World J. Surg. 22, 158–163, 1998

WORLD
Journal of
SURGERY



# Tertiary Peritonitis: Clinical Features of a Complex Nosocomial Infection

Avery B. Nathens, M.D., Ori D. Rotstein, M.D., John C. Marshall, M.D.

Department of Surgery, University of Toronto and the Surgical Intensive Care Unit, Eaton North 2-934, Toronto General Hospital, 200 Elizabeth Street, Toronto, Ontario, Canada M5G 2C4

**Abstract. The objective of this study was to define risk factors for and the clinical course of recurrent or tertiary peritonitis. Intensive supportive care of patients with life-threatening intraabdominal infections has led to the emergence of a new clinical syndrome, tertiary peritonitis, defined as the persistence or recurrence of intraabdominal infection following apparently adequate therapy of primary or secondary peritonitis. We undertook a retrospective study of 59 patients admitted with intraabdominal infection to a surgical intensive care unit (ICU). Tertiary peritonitis developed in 74% (44/59) of patients. Despite comparable premorbid health status, source of peritonitis, and admission APACHE II scores, patients with tertiary peritonitis had a significantly longer ICU stay (21.8 ± 14.9 vs. 8.5 ± 7.9 days), more advanced organ dysfunction reflected in higher organ dysfunction scores (13.3 ± 5.1 vs. 7.7 ± 3.3), and higher ICU mortality (64% vs. 33%) than patients with uncomplicated secondary peritonitis. The most common infecting organisms in patients with tertiary peritonitis were *Enterococcus*, *Candida*, *Staphylococcus epidermidis*, and *Enterobacter*. Infectious foci were rarely amenable to percutaneous drainage and were found to be poorly localized at laparotomy. Recurrent, or tertiary, peritonitis is a common complication of intraabdominal infection in patients admitted to an ICU. It differs from uncomplicated secondary peritonitis in its microbial flora and lack of response to appropriate surgical and antibiotic therapy. Like nosocomial pneumonia in the critically ill patient, the syndrome appears to be more a reflection than a cause of adverse outcome.**

Peritonitis encompasses a spectrum of disease processes with differing causes and clinical courses [1]. *Primary peritonitis* occurs in young girls or cirrhotics [2]. In the former, infecting organisms are believed to arise from the genital tract, whereas in the latter the infecting species translocate from the gut, perhaps as a consequence of proximal gut microbial overgrowth. The flora of primary peritonitis is typically monomicrobial [3, 4]. *Secondary peritonitis* is more common and is the result of an anatomic breach of the gastrointestinal tract [5, 6]. The microbial flora is that of the adjacent gut; therefore these infections are characteristically polymicrobial. Infection of the peritoneal cavity may also arise secondary to infection of an indwelling dialysis catheter or ventriculoperitoneal shunt or by direct spread from a retroperitoneal focus, as may occur with infected peripancreatic necrosis. Whereas antibiotics are the mainstay of therapy of primary peritonitis, source control in the form of surgical or percutaneous drainage or removal of a colonized device is needed to resolve secondary peritonitis [7]. These measures, combined with adequate physiologic support, result in cure of the infectious process in most patients [8].

It has been recognized that appropriate surgical and antimicrobial therapy does not result in full resolution of all cases of peritonitis, particularly in the most gravely ill patients [9]. Rather, a clinical syndrome evolves characterized by organ dysfunction and prolonged systemic inflammation in association with recurrent peritoneal infection with organisms of low intrinsic pathogenicity. Mortality is high and therapy disappointingly ineffectual. This syndrome has been termed *tertiary peritonitis* [10] and may be defined as the persistence or recurrence of intraabdominal infection after apparently adequate therapy for primary or secondary peritonitis.

Tertiary peritonitis is a commonly encountered but poorly defined entity. This retrospective study was undertaken to characterize the clinical course and microbiology of tertiary peritonitis in critically ill patients with intraabdominal infection.

## Patients and Methods

We studied all patients admitted to the surgical intensive care unit (ICU) of the Toronto Hospital, General Division, between 1988 and 1992 who had undergone a laparotomy for intraabdominal infection prior to or during their ICU admission. Cases of pancreatitis were included when positive cultures were obtained from peripancreatic tissues. Patients whose ICU length of stay or survival was less than 24 hours were excluded.

Patients were considered to have uncomplicated secondary peritonitis if clinical and bacteriologic resolution of infection followed a single operative or percutaneous intervention. Tertiary peritonitis was defined as culture-proved intraabdominal infection persisting or recurring at least 48 hours after apparently adequate treatment of secondary bacterial peritonitis.

In most cases preoperative imaging by ultrasonography or computed tomography (CT) was performed prior to laparotomy, and if a well defined collection was visualized percutaneous drainage was attempted. The number of interventions required to control intraabdominal infection was recorded; for patients developing postoperative peritonitis following an elective procedure, the surgical intervention required for control of peritoneal contamination was considered to be the first laparotomy. The term

*Correspondence to:* J.C. Marshall, M.D.

