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The Characteristics and Outcomes of Secondary Peritonitis in a Tertiary Hospital, Benghazi, Libya

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Abstract

Background: Despite improvements in treatment, secondary peritonitis is still associated with high morbidity and mortality rates. Better knowledge of real-life clinical practices might improve management.

Objectives: To identify the common causes and highlight the morbidity and mortality of secondary peritonitis in Al-Jala hospital, Benghazi, Libya.

Patients and Methods: Retrospective study (January 2009–August 2010) of 137 patients with secondary peritonitis is reported. **Results:** Appendicitis and gastroduodenal perforations were the commonest causes of secondary peritonitis, occurring in 61% and 20% of the patients

respectively. Other conditions (small bowel perforations, colonic perforations, biliary peritonitis, ruptured hydatid cyst and pancreatitis) accounted for less than 20% of cases. The overall mortality rate was 4.37%. Morbidity developed in 23% of Patients.

Conclusions: Acute appendicitis is the most common cause of intra-abdominal infection in our study. The clinical outcomes associated with secondary peritonitis are highly dependent upon the site of contamination (versus others), as well as local and systemic factors.

Key words: Secondary peritonitis, Acute appendicitis, Intraabdominal sepsis.

Introduction

A variety of factors influenced the reduction in mortality from peritonitis over the last century including safer anesthetic techniques, better fluid management, the advent of blood banking, improvements in critical care, more rapid and accurate diagnostic studies, and more effective antibiotics. Newer challenges have arisen

threatening to offset these advances (1). Patients with intra-abdominal infection are older, have more comorbid conditions, often have associated immune suppression due to chronic diseases or their treatment, and more frequently have decreased physiologic reserve with either sub-clinical or evident organ failure (2). Successful management of intra-abdominal infection is predicated on

the use of appropriate operative measures to address peritoneal infection (2)

Prospective clinical trials have also taught us the importance of "source control". This generally involves drainage of abscesses or infected fluid collections; whether contamination is localized or diffuse are important. The systemic response to infection depends upon immune status as well as innate genetically coded responses to infection (4).

There are limited data on these clinical problems in our region. Better knowledge of real-life clinical practices might improve management. Therefore, we have therefore, aimed to identify the common causes and highlight the morbidity and mortality of secondary peritonitis.

Patients and Methods

A retrospective study describing one hundred and thirty seven patients with secondary peritonitis who were seen between January 2009 and August 2010. They were treated at the Department of General Surgery at Al-Jala Hospital, Benghazi, Libya. Patients' demographic characteristics origin of peritonitis and microbiological data (peritoneal fluid) were recorded. Additionally, diagnostic aids used and clinical outcomes were analyzed.

Results

Demographic characteristics

One hundred and thirty seven patients were diagnosed with secondary peritonitis. Ninety four patients (69%) were males. The age ranged from 15 to 87 years with a mean age of 18 years (Figure 1).

Anatomical locations of perforation

The anatomical locations of perforations are presented in Figure 2. The most common two causes of secondary peritonitis were perforated appendicitis (84 patients) and perforated peptic ulcer (28 patients). Small bowel perforation found in 9 patients. Most small intestinal perforations are due to unrecognized traumatic injuries or intestinal ischemia; but two were typhoid ileal perforations. Less common causes were colonic perforation in 7 patients (5.1%). Peritonitis of biliary origin was recorded in 7 cases; 5 due to perforated gall bladder (3.6%) and 2 due to biliary tract injury. Ruptured

debridement of necrotic or infected tissues; and definitive measures to control the source of contamination and to restore anatomy and function (3). The response to intra-abdominal infection depends upon the complex interaction of a variety of factors. The degree of microbial contamination, the site of origin of contamination and infected hydatid cyst was seen in one patient and pancreatitis in another. Postoperative peritonitis was seen in four patients (3%), two cases were secondary to bile duct injury and two due to anastomotic leakage.

Results of diagnostic investigations

White cell count was more than 11.000 cells in 64.2% of cases. *E. Coli*, anaerobes and *Enterococcus* were the most frequently isolated microorganisms. X-ray of the chest (CXR) was done in 97 patients (70.8%). It was normal in 72 patients (74.2%) and revealed free air under diaphragm in 25 patients (25.7%). An ultrasound scan examination (US) was performed in 81 patients (59.1%); free fluid appears in 45 patients (55.5%). Appendicular disease showed in 19 patients (23.4%) while gallbladder disease was seen in 5 cases (6.2%). Dilated bowel loops were seen in 5 cases (6.2%). US scans revealed no abnormality in seven cases (8.6%). Computed tomography (CT) scans were performed in 3 cases only (2.2%), one revealed appendicular pathology, another showed gall bladder pathology and the third confirmed a recto-sigmoid tumor.

