis, not requiring additional antibiotic therapy for more than 3 to 5 postoperative days (25–28).

## OPERATIVE STRATEGIES

Reduction in mortality is not possible without effective source control (21). The mortality of intra-abdominal infection was about 90% at the end of the 19th

century, when management was mainly non-operative. Source control done in a single operation reduced mortality by more than 50% (21).

The classical, single operation for IAI accomplishes the main goal: surgical source control and a one-time removal of toxins, bacteria, and necrotic tissue (21). The single operation is sufficient in the majority of cases. Only 15% of patients present with advanced disease that require multiple abdominal re-entries (21, 28). The choice of the procedure, and whether the ends of resected bowel are anastomosed, exteriorized, or simply closed, depends on the anatomical source of infection, the degree of peritoneal inflammation and generalized septic response, patient’s comorbid conditions and physiological reserve. All infectious fluids should be aspirated and particulate matter removed by swabbing. Although, cosmetically appealing and popular with surgeons, there is more evidence other than washing out bacteria that intraoperative peritoneal lavage reduces mortality or the incidence of septic complications in patients receiving adequate systemic antibiotics. Drains are still commonly used and misused. In addition to the false sense of security and reassurance they provide, drains can erode into intestine or blood vessels and promote infective complications. Their use should be limited to the evacuation of an established abscess, to allow escape of potential visceral secretions as biliary, or pancreatic and to establish a controlled intestinal fistula when the latter cannot be exteriorized.

Decompression of the abdominal compartment and intra-abdominal hypertension (IAH) is addressed by the decompression methods, mainly the “leaving the abdomen open” techniques (28). Adkins Temporary closure of the abdomen may be achieved using self-adhesive membrane dressings, absorbable meshes, nonabsorbable meshes, zippers and vacuum-assisted closure (VAC) devices (29, 30). Today Vacuum-assisted fascial closure (VAC) has become an option for the treatment of open abdomen (29–32). The surgical treatment strategies following an initial emergency laparotomy may include either a relaparotomy, only when the patient’s condition demands it (“relaparotomy on-demand”), or a planned relaparotomy after 36–48 hours with temporarily abdomen closure or open abdomen (33). Wild STAR (Stage Abdominal Repair) permits continuous control of anastomoses and intra-abdominal healing and effective bacterial elimination (34). This is the only method where post-operative complications are diagnosed early before progressing to major damages. Additionally, peritoneal fluid losses can be measured and protein losses replaced by FFP exactly to match the losses. If a patient develops recurrent peritonitis, a re-intervention is required. This situation has been named “relaparotomy on demand.”

It is associated with high mortality rates because the diagnosis of post-operative peritonitis delayed and patients are operated too late. They often presented with organ failure and advanced disease that is responsible for bad outcome. Ruler et al. published a randomized, clinical trial comparing on-Demand vs Planned Relaparotomy strategy in patients with severe peritonitis (35). The patients in the on-demand relaparotomy group did not have a significantly lower rate of death or major peritonitis-related morbidity compared with the planned relaparotomy group but did have a substantial reduction in relaparotomies, health care utilization, and medical costs (35).

### Source control of appendix perforation

Acute appendicitis is the most common intra-abdominal condition requiring emergency surgery. Delayed diagnosis and treatment of appendicitis also may lead to perforation with diffuse peritonitis. Disseminated intra-abdominal infection from appendicitis, however, is not seen as often today as in the first decades of the 20th century, when appendicitis was the major cause of severe peritonitis and peritonitis-related mortality (36, 37, 38). Although antibiotics may be used as primary treatment for selected patients with suspected uncomplicated appendicitis, appendectomy is still the gold standard therapy for acute appendicitis (36, 37, 38). Treatment includes source control by appendectomy, rarely in the most severe cases staged abdominal repair in cases when peritoneal edema has led to abdominal compartment syndrome. Studies have demonstrated that antibiotics alone may be useful to treat patients with early, non perforated appendicitis, even if there is a risk of recurrence (36, 37, 38). Randomized clinical trial by Hanson et al. compared antibiotic therapy versus appendectomy as primary treatment of acute appendicitis (39). Treatment efficacy was 90.8% for antibiotic therapy and 89.2% for surgery. Recurrent appendicitis occurred in 13.9% of patients treated conservatively after a median of 1 year (39). The course of appendicitis leading to massive necrosis and life threatening infection, often surpassing the omentum's capability to contain the infection and form a perityphlic abscess and diffuse suppurative peritonitis then results (40, 41). Appendix abscess occurs in 10% of patients with acute appendicitis (40). There is much controversy whether interval appendicectomy is appropriate for adults with an appendiceal abscess. The traditional management of appendiceal mass has been initial conservative treatment followed by interval appendicectomy (40). Deakin et al. demonstrated that conservative management approach was successful in the majority of patients presenting with an appendix mass (42).

The authors concluded that after initial successful conservative management, routine use of interval appendicectomy was not justified in asymptomatic patients (42).