**Table 1.** Demographic data: secondary versus tertiary peritonitis.*

| Parameter | Secondary peritonitis ($n$ = 15) | Tertiary peritonitis ($n$ = 44) |
|---|---|---|
| Age (years)[a] | 58 ± 14 | 60 ± 16 |
| Sex (male) (%) | 61 | 80 |
| Transfer from another hospital (%) | 47 | 68 |
| APACHE II score[a] | 20.5 ± 5 | 21.5 ± 8 |
| Acute physiology score | 15.6 ± 4.7 | 17.6 ± 6.8 |
| Chronic health evaluation | 4.9 ± 2.9 | 4.3 ± 2.4 |
| Type of immunocompromise (%) | | |
| None | 60.0 | 66.0 |
| Chronic renal failure | 20.0 | 4.5 |
| Diabetes mellitus | 33.0 | 14.0 |
| Corticosteroids | 0 | 4.5 |
| CSA/ALG or azathioprine | 17.0 | 16.0 |

CSA: cyclosporin A; ALG: anti-lymphocyte globulin.
[a]Data are presented as mean ± SD.
*There were no significant differences between the two groups for any parameter.

**Table 2.** Diagnoses at admission to surgical ICU: secondary versus tertiary peritonitis.

| Cause of peritonitis | No. of patients: Secondary peritonitis ($n$ = 15) | No. of patients: Tertiary peritonitis ($n$ = 44) |
|---|---|---|
| Postoperative peritonitis | 7 (47)[a] | 13 (30) |
| Perforated ulcer | 2 (13) | 7 (16) |
| Bowel herniation and perforation | 2 (13) | 0 |
| Pancreatitis | 1 (7) | 12 (27) |
| Necrotic bowel | 1 (7) | 9 (20) |
| Appendicitis | 1 (7) | 1 (2) |
| Diverticulitis | 1 (7) | 2 (5) |

[a]Numbers in parentheses indicate percent.

**Table 3.** Outcome: secondary versus tertiary peritonitis.

| Outcome variable | Secondary peritonitis | Tertiary peritonitis | $p$ |
|---|---|---|---|
| MOD score (mean ± SD) | 7.7 ± 3.3 | 13.3 ± 5.1 | 0.0002 |
| Length of ICU admission (mean ± SD) | 8.5 ± 7.9 | 21.8 ± 14.9 | 0.002 |
| ICU mortality (%) | 13.3 | 56.8 | 0.006 |
| Overall mortality, total (%) | 33.3 | 63.6 | 0.04 |

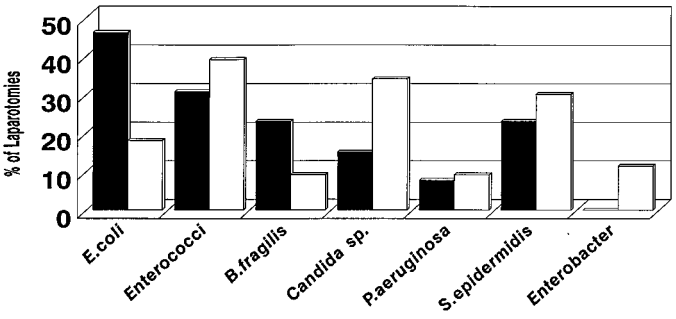


**Fig. 1.** Peritoneal cultures: secondary (closed bars) vs. tertiary (open bars) peritonitis. The most common microbial isolates from patients with secondary peritonitis were *Escherichia coli*, *Enterococcus*, and *Bacteroides fragilis*. In contrast, the predominant organisms isolated at laparotomy in those with tertiary peritonitis were *Enterococcus*, *Candida*, *Staphylococcus epidermidis*, and *Enterobacter.*

"scheduled laparotomy" includes both planned reexploration in the operating room and laparostomy in the ICU.

Only culture data obtained at laparotomy or after insertion of a fresh percutaneous drain were recorded; culture results from previously placed drains, drain tips, and wound swabs were excluded to minimize the likelihood that results reflected surface colonization or contamination of a foreign body. All antibiotics initiated within 2 weeks of laparotomy were recorded, regardless of whether they were administered specifically for treatment of intraperitoneal infection.

APACHE II scores were calculated using the most abnormal values within the first 24 hours of admission to the ICU [11]. The severity of organ dysfunction over the ICU stay was quantitated by the Multiple Organ Dysfunction Score (MOD score), reflecting graded physiologic dysfunction in six organ systems [12]. ICU mortality and hospital mortality were recorded.

Comparison of categorical variables was performed using $\chi^2$ analysis with Yates' continuity correction or a two-tailed Fisher exact test. Continuous variables were compared by two-tailed unpaired Student's $t$-test. An $\alpha$ level of less than 0.05 was considered significant. All results are presented as mean ± standard deviation (SD).