Morbidity and mortality

Complications developed in 32 patients (23.4%). Wound infection were seen in 15 patients. chest infection in 10, residual collection in 3, renal failure in 2 and wound dehiscence in 2 (Figure 3). Six patients died out of 137 patients (mortality rate was 4.4%). Three deaths were due to septicemia and septic shock, 2 deaths due to cardiopulmonary arrest and one patient died due to myocardial infarction.

Discussion

The diagnosis of intra-abdominal infection is usually based on history and physical examination. Many patients will have abdominal computed tomography (CT) scanning to establish the diagnosis. CT is also useful to identify patients with localized abscesses who are candidates for percutaneous drainage rather than operation.

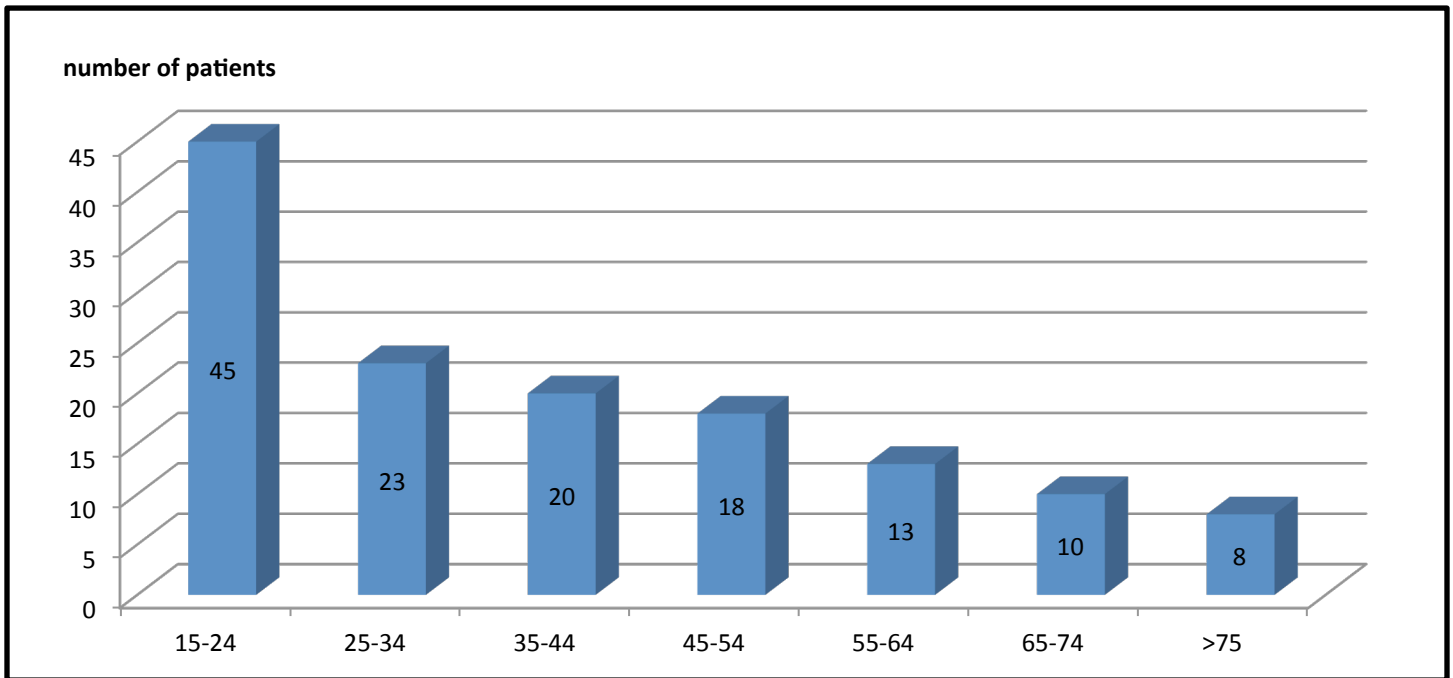


Figure 1. The age distribution of all the 137 patients (years). The preponderance of young adults is clearly evident.

