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Short courses of antibiotics in intra-abdominal infection: review and update

Ciclos cortos de antibióticos en infección intrabdominal: revisión y actualización

Aldo Israel Olán-De Los Santos,* Enrique Corona-Díaz,‡ Dafne Alejandra Torres-Torres,§ Daniel Antonio Domínguez-Díaz§

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* Third-year resident of General Surgery, *Hospital General Tacuba*, *Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado* (ISSSTE). Mexico City.
‡ First year Internal Medicine Resident, *Hospital General "Dr. Gaudencio González Garza" Centro Médico Nacional La Raza, Instituto Mexicano del Seguro Social*. Mexico City.
§ Fourth-year resident of General Surgery, *Hospital General Tacuba*, ISSSTE. Mexico City.

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ABSTRACT

Intra-abdominal infections have different etiologies, which can manifest in a complicated form with the appearance of peritonitis and sepsis of abdominal origin. The concept of abdominal sepsis has been modified to the present day, where it is defined as a focus of abdominal infection that results in two or more points on the qSOFA scale and septic shock if vasopressors are required for the maintenance (regardless of the amount of adequate resuscitation) of mean arterial pressure. The choice of the duration of antibiotic treatment is made empirically with duration periods of up to 14 days, conditioning multiple consequences such as bacterial resistance, superinfection, adverse effects related to antibiotic treatments, and increased costs associated with treatments, to name a few. There are several recommendations for clinical guidelines for the duration of treatments; however, few satisfactory methodological studies are found in the literature. Even so, multiple randomized trials have concluded that short-term treatment with antibiotic therapy until the resolution of physiological abnormalities is not inferior to conventional treatment. A review of published manuscripts was carried out, among which those with the highest level of evidence and degree of recommendation on abdominal sepsis and the establishment of administration of shorter schemes of antibiotic treatment compared to conventional schemes in cases of intra-abdominal infections were selected.

RESUMEN

Las infecciones intrabdominales tienen distintas etiologías, las cuales pueden manifestarse en una forma complicada con la aparición de peritonitis y sepsis de origen abdominal. El concepto de sepsis abdominal ha sido modificado hasta la actualidad donde se define como aquel foco de infección abdominal que condiciona dos o más puntos en la escala qSOFA y choque séptico si se requieren vasopresores para el mantenimiento (independientemente de la cantidad de reanimación adecuada) de la presión arterial media. La elección de la duración del tratamiento antibiótico se hace de forma empírica con periodos de duración de hasta 14 días, condicionando múltiples consecuencias como resistencia bacteriana, sobreinfección, efectos adversos relacionados con los tratamientos antibióticos, incremento en los costos asociados con los tratamientos, etc. Existen varias recomendaciones de pautas clínicas para la duración de los tratamientos; sin embargo, pocos estudios metodológicos satisfactorios se encuentran en la literatura. Aun así, actualmente múltiples ensayos aleatorizados concluyeron que el tratamiento a corto plazo con terapia antibiótica hasta la resolución de las anomalías fisiológicas no era inferior al tratamiento convencional. Se realizó una revisión de manuscritos publicados, entre los cuales fueron seleccionados aquellos con mayor nivel de evidencia y grado de recomendación sobre la sepsis abdominal y la instauración de la administración de esquemas más cortos de tratamiento antibiótico comparado con los esquemas convencionales en casos de infecciones intraabdominales.

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Abbreviations:
qSOFA = quick SOFA.
SOFA = Sequential Organ Failure Assessment.
SIRS = systemic inflammatory response syndrome.