## Results

### *Demographic Data and Clinical Outcomes*

Tertiary peritonitis developed in 44 of the 59 patients with intraabdominal infection (74%). Admission demographic characteristics of patients developing tertiary peritonitis did not differ from those of patients with uncomplicated secondary peritonitis (Table 1). The causes of intraabdominal infection are summarized in Table 2. There were no significant differences in admission diagnoses between patients with uncomplicated secondary peritonitis and those developing tertiary peritonitis.

Despite comparable baseline characteristics, patients developing tertiary peritonitis had significantly worse outcomes (Table 3). Organ dysfunction was significantly greater and the average ICU stay longer. The mortality associated with tertiary peritonitis was almost twice that seen with uncomplicated intraabdominal infection; most of these deaths (25/28) occurred in the ICU.

### *Microbiology*

The organisms isolated from patients with uncomplicated secondary peritonitis and those with tertiary peritonitis are summarized in Figure 1. The most common isolates from patients with secondary peritonitis were *Escherichia coli*, *Enterococcus*, and *Bacteroides fragilis*. In contrast, the predominant organisms isolated from patients with tertiary peritonitis were *Enterococcus*, *Candida* spp., and *Staphylococcus epidermidis*. Cultures from patients with secondary peritonitis and those eventually developing tertiary peritonitis were similar at the time of the first intervention (Table 4). The microbiologic shift occurred over the course of the ICU stay; as shown in Figure 2, between the first and last intervention the percentage of specimens yielding *E. coli*, *B.*

**Table 4.** Microbial isolates from first intervention.

| Organism | Percent of patients with isolate | |
|---|---|---|
| | Secondary peritonitis ($n$ = 15) | Tertiary peritonitis ($n$ = 44) |
| *Escherichia coli* | 33 | 37 |
| *Enterococcus* | 29 | 28 |
| *Bacteroides fragilis* | 22 | 10 |
| *Staphylococcus epidermidis* | 22 | 28 |
| *Candida* | 22 | 20 |
| No organism | 6 | 15 |

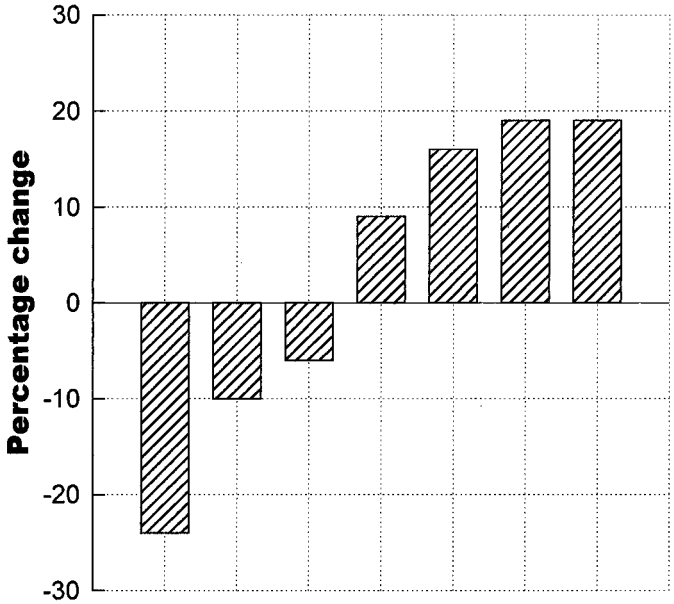


**Fig. 2.** Organisms isolated at the first laparotomy in patients progressing to tertiary peritonitis were similar to those seen in patients with secondary peritonitis. By the time of the final laparotomy, a shift had occurred to the characteristic flora of tertiary peritonitis. Isolates of *E. coli* (bar 1), *B. fragilis* (bar 2), and *Pseudomonas* (bar 3) diminished in frequency, whereas *Enterobacter* (bar 4), *Enterococcus* (bar 5), *Candida* (bar 6), and *S. epidermidis* (bar 7) became more common. The graph denotes the absolute percentage change in frequency of isolation between the first and last surgical intervention.

*fragilis*, and *Pseudomonas* decreased, whereas the percentage of isolates of *Candida*, coagulase-negative staphylococci, enterococci, and *Enterobacter* increased. MOD scores tended to be higher in patients infected with these organisms, although the difference achieved statistical significance only for *Enterococcus* (Table 5).

### *Antimicrobial Therapy*

It was not possible in a retrospective review to determine which antimicrobial agents were administered specifically for the treatment of intraabdominal infection. However, the average number of antibiotics administered to patients with secondary or tertiary peritonitis was comparable (4.3 ± 1.5 vs. 5.2 ± 2.3, respectively), and the use of antibiotics in patients with tertiary peritonitis was similar when survivors were compared to nonsurvivors (survivors 4.9 ± 1.5 antibiotics vs. nonsurvivors 5.3 ± 2.7 antibiotics).