### Source control of gastro-duodenal perforation

Gastroduodenal perforations have decreased significantly in the last years thanks to the widespread adoption of medical therapies for peptic ulcer disease and stress ulcer prophylaxis among critically ill patients. Successful laparoscopic repair of perforated gastric and duodenal ulcers has been reported but the technique has yet to be universally accepted (43, 44, 45). This form of peritonitis is initially chemical but in a short time becomes infected. The proper management is simple closure. Antibiotic therapy may be given over a very short period in the range of 1 to 3 days because bacterial numbers are generally small and the source can be closed safely. The high mortality rate of anastomotic leakage or suture line breakdown after gastro-duodenal operations Billroth I and Billroth II resections are explained by the fact that the duodenum is retroperitoneally fixed and cannot be exteriorized, and the source of infection often cannot be adequately controlled or closed.

### Source Control of Colon Perforation

Antibiotics are the standard of care for uncomplicated diverticulitis (46). Percutaneous drainage is the intervention of choice for simple uniloculated abscesses (46). It has a success rate of more than 80%, but it may have a high failure rate in cases of complex multiloculated or inaccessible abscesses (46). Colon perforation due to diverticulitis or cancer is a common cause of diffuse, fecal peritonitis (Figure 1). Urgent surgery for colonic diverticula perforations is indicated in patients with large or/and multiloculated diverticular abscesses inaccessible to percutaneous drainage or in whom clinical symptoms persist after CT guided percutaneous drainage, diverticulitis associated with free perforation and purulent or fecal diffuse peritonitis. This factor, together with the many associated diseases in the population of elderly patients with colon disease, contributes to the high mortality rate of 37% (46). There is still controversy about the optimal surgical management of peritonitis caused by colonic diverticular disease (46, 47). Hartmann's resection has been the procedure of choice in patients with generalized peritonitis and remains a safe technique for emergency colectomy in perforated diverticulitis, especially in elderly patients with multiple co-morbidities. This group of patients particularly benefits from staged abdominal

repair for which the overall mortality rate is less than 20% (46). Antibiotic therapy needs to cover both facultative and obligate anaerobic bacteria and can be discontinued after five days duration (46). More recently, some reports have suggested that primary resection and anastomosis is the preferred approach to diverticulitis, even in the presence of diffuse peritonitis (46, 47).

### Peritonitis of Biliary Origin

Because of the delay of diagnosis, biliary peritonitis is associated with mortality rates exceeding 30%. Early suspicion is key. Removal of the gallbladder and common duct stones represents effective source control (48–54). Early laparoscopic cholecystectomy for acute cholecystitis is safer and shows lower rates of conversions than delay laparoscopic cholecystectomy (50). Half of the mortality of biliary peritonitis is caused by *E. coli* and one fourth by *clostridium perfringens*, which may cause a fulminate necrotizing infection leading to death within hours (50–54). The latter may require immediate re-operation, high doses of penicillin (10 million units every 6 hours) Intensive Care and hyperbaric oxygen. Most cases of acute cholecystitis as a complication of Intensive Care are acalculous and likely represent complications of microvascular and mucosal dysfunction. This condition, often presents during sepsis originating in the necrotic gallbladder that becomes infected and must be removed for effective source control. Despite the evidence, early laparoscopic cholecystectomy is not the most common treatment for acute cholecystitis in practice and wrongly it remains common practice to treat acute cholecystitis with intravenous antibiotic therapy and interval laparoscopic cholecystectomy preferentially (55).

### Source Control of Infected Pancreatic Necrosis

In the acute phase of pancreatitis, antibiotic therapy, supportive therapy and intravenous hydration aimed at maintaining the patient's intravascular volume and perfusion pressures is the mainstay of treatment. Usually during the third week after onset of acute pancreatitis, the disease has progressed from initial chemical inflammation to intraabdominal infection with high mortality because source control is difficult (Figure 1) (56). Endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy are indicated in patients with biliary pancreatitis and impacted gall stones, biliary sepsis, or obstructive jaundice (56). In septic patients with necrotizing pancreatitis, a Fine-needle aspiration (FNA) should be performed for differentiation of sterile and infected pancreatic necro-

sis (56). Adequate volume resuscitation and analgesic treatment are the most important treatment of acute pancreatitis. Antibiotic prophylaxis reduces septic complications in severe necrotizing pancreatitis and should be started early, best with 1 gram of imipenem/cilastatin every 6 to 8 hours at the onset of acute pancreatitis before infection can be proven. Antibiotics must be administered for longer periods (56). Surgical therapy is indicated in patients with infected pancreatic necrosis (56, 57). The optimal time point for the surgical intervention is the 3rd to 4th week after onset of the disease, in that time necrotic tissue will be well demarcated. The surgical technique of choice is necrosectomy with postoperative closed lavage (56, 57, 58). Before closure, large abdominal drains must be placed into the pancreatic bed to further drain necrotic areas and to collect pancreatic juice preventing further intra-abdominal spread of digestive enzymes. When most necroses are removed or overgrown by granulation tissue, usually after 8–12 abdominal entries if STAR is done, and peritoneal edema disappears, the abdomen can be closed fascia-to fascia without meshes. Cholecystectomy should be performed to avoid recurrence of gallstone-associated acute pancreatitis.