INTRODUCTION

There is no solid scientific data to determine the duration of antibiotic treatment,[1-3] and empirical considerations determine the duration of antibiotic therapy in patients with septic infectious processes.[4] Previously, most experts advised a treatment duration of more than 14 days for hospital-acquired infections.[5] Organizations such as the World Health Organization (WHO)[6] have established limitations of antibiotic treatment since overuse of antibiotics can cause antibiotic resistance (as in the case of fluoroquinolones and carbapenems),[4,5] which is a natural biological reaction. However, with the rapid emergence of bacterial resistance, prevention of unwanted toxic effects and improvement of quality of life, achieving a decrease in their use by reducing the period of exposure should be a significant concern, considering that prolonged use of antibiotics in intensive care patients may increase the risk of infection by multidrug-resistant organisms (*Pseudomonas aeruginosa*, *Acinetobacter*, methicillin-resistant *Staphylococcus aureus*, *Enterobacteriaceae*, and *Clostridium difficile*). In addition, due to the low costs and associated side effects, short-term treatments are gaining popularity.[4,7]

Intra-abdominal infections have a variety of causes, including spontaneous and postoperative infections caused by intra-abdominal visceral perforations or primary infectious foci in the abdomen.[8] Although these infections have different origins, there are similar management methods to control the infectious focus, such as administering antibiotic treatment and draining fluid from the abdominal cavity. These infections, in part due to the variety of pathologies they cause, are difficult to control and have a high rate of morbidity and mortality.[9]

The recommended duration of antibiotic treatment for intra-abdominal infections is controversial, and unfortunately, few satisfactory methodological studies are found in the literature, and the methodology used in these studies is poor; however, multiple randomized trials concluded that short-term treatment with antibiotic therapy until resolution of physiological abnormalities was not inferior to conventional treatment.[10] There are several clinical guideline recommendations for the diagnosis and treatment of complex intra-abdominal infections that suggest short-term antibiotic treatment. In immunocompetent patients with adequate local control, three days of antibiotic therapy may be sufficient for mild to moderate intra-abdominal infection.[11] In stable patients with severe infections, antibiotic treatment can be discontinued after five days, when bowel function is restored and the inflammatory response subsides. Patients with advanced necrotizing retroperitoneal infection, those whose primary focus is not adequately controlled, and cases with tertiary peritonitis who have received repeated or refractory therapeutic interventions may require long-term treatment.[12-16] Therefore, the ideal duration of postoperative prophylaxis after a surgical event secondary to intra-abdominal infection is not established. To evaluate the scientific evidence in this field, we reviewed current scientific articles, including controlled clinical trials and review articles.

TERMINOLOGY

Intra-abdominal infections

They are those infections found in the abdomen, including intraperitoneal infections that originate only in the cavity covered by the visceral and parietal peritoneum (duodenum, small intestine, colon, rectum, liver, spleen, and bile duct), and retro or extraperitoneal infections (posterior duodenum, posterior colon, pancreas, kidneys, aorta, cava).[17-19] Intra-abdominal infections arise from three sources which can be endogenous gastrointestinal microbiota colonies, acquired in an external environment secondary to the hospital in a community setting in cases such as trauma,

as well as cases in which the infection occurs within a hospital, at any time between 48 hours after the patient's admission and 30 days after discharge or during immediate hospitalization after surgery, and is called nosocomial infection. Intra-abdominal infections can be simple if they are localized and do not penetrate the serosa or fascia of the tissue of origin (inflammatory processes without viscera perforation), or complex if they manifest as abscesses or generalized contamination of the abdominal cavity peritonitis.[20] Primary peritonitis is a diffuse primary abdominal cavity infection that does not develop or originate from other intra-abdominal infections. Secondary peritonitis results from perforation of infected or necrotic abdominal viscera with externalization of their contents into the abdominal cavity. Tertiary peritonitis refers to persistent secondary peritonitis and re-infection due to failure of previous antimicrobial treatment.[21]