**Table 5.** Degree of organ dysfunction in relation to peritoneal isolates.

| Organism | MOD score (mean ± SD) | |
|---|---|---|
| | Present | Absent |
| *Enterococcus* | 15.4 ± 4.0* | 12.0 ± 5.4 |
| *Enterobacter* | 15.2 ± 4.1 | 12.9 ± 5.3 |
| *Candida* spp. | 14.2 ± 4.7 | 12.9 ± 5.3 |
| *Staphylococcus epidermidis* | 13.7 ± 4.8 | 12.6 ± 5.7 |

*$p < 0.05$ vs. *Enterococcus* absent.

**Table 6.** Management of tertiary peritonitis: survivors versus nonsurvivors.

| Parameter | Survivors | Nonsurvivors |
|---|---|---|
| Drainage (mean ± SD) | | |
| Open | 2.8 ± 1.5 | 3.9 ± 3.4* |
| Percutaneous | 1.2 ± 0.8 | 0.3 ± 0.6* |
| Regimen (%) | | |
| On demand | 81.3 | 64.3 |
| Scheduled relaparotomy | 18.8 | 28.6 |

*$p < 0.05$ vs. survivors.

The use of appropriate antimicrobial therapy could not be shown to alter the prognosis for patients with tertiary peritonitis. Of 21 patients with *Candida* isolated at laparotomy, only 4 were treated with amphotericin B for at least 5 days. Only 1 of these 4 patients (25%) survived compared to 5 of 17 (29%) patients not receiving antifungal therapy. *Enterococcus* was isolated at laparotomy in 29 patients, 13 of whom were treated with a combination of ampicillin and an aminoglycoside or vancomycin for at least 5 days. Survival was 38% (5/13), which did not differ from that of patients who did not receive appropriate therapy (31%, 5/16 patients). *S. epidermidis* was isolated at laparotomy in 22 patients, only 2 of whom received at least 5 days of vancomycin therapy. Similarly, bacteremia was documented only rarely in patients with tertiary peritonitis. Fungemia occurred in only 2 of 21 patients, and enterococcal bacteremia did not occur in any of the patients with this organism isolated at laparotomy. *S. epidermidis* bacteremia occurred in 2 of 19 patients demonstrating this organism at laparotomy without concurrent positive central venous or arterial line cultures. On the other hand, *Enterobacter* bacteremia occurred in 44% (4/9) of patients in whom this organism was isolated at laparotomy.

### *Surgical Management*

The surgical management of survivors and nonsurvivors with tertiary peritonitis is summarized in Table 6. Nonsurvivors with tertiary peritonitis were less likely to have collections amenable to percutaneous drainage than survivors, and their deaths occurred despite their having undergone more open laparotomies. Moreover, the use of on-demand or scheduled relaparotomy had no obvious influence on survival. The predominant finding noted at laparotomy in patients with tertiary peritonitis was the presence of poorly localized collections of fluid, rather than discrete abscesses.

### *Outcomes*

Five of the fifteen patients with secondary peritonitis died in hospital (33.3%); two of these deaths occurred in the ICU (Table

3). Only one patient had evidence of ongoing infection at the time of death: a 58-year-old man with diabetes and chronic renal failure who was discharged from the ICU 48 hours after resection of a segment of necrotic small bowel. Following discharge from the ICU the patient had repeated episodes of gram-positive bacteremia, presumably from a central venous line, and died 6 weeks after laparotomy. One other patient died of aspiration with evidence of pneumonia and a recent myocardial infarction at autopsy. All other deaths of these patients were related to cardiac causes.

Patients with tertiary peritonitis had significantly higher hospital mortality (28/44, 63.6%) (Table 3); all but three of these deaths occurred in the ICU. Eight patients underwent autopsy. Discrete abscesses were found in three patients; another two patients had positive peritoneal cultures, and one patient each had necrotic small bowel and peripancreatic necrosis that demonstrated *Candida* histologically. Only one patient had no evidence of ongoing intraabdominal infection at the time of death.

## Discussion

Prompt surgical intervention and adjunctive antibiotic therapy usually comprise effective therapy for patients with secondary peritonitis. However, a few patients develop a clinical syndrome characterized by poorly localized intraabdominal infection, an altered microbial flora, progressive organ dysfunction, and significantly higher mortality; this syndrome has been termed tertiary peritonitis [10].

Although tertiary peritonitis is a clinically distinct entity on the basis of its altered flora and highly morbid course, we were unable to identify factors that could predict which patients with secondary peritonitis would develop the syndrome. Premorbid factors usually believed to result in impaired resolution of intraabdominal infection, including advanced age [13], chronic renal failure, diabetes, or the use of corticosteroids and other immunosuppressive agents [14, 15], were not associated with the development of tertiary peritonitis. Similarly, the initial disease process responsible for peritoneal contamination was not a risk factor for the subsequent development of tertiary peritonitis. Delayed treatment of secondary peritonitis also did not appear to be a risk factor, as postoperative peritonitis, classically associated with delayed intervention, was documented with equal frequency in both groups. Finally, the degree of physiologic derangement at the time of ICU admission, as measured by APACHE II scores, did not predict the subsequent development of tertiary peritonitis, although outcomes measured by the MOD score and mortality were strikingly different. The APACHE II score has been shown to have limitations in this patient population [16]. In patients with peritonitis, resuscitation frequently occurs in places other than the ICU, including the operating room, emergency room, and transferring institution. As a result, the patient may be relatively stable at the time of ICU admission, and the APACHE II score may not reflect the initial magnitude of physiologic derangement.