### Source Control of Small Bowel Perforation

Most non-traumatic small intestinal perforations are due to unrecognized bowel strangulation and intestinal ischemia (59, 60, 61). Source control includes resection of the diseased segment and anastomosis (59, 60, 61). The high mortality rate of more than 50% can be reduced by treating advanced cases with the STAR operation that permits inspection of the anastomosis at subsequent abdominal entries, and diagnose and treat new necrosis early. In addition, anastomoses can be deferred and the bowel simply stapled off at the first STAR in deteriorating patients who will not tolerate extensive procedures during sepsis. If small bowel perforations operated before peritonitis develops it can have an excellent prognosis. Source control is usually achieved by simple suture closure, rarely by resection of the perforated segment and anastomosis.

### Source Control in Postoperative Peritonitis

Postoperative peritonitis is usually due to a leak from a suture line (4, 62). Diagnosis is often delayed, and patients, as a rule, are re-explored between the fifth and seventh postoperative day, which contributed to the high mortality rate. A suture line leak is easier to repair if it is observed in the colon, small bowel or stomach

compared with leaks of the duodenum or esophagus. Upper gastrointestinal tract disease after an operation allows only a limited therapeutic correction to control the source, because these organs are fixed or closely attached to the retroperitoneum and the infectious source cannot be totally excluded under most circumstances. Resection of the anastomosis or bowel segment is better than repair. Staged abdominal repair using a temporary abdominal closure device may be of particular benefit to this subset of patients with intraabdominal infections; we were able to reduce mortality to 24% (62).

### Source Control in Posttraumatic Peritonitis

Peritonitis caused by intestine perforation may develop in patients after blunt abdominal trauma (63). This type of intraabdominal infection is usually severe because it is masked by other injuries and often recognized late, even when an initial CT was done. This causes delay in diagnosis and most cases present with a diffuse peritonitis (63). General principles of treatment do not differ from that of intraabdominal infection (63). Contamination of the abdominal cavity seen after penetrating abdominal trauma is not considered an intraabdominal infection.

In a conclusion, intra-abdominal infections present a complex inflammatory response of the peritoneum to microorganisms. Despite all advances in management of patients with secondary peritonitis, prognosis is still very poor, with high mortality rate. Acute generalized secondary peritonitis is still very interesting for surgeons and adequate source control is *sine qua non* of treatment.

### Abbreviations

**IAI** — Intra-abdominal infection

**CAIAIs** — community-acquired intra-abdominal infections

**HA-IAIs** — healthcare-acquired intra-abdominal infections

**ICU** — intensive care unit

**APACHE II** — Acute Physiology and Chronic Health Evaluation II

**SAPS II** — Simplified Acute Physiology Score

**MPI** — Mannheim Peritonitis Index

**MOD** — multiple organ dysfunction

**VAC** — vacuum-assisted closure

**STAR** — Stage Abdominal Repair

### Sažetak

## INTRA-ABDOMINALNA INFEKCIJA I AKUTNI ABDOMEN-EPIDEMIOLOGIJA, DIJAGNOZA I OPŠTI PRINCIPI HIRURŠKOG REŠAVANJA

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Intra-abdominalne infekcije (IAI) su multifaktorijske i predstavljaju složeni inflamatorni odgovor peritoneuma na prisustvo patogenih mikroorganizama, sa eksudacijom u trbušnu duplju i sistemskim odgovorom. Uprkos napretku u zbrinjavanju pacijenata sa akutnim difuznim peritonitisom koji je posledica perforacije šupljeg trbušnog organa, prognoza je i dalje veoma loša, sa visokom stopom smrtnosti. Rano postavljanje dijagnoze i adekvatno lečenje su od suštinskog značaja za minimiziranje komplikacija kod pacijenta sa akutnim abdomenom. Prognostička evaluacija komplikovanih IAI savremenim bodovnim sistemima je važna zbog procene težine bolesti i prognoze. Kontrola izvora infekcije može biti ne-hirurška i hirurška. Ne-hirurške interventne procedure uklju-

čuju drenažu gnojne kolekcije/apscosa. Lečenje sekundarnog peritonitisa je kompleksno i uključuje: hiruršku kontrolu izvora infekcije, uklanjanje nekrotičnog tkiva i detritusa iz trbuha, antibiotsku terapiju, supsticionu i simptomatsku terapiju, i ishranu. „Kontrola izvora infekcije“ je *sine qua non* adekvatnog lečenja, a obuhvata: suturu mesta perforacije šupljeg organa gastrointestinalnog trakta, resekciju ishemično-nekrotičnog tkiva, uklanjanje organa u celini (*appendectomy*, *cholecystectomy*), lavazu trbuha u slučaju difuznog peritonitisa, kao i drenažu sva četiri kvadranta, uz plasiranje kontaktnog drena na mestu hirurškog rada.

**Ključne reči:** Intraabdominalna infekcija, sekundarni peritonitis, kontrola izvora, hirurgija.

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