Sepsis

Originally sepsis was defined by the presence of an identified source of infection and systemic inflammatory response syndrome (SIRS). Subsequent organ failure is termed severe sepsis and SIRS due to cardiac and circulatory failure is termed septic shock.[22] However, due to its low specificity and the SIRS criteria proving unsatisfactory in explaining many manifestations of sepsis, as it was recognized that organ dysfunction may be the first symptom observed, it has now been modified. In the third international consensus on the definition of sepsis and septic shock (Sepsis-3),[23] specific sepsis is defined as a life-threatening organ dysfunction resulting from a dysregulated host response to infection. Organ dysfunction is measured by the Sequential Organ Failure Assessment Scale (SOFA) score; however, the SIRS criteria were removed from the definition and SOFA scale, and the authors of Sepsis-3 proposed the qSOFA *(Figure 1)* as a screening tool for sepsis with the following criteria: a) changes in mental status (Glasgow coma < 15), b) respiratory rate ≥ 22 breaths per minute, and c) systolic blood pressure less than 100 mmHg. An increase in score of two or more points is considered "life-threatening".

The classic sequence of infection, sepsis, severe sepsis, and septic shock was replaced by the concepts of infection, sepsis, and septic shock. The concept of "severe sepsis" was eliminated.[24]

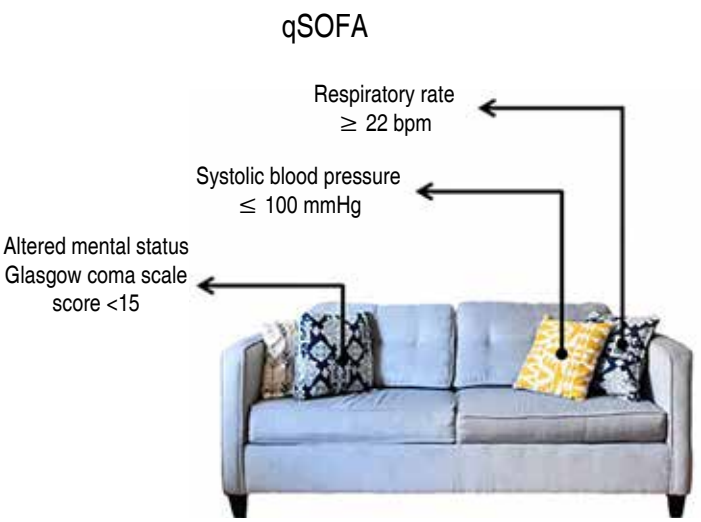

Figure 1: qSOFA scale for the diagnosis of sepsis. Two or more points and identification of an infectious focus are criteria for diagnosing sepsis.
qSOFA = quick SOFA. bpm = breaths per minute.

Abdominal sepsis

It is an intra-abdominal infection that causes qSOFA to increase by two or more points. The clinical condition is defined as a septic shock if vasopressors are required for the maintenance (regardless of the amount of adequate resuscitation) of mean arterial pressure (MAP) of at least 65 mmHg and serum lactate greater than 2 mmol/l.[25]

Short courses of antibiotics

This term refers to administering antibiotics in shorter courses than standard empiric antibiotic courses (seven to 10 days or more) to prevent complications. Shorter courses may reduce side effects and costs, but there is concern that they may reduce the chances of remission of infection and increase the

risk of recurrence. Different studies and clinical trials have shown that short courses of antibiotics (different periods ranging from two to five days) are as effective as a standard course that can prevent infection without increasing relapse.

EPIDEMIOLOGY

Intra-abdominal infections remain one of the main etiologies of sepsis, being a morbidity and mortality problem. Of the 11 million sepsis-related deaths worldwide, intra-abdominal sepsis is the second leading cause of death from sepsis. This outcome is because it is a serious medical-surgical emergency that affects the whole organism, and even in our times, health professionals do not fully understand its management.[26]

The evidence until 2010 came from literature from developed countries, where 2.8 million deaths were attributed to sepsis; the incidence of sepsis is higher because many suspected cases go unreported. Recently published epidemiologic data suggest that sepsis causes one-third to one-half of hospital mortality in the US. Estimates suggest that about 1,400 patients die per day from sepsis worldwide. It has been observed that in 66% of all post-surgical patients with sepsis, an intra-abdominal infectious focus could be detected.[25]

The abdomen has a massive microbial population that is extremely sensitive to shock and stress. Its management is challenging from an anatomical and physiological point of view, due to factors that influence the gastrointestinal tract such as intra-abdominal pressure, shock and altered perfusion.[26]

Acute appendicitis is one of the most common emergency surgeries in both children and adults worldwide. The treatment of choice remains surgery, although it has recently been questioned. In 2015, 378,000 cases of appendicitis were recorded in the USA. On the other hand, in the Netherlands approximately 12,000 appendectomies are performed each year for acute appendicitis.