Cultures from patients with secondary bacterial peritonitis are usually polymicrobial, but a predictable flora predominates including aerobic gram-negative organisms such as *E. coli*, anerobes such as *B. fragilis* and *Clostridium*, and *Enterococcus* [17]. In contrast, the relatively nonpathogenic organisms—*Enterococcus*, *Candida* spp. and *S. epidermidis*—are the most common isolates from episodes of tertiary peritonitis [9, 18, 19]. The rare documentation of *Candida*, *Enterococcus*, or *S. epidermidis* in peripheral blood cultures attests to the low intrinsic virulence of the organisms producing tertiary peritonitis. An exception in this series was *Enterobacter* spp.; almost half of all patients with positive peritoneal cultures with this organism had concomitant bacteremia.

The presence of organisms of low pathogenicity may have several possible explanations. The microbial flora in patients developing tertiary peritonitis was similar to that of patients with secondary peritonitis at the time of the first laparotomy, implying that the characteristic microbial flora emerges as the syndrome evolves. This shift may reflect antibiotic pressures, as these organisms are resistant to most first-line antibiotics used in the surgical ICU. Alternatively, the emergence of these nonvirulent organisms may reflect increasing degrees of immune dysfunction coincident with global deterioration of organ function [18]. Additional supportive evidence for the latter hypothesis is the greater prevalence of these isolates in patients who ultimately die with peritonitis.

Several authors have considered the association between these organisms, prolonged surgical ICU admission, broad-spectrum antibiotic therapy, organ failure, and death [19]. Solomkin et al. [20] studied the significance of *Candida* isolated from the peritoneal cavity in a similar series of patients. Mortality for patients with *Candida* was as high as 70%; but it was significantly reduced in patients who received antifungal therapy prior to the development of organ dysfunction or candidemia. Similarly, Calandra et al. [21] documented a mortality of 63% in patients with *Candida* isolates from the peritoneal cavity and recommended treatment with antifungal therapy in the presence of unremitting intraabdominal infection. In the present study there was no evidence that amphotericin B treatment offered any survival benefit.

The role of *Enterococcus* in intraabdominal infection is controversial. It is generally accepted that no specific antienterococcal coverage is required for patients with uncomplicated secondary peritonitis [22]. Persistent isolation of enterococci in a compromised or critically ill patient is an indication for specific antimicrobial therapy [23]. The ability of broad-spectrum antibiotic therapy to select out the *Enterococcus* may reflect an inability of endogenous host defenses to contend effectively with certain pathogens [24], and the presence of enterococci may be considered a marker of host defense failure [25]. Specific antienterococcal therapy did not confer a survival benefit, suggesting that it may be more a marker of advanced disease than a pathogen contributing to disease progression.

*Staphylococcus epidermidis*, like *Enterococcus*, *Candida*, and *Enterobacter* [26], is selected out by broad-spectrum antibiotic therapy [27]. The role of *S. epidermidis* in intraabdominal infection has not yet been defined, but its presence in critically ill patients with peritonitis is well documented [9, 19, 28]. Whether specific antimicrobial therapy alters outcome is unknown.

Tertiary peritonitis is characterized by persistent infection with a characteristic flora in association with evolving organ dysfunction. Although risk factors for its development remain elusive, three explanations for the syndrome appear plausible.

First, the syndrome of tertiary peritonitis may reflect inadequate therapy of primary or secondary peritonitis, with persistent undrained foci of infection that become colonized under antibiotic pressures with the typical antibiotic-resistant strains. During the era prior to the wide availability of ultrasonography and CT

scanning, inadequately treated intraabdominal infection was a leading cause of the multiple organ dysfunction syndrome [29, 30]; conversely, organ dysfunction was considered an indication for blind laparotomy [31]. In our series, seven of eight patients undergoing autopsy had evidence of untreated intraabdominal infection, lending support to the concept that tertiary peritonitis is associated with inadequate surgical source control. On the other hand, we could not show any obvious association of the development of tertiary peritonitis with the mode of surgical therapy, and others have reported that organ failure may not be reversed even when adequate surgical control is accomplished [18, 32]. Moreover, reoperation is associated with an exaggerated host inflammatory response [33] without obvious benefit to the clinical course of the disease [34].

An alternate explanation is that infection of the peritoneal cavity arises secondary to dissemination of ICU-acquired infections at other sites. The flora of tertiary peritonitis is identical to that predominating in nosocomial ICU-acquired infection [18, 35]. Moreover, although we did not commonly demonstrate bacteremia in association with episodes of tertiary peritonitis, all of the predominant organisms—coagulase-negative staphylococci [36, 37], *Candida* [38, 39], enterococci [25, 40], and *Enterobacter* [26]—are common causes of bacteremia in the ICU. Injury to the peritoneum from primary or secondary peritonitis could predispose to such metastatic spread. A clear-cut history of prior nosocomial infection was not apparent in our population, and disseminated, multifocal infection is the exception, rather than the rule, with these species.

Finally, and perhaps most convincingly, the superinfection of tertiary peritonitis may arise as a result of the translocation of the infecting species from the adjacent gastrointestinal tract. The characteristic flora of tertiary peritonitis includes the same organisms that have been shown to overgrow the proximal gastrointestinal tract of the critically ill patient [28, 41], and there is a strong correlation between gut colonization and the development of peritoneal infection with the same species [28].

After appropriate surgical management, the combination of intact host defenses and appropriate antimicrobial therapy results in complete resolution of most cases of secondary peritonitis. Tertiary peritonitis develops when the interaction of therapeutic intervention and host defenses fails [42]. It is not clear that the failure of infection control per se leads to an adverse outcome. Rather, tertiary peritonitis can be viewed as other ICU-acquired infections, such as pneumonia, in which the role for antimicrobial (or surgical) therapy in improving outcome is modest [43] and the specific impact of infection on mortality is uncertain [44–47]. It is entirely plausible that patients are dying *with*, rather than *of*, tertiary peritonitis; persistent infection may simply be a manifestation of end-stage organ dysfunction.

**Résumé**

Objectifs: Définir les facteurs de risque et l'évolution clinique de la péritonite récidivente ou tertiaire. Fond de problème: Les soins intensifs des patients ayant une infection intra-abdominale menaçant le pronostic vital est responsable d'un nouveau syndrome clinique, la péritonite tertiaire, définie comme la persistance ou la récidive de l'infection intra-abdominale après la traitement apparemment complet d'une péritonite primitive ou secondaire. Méthodes: Nous avons entrepris une étude rétrospective de 59 patients admis pour infection intra-péritonéale dans une unité de soins intensifs chirurgicale (SIC). Résultats: On a vu se développer une péritonite tertiaire dans 74 pourcent (44/59) des patients. Malgré le fait que l'état général des patients, la source de péritonite et le score Apache II à l'admission étaient similaires, les patients ayant une péritonite tertiaire sont restés plus longtemps en SIC (21.8 ± 14.9 vs 8.5 ± 7.9 jours), avaient un score de défaillance organique plus élevé (13.3 ± 5.1 vs 7.7 ± 3.3 jours) et une mortalité SIC plus élevée (64% vs 33%) par rapport aux patients ayant une péritonite secondaire non compliquée. Les organismes les plus souvent rancontrés dans le péritonite tertiaire étaient l'Enterococci, le Candida, le S epidermidis, et l'Enterobacter. Les foyers d'infection étaient rarement accessibles au drainage percutané et n'étaient pas bien localisés lors de la laparotomie. Conclusions: La péritonite récidivante, ou tertiaire, est une complication fréquente de l'infection intra-abdominale chez le patient admis en SIC. Elle diffère de la péritonite secondaire non-compliquée de par se flore microbienne et l'absence de réponse à la thérapeutique chirurgicale et antibiotique suffisante. Tout comme l'infection pulmonaire nosocomiale de ces patients sévèrement atteints, ce syndrome apparait comme le témoin plutôt que la cause, d'une évolution fâcheuse.

**Resumen**

Objetivo: Definir los factores de riesgo y la evolución clínica de la peritonitis recurrente o terciaria. Antecedentes: El cuidado intensivo y el soporte de pacientes con infecciones intraabdominales ha llevado a la emergencia de un nuevo síndrome clínico, la peritonitis terciaria, la cual se define como la persistencia o la recurrencia de infección intraabdominal luego de tratamiento aparentemente adecuado de una peritonitis primaria o secundaria. Métodos: Estudio retrospectivo sobre 59 pacientes con infección intraabdominal en una unidad de cuidado intensivo quirúrgico (UCI). Resultados: 74% (44/59) de los pacientes desarrollaron peritonitis terciaria. A pesar de poseer comparable estado de salud premórbido, origen de la peritonitis y puntaje APACHE en el momento de la admisión, los pacientes con peritonitis terciaria exhibieron una estadía significativamente más prolongada en la UCI (21.8 ± 14.9 vs. 8.5 ± 7.9 días), índices de disfunción orgánica más altos (13.3 ± 5.1 vs. 7.7 ± 3.3) y mayor mortalidad (64% vs. 33%) que los pacientes con peritonitis secundaria no complicada. Los microorganismos más frecuentemente aislados en los casos de peritonitis terciaria fueron Enterococci, Candida, S. epidermidis y Enterobacter. Los focos sépticos rara vez fueron susceptibles de drenaje percutáneo y se los halló de difícil localización en la laparotomía. Conclusiones: La peritonitis recurrente o terciaria, es una complicación común de la infección intraabdominal en pacientes que ingresan a una UCI. Difiere de la peritonitis secundaria no complicada en cuanto a la flora microbiana y a la no respuesta al tratamiento quirúrgico ni a la terapia antibiótica adecuados. Al igual que una neumonía nosocomial en el paciente en estado crítico, el síndrome parece ser un reflejo, más que la causa, de un resultado final desfavorable.