Acute appendicitis is classified during surgery into simple and complicated. An edematous and phlegmonous appendicitis would fall into the group of simple acute appendicitis. In contrast, a gangrenous and/or perforated appendix, as well as any appendicitis with an intra-abdominal or pelvic abscess, would be considered as a complicated acute appendicitis. About 25-30% of all patients with appendicitis have complicated appendicitis, which is associated with an increased risk of postoperative infectious complications. Therefore, in complicated appendicitis, the use of antibiotics during the postoperative period is recommended.[7]

DURATION OF ANTIBIOTIC TREATMENT

The first antibiotic tapering schemes were applied to infections such as tonsillitis which can be treated for 3-5 days with azithromycin,[27] single dose treatments for sexually transmitted infections (syphilis, chlamydia, or gonococcus) and lower urinary tract infections, or infectious diarrhea which can be treated with a single dose of fluoroquinolone.[28]

Limiting the duration of antibiotic treatment to the minimum necessarily has the following advantages:[8]

1. To reduce antibiotic exposure in the population and thereby limit the emergence of bacterial resistance.[29,30]
2. To reduce the number of adverse effects related to antibiotic treatments (superinfection by *Clostridium difficile*, tendinopathies, morbidity, mortality, and others).
3. To reduce costs associated with treatments.
4. To improve compliance with antibiotic treatment.

Therefore, the short duration of antibiotic treatment is a benefit/risk trade-off between the risk of individual failure versus collective control of bacterial resistance and the reduction of adverse effects and costs.[31]

Maceda and Gilsanz comment in favor of short-duration antibiotic treatments as effective and safe for the control of the infectious focus and the decrease of the bacterial load since, in an individual with

an appropriate immune response and after adequate control of the focus, the residual inoculum may respond to shorter antibiotic treatment.[32]

The evidence on the optimal duration of antibiotic treatment in intra-abdominal infection with spontaneous peritonitis shows that no significant differences were found between groups treated with antibiotics for five and ten days.

Secondary intra-abdominal infection evaluates the intraoperative findings and clinical evolution as a guide for the duration of antibiotic treatment. Intraoperative findings during the initial operation depend on localized or more extensive peritonitis; in the case of the former, after receiving antibiotic management for only two days and in the latter a five-day regimen, no increase in the failure rate was demonstrated compared to what was previously seen, except for the appearance of a subhepatic abscess and three surgical wound infections.[33]

For evaluating clinical evolution, the parameters used are normalization of the white blood cell count, lack of fever, and recovery of intestinal function; with a low risk of therapeutic failure in afebrile individuals, with normal white blood cell count, and as soon as signs of infection disappear, the withdrawal of antibiotics is as effective as antibiotic therapy of a predetermined duration.

Alcocer and Maseda recommend a mean duration of antibiotic treatment of five days in patients with extensive peritonitis when antibiotic treatment is guided by clinical evolution based on clinical and laboratory indicators.[34,35]

DIAGNOSIS

Currently, diagnosis is based on adopting a uniform and unequivocal definition of sepsis to facilitate early recognition. There is no reference diagnostic test, although clinical signs and symptoms in patients with suspected infection can be identified.