**References**

1. Johnson, C.C., Baldessarre, J., Levison, M.E.: Peritonitis: update on pathophysiology, clinical manifestations, and management. Clin. Infect. Dis. *24*:1035, 1997

2. Crossley, I.R., Williams, R.: Spontaneous bacterial peritonitis. Gut *26*:325, 1985
3. Conn, H.O.: Spontaneous peritonitis and bacteremia in Laennec's cirrhosis caused by enteric organisms. Ann. Intern. Med. *60*:568, 1964
4. Correia, J.P., Conn, H.O.: Spontaneous bacterial peritonitis in cirrhosis: endemic or epidemic? Med. Clin. North Am. *59*:963, 1975
5. Bohnen, J., Boulanger, M., Meakins, J.L., Mclean, A.P.H.: Prognosis in generalized peritonitis: relation to cause and risk factors. Arch. Surg. *118*:285, 1983
6. Pine, R.W., Wertz, M.J., Lennard, E.S., Dellinger, E.P., Carrico, C.J., Minshew, B.H.: Determinants of organ malfunction or death in patients with intra-abdominal sepsis: a discriminant analysis. Arch. Surg. *118*:242, 1983
7. Schein, M., Hirshberg, A., Hashmonai, M.: Current surgical management of severe intraabdominal infection. Surgery *112*:489, 1992
8. Nathens, A.B., Rotstein, O.D.: Therapeutic options in peritonitis. Surg. Clin. North Am. *74*:677, 1997
9. Rotstein, O.D., Pruett, T.L., Simmons, R.L.: Microbiologic features and treatment of persistent peritonitis in patients in the intensive care unit. Can. J. Surg. *29*:247, 1986
10. Rotstein, O.D., Meakins, J.L.: Diagnostic and therapeutic challenges of intraabdominal infections. World J. Surg. *14*:159, 1990
11. Knaus, W.A., Draper, E.A., Wagner, D.P., Zimmerman, J.E.: APACHE II: a severity of disease classification system. Crit. Care Med. *13*:818, 1985
12. Marshall, J.C., Cook, D.J., Christou, N.V., Bernard, G.R., Sprung, C.L., Sibbald, W.J.: Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit. Care Med. *23*:1638, 1995
13. Dellinger, E.P., Wertz, M.J., Meakins, J.L., Solomkin, J.S., Allo, M.D., Howard, R.J., Simmons, R.L.: Surgical infection stratification system for intra-abdominal infection: multicenter trial. Arch. Surg. *120*:21, 1985
14. Bohnen, J.M.A., Mustard, R.A., Oxholm, S.E., Schouten, D.: APACHE II score and the outcome of abdominal infection. Arch. Surg. *129*:33, 1994
15. Bohnen, J.M.A., Mustard, R.A., Oxholm, S.E., Schouten, D.: APACHE II score and abdominal sepsis. Arch. Surg. *123*:225, 1988
16. Cerra, F.B., Negro, F., Abrams, J.: APACHE II score does not predict multiple organ failure or mortality in postoperative surgical patients. Arch. Surg. *125*:519, 1990
17. Lorber, B., Swenson, R.M.: The bacteriology of intra-abdominal infections. Surg. Clin. North Am. *55*:1349, 1975
18. Marshall, J.C., Christou, N.V., Horn, R., Meakins, J.L.: The microbiology of multiple organ failure: the proximal GI tract as an occult reservoir of pathogens. Arch. Surg. *123*:309, 1988
19. Sawyer, R.G., Rosenlof, L.K., Adams, R.B., May, A.K., Spengler, M.D., Pruett, T.L.: Peritonitis into the 1990's: changing pathogens and changing strategies in the critically ill. Am. Surg. *58*:82, 1992
20. Solomkin, J.S., Flohr, A.B., Quie, P.G., Simmons, R.L.: The role of Candida in intraperitoneal infections. Surgery *88*:524, 1980
21. Calandra, T., Bille, J., Schneider, R., Mosimann, F., Francioli, P.: Clinical significance of Candida isolated from peritoneum in surgical patients. Lancet *2*:1437, 1989
22. Bohnen, J.M.A., Solomkin, J.S., Dellinger, E.P., Bjornson, H.S., Page, C.P.: Guidelines for clinical care: anti-infective agents for intra-abdominal infection: a Surgical Infection Society policy statement. Arch. Surg. *127*:83, 1992
23. Barie, P.S., Christou, N.V., Dellinger, E.P., Rout, W.R., Stone, H.H., Waymack, J.P.: Pathogenicity of the Enterococcus in surgical infections. Ann. Surg. *212*:155, 1990