The non-specific criteria for SIRS indicate the presence of infection; however, SIRS may simply reflect an appropriate host response, which is often adaptive.[23,36] The Quick SOFA sepsis identification score was recently proposed in 2016 as a parameter to assess the initial high-risk likelihood of patients with suspected sepsis. A low score does not rule out the possibility of sepsis, so further patient examination is recommended if suspicion persists. In contrast, high scores call for more specific treatment measures, including lactate measurement, specific antibiotic therapy, intravenous fluid resuscitation with or without the use of vasopressor amines, and SOFA (sepsis-related organ failure assessment) evaluation *(Table 1)* to estimate organ dysfunction; a SOFA score ≥ 2 points is a parameter of sepsis.[37,38]

CHOICE OF ANTIBIOTIC TREATMENT

Anecdotal and published observations suggest that many patients receive longer "prophylactic" courses of antibiotics when they are thought to be at particularly high risk for septic complications. This practice has led to the overuse of antibiotics, increased risk of developing bacterial resistance, and excessive costs associated with treatment.

The published scientific evidence on the duration of empirical antibiotic treatment in surgical intra-abdominal infections with effective control of the focus recommends that this should be as limited as possible in patients without risk factors and evaluated individually in patients with risk factors.[38] The advisable duration of antibiotic treatment in intra-abdominal infection is controversial. Consensus has not been reached due to the absence of controlled studies determining sufficient scientific evidence.

Although the origins of these infections differ, there are similar management strategies aimed at controlling the origin, such as intra-abdominal fluid drainage and antibiotic administration.[39] Early empirical antibiotic therapy should be initiated, within the first hour of recognition of sepsis and persistent septic shock, depending on the infection focus and source. According to the most frequently isolated bacteria, intravenous antibiotic therapy should be administered immediately to prevent

Table 1: SOFA (sequential organ failure assessment) scale for organ dysfunction.

Criteria	Score 0	1	2	3	4
Neurological					
Glasgow coma scale	15	13-14	10-12	6-9	< 6
Renal					
Creatinine (mg/dl)	< 1.2	1.2-1.9	2-3.4	3.5-4.9	> 5
Uresis (ml/day)				< 500	< 200
Hepatic					
Total bilirubin (mg/dl)	< 1.2	1.2-1.9	2-5.9	6-11.9	> 12
Coagulation					
Platelets (10^3/mm^3)	≥ 150	< 150	< 100	< 50	< 20
Respiratory					
PaO_2/FiO_2 (mmHg)	≥ 400	< 400	< 300	< 200 + invasive mechanical ventilation	< 100 + invasive mechanical ventilation
Cardiovascular					
Mean blood pressure (mmHg)	≥ 70	< 70	Use of vasopressor amines		

PaO_2/FiO_2 = ratio between the arterial oxygen partial pressure and the fraction of inspired oxygen.

morbidity and mortality, followed by treatment with specific antibiotics, according to the culture results and susceptibility profile.[40]

The main pathogens of community abdominal infections are gastrointestinal microbiota such as *Enterobacteriaceae*, *Streptococcus*, and anaerobic bacteria such as *Bacteroides fragilis*. Extended-spectrum beta-lactamase (ESBL)-producing *Enterobacteriaceae* are the most resistant.[41,42]

Recommendations for the use of antibiotic therapy include broad-spectrum drugs with good penetration at the site of suspected infection and re-evaluation of antibiotic regimens daily, at appropriate doses and tapering strategies since possible pathophysiological changes may significantly alter the drug profile in critically ill patients, ultimately preventing resistance, avoiding toxicity and reducing the cost of infection individually according to the clinical course.[40,43]

In adults with sepsis without shock, the likelihood of infectious versus non-infectious causes of acute illness should be assessed. In case of high suspicion, antimicrobials should be administered within three hours.[43]

MICROBIOLOGY OF INTRA-ABDOMINAL INFECTIONS

Community-acquired intra-abdominal infections is led by Gram-negative bacteria, mainly *Escherichia coli* (25-30%), followed by *Klebsiella* species and *Pseudomonas aeruginosa* (3-6%). Anaerobic microorganisms come mainly from the *Bacteroides fragilis* group, which ranks third in frequency of microbial culture (8.6-14.3%). Among Gram-positive bacteria, *Streptococcus* is the most prominent (16%), followed by *Staphylococcus spp.* (5.2%) and to a lesser extent, *Enterococcus spp.* (4.7%), mainly *Enterococcus faecalis*.[44,45]