24. Dougherty, S.H.: Role of Enterococcus in intraabdominal sepsis. Am. J. Surg. *148*:308, 1984
25. Garrison, R.N., Fry, D.E., Berberich, S., Polk, H.C.: Enterococcal bacteremia: clinical implications and determinants of death. Ann. Surg. *196*:43, 1982
26. Burchard, K.W., Barrall, D.T., Reed, M., Slotman, G.J.: Enterobacter bacteremia in surgical patients. Surgery *100*:857, 1986
27. Christensen, G.D., Bisno, A.L., Parisi, J.T., McLaughlin, B., Hester, M.G., Luther, R.W.: Nosocomial septicemia due to multiply antibiotic resistant Staphylococcus epidermidis. Ann. Intern. Med. *96*:1, 1982
28. Marshall, J.C., Christou, N.V., Meakins, J.L.: The gastrointestinal tract: the "undrained abscess" of multiple organ failure. Ann. Surg. *218*:111, 1993
29. Fry, D.E., Pearlstein, L., Fulton, R.L., Polk, H.C.: Multiple system organ failure: the role of uncontrolled infection. Arch. Surg. *115*:136, 1980
30. Polk, H.C., Shields, C.L.: Remote organ failure: a valid sign of occult intraabdominal infection. Surgery *81*:310, 1977
31. Ferraris, V.A.: Exploratory laparotomy for potential abdominal sepsis in patients with multiple organ failure. Arch. Surg. *118*:1130, 1983
32. Norton, L.W.: Does drainage of intraabdominal pus reverse multiple organ failure? Am. J. Surg. *149*:347, 1985
33. Sautner, T., Gotzinger, P., Redl-Wenzl, E.M., Dittrich, K., Felfernig, M., Sporn, P., Roth, E., Fugger, R.: Does reoperation for abdominal sepsis enhance the inflammatory host response? Arch. Surg. *132*:250, 1997
34. Van Goor, H., Hulsebos, R.G., Bleichrodt, R.P.: Complications of planned relaparotomy in patients with severe general peritonitis. Eur. J. Surg. *163*:61, 1997
35. Nathens, A.B., Chu, P.T.Y., Marshall, J.C.: Nosocomial infection in the surgical intensive care unit. Infect. Dis. Clin. North Am. *6*:657, 1992
36. Forse, R.A., Dixon, C., Bernard, K., Martinez, L., Mclean, A.P.H., Meakins, J.L.: Staphylococcus epidermidis: an important pathogen. Surgery *85*:507, 1979
37. Martin, M.A., Pfaller, M.A., Wenzel, R.P.: Coagulase-negative staphylococcal bacteremia: mortality and hospital stay. Ann. Intern. Med. *110*:9, 1989
38. Dyess, D.L., Garrison, R.N., Fry, D.E.: Candida sepsis: implications of polymicrobial blood borne infection. Arch. Surg. *120*:345, 1985
39. Wey, S.B., Mori, M., Pfaller, M.A., Woolson, R.F., Wenzel, R.P.: Risk factors for hospital acquired Candidemia: a matched case control study. Arch. Intern. Med. *149*:2349, 1989
40. Barrall, D.T., Kenney, P.R., Slotman, G.J., Burchard, K.W.: Enterococcal bacteremia in surgical patients. Arch. Surg. *120*:57, 1985
41. Garvey, B.M., McCambley, J.A., Tuxen, D.V.: Effects of gastric alkalization on bacterial colonization in critically ill patients. Crit. Care Med. *17*:211, 1989
42. Reemst, P.H., van Goor, H., Goris, R.J.: SIRS, MODS, and tertiary peritonitis. Eur. J. Surg. *576*(Suppl.):47, 1997
43. Rello, J., Ausina, V., Ricart, M., Castella, J., Prats, G.: Impact of previous antimicrobial therapy on the etiology and outcome of ventilator associated pneumonia. Chest *104*:1230, 1993
44. Rello, J., Quintana, E., Ausina, V., Castella, J., Luquin, M., Net, A., Prats, G.: Incidence, etiology, and outcome of nosocomial pneumonia in mechanically ventilated patients. Chest *100*:439, 1991
45. Leu, H.S., Kaiser, D.L., Mori, M., Woolson, R.F., Wenzel, R.P.: Hospital-acquired pneumonia: attributable mortality and morbidity. Am. J. Epidemiol. *129*:1258, 1989
46. Poole, G.V., Muakkassa, F.F., Griswold, J.A.: The role of infection in outcome of multiple organ failure. Am. Surg. *59*:727, 1993
47. Marshall, J.C., Sweeney, D.: Microbial infection and the septic response in critical surgical illness: sepsis, not infection, determines outcome. Arch. Surg. *125*:17, 1990