In intra-abdominal infections of nosocomial origin due to peritonitis and postoperative abscesses, the dominant bacterium is still *E. coli* (22%) together with *Enterobacter spp.* (12%). *Bacteroides fragilis* was present to a lesser extent (5.5%), and *Enterococcus spp.* had a higher prevalence (17%), including *Enterococcus faecium*. On the other hand, *P. aeruginosa* has a greater resistance.[44,46-48]

ETIOLOGY

It is important to recognize abdominal sepsis as a clinical entity resulting from multiple factors that interact with each other in a complex manner. Therefore, the range of alterations that may cause is extensive.[39,49]

The gastrointestinal tract is recognized worldwide as the main site of origin of intra-abdominal infectious processes leading to sepsis, with acute appendicitis being the etiology with the highest incidence.[48]

Spontaneous perforation is a determinant for the onset of the infectious process; some of the most common causes are complicated diverticular disease, peptic ulcers, and open or closed abdominal trauma, and it can even be a complication associated with surgical interventions.[39] All these entities are usually associated with polymicrobial infections with the presence of Gram-negative enterobacteria, *Enterococcus* and *Staphylococcus*, in addition to other anaerobic microorganisms and *Candida*.[49]

Cases where the intra-abdominal process is not characterized by a structural lesion of a hollow viscera, as in spontaneous bacterial peritonitis, are less frequent.[49]

As for the rest of the intra-abdominal organs, urinary tract infections are the second cause of septic processes, followed by pathologies such as cholangitis, pancreatitis, and hepatic abscesses.[39]

PATHOPHYSIOLOGY

As previously mentioned, developing a septic process involves participating in multiple components dynamically and evolving in conditions of variable severity.[47] Current theories consider the host immune response as the main determinant of the severity of the septic process; the response depends on factors intrinsic to the organism (e.g., genetic characteristics and concomitant pathologies)[49] and factors of the causative pathogen (e.g., virulence and amount of inoculum).[50]

The anatomical location of the initial lesion is also an important determinant since perforations at higher levels of the digestive tract, such as the stomach and duodenum, usually have less serious consequences, while perforations at the level of the colon and rectum result in severe bacterial contamination.[48]

Despite being a systemic process, the pathophysiology varies between organs and systems, with different responses to infection at local and regional levels.[47] In general, the inflammatory process usually remains initially contained in the peritoneal cavity when there is an interaction between the molecular patterns associated with the pathogen and the receptors expressed on the cell surface, in the endosome, or the cytoplasm, initiating the release of multiple proinflammatory cytokines whose objective is the elimination of the pathogen, which are also responsible for tissue injury as collateral damage.[50]

Microorganisms often have mechanisms that allow them to adhere to the human endothelium. This process causes dysregulation of normal endothelial homeostasis, characterized by loss of cell barrier integrity and apoptosis maintaining a sustained release of cytokines that perpetuate the immune response and a state of vasodilatation, and promoting coagulation abnormalities.[49] Recurrent tissue factor-mediated activation of coagulation, reduced activity of endogenous anticoagulant pathways plus impaired fibrinolysis facilitate all thrombosis of the microvasculature resulting in tissue hypoperfusion, which is aggravated by hypotension resulting from systemic vasodilatation. This tissue hypoperfusion is the basis of organ failure that occurs when the septic process is not controlled in time.[50]

Some theories consider that anti-inflammatory mechanisms are activated to control the excessive inflammatory response, but they could also be one of those responsible for increasing the risk of secondary infections in patients with severe sepsis.[50]

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Correspondence:
Aldo Israel Olán-De Los Santos
E-mail: aldoi27@hotmail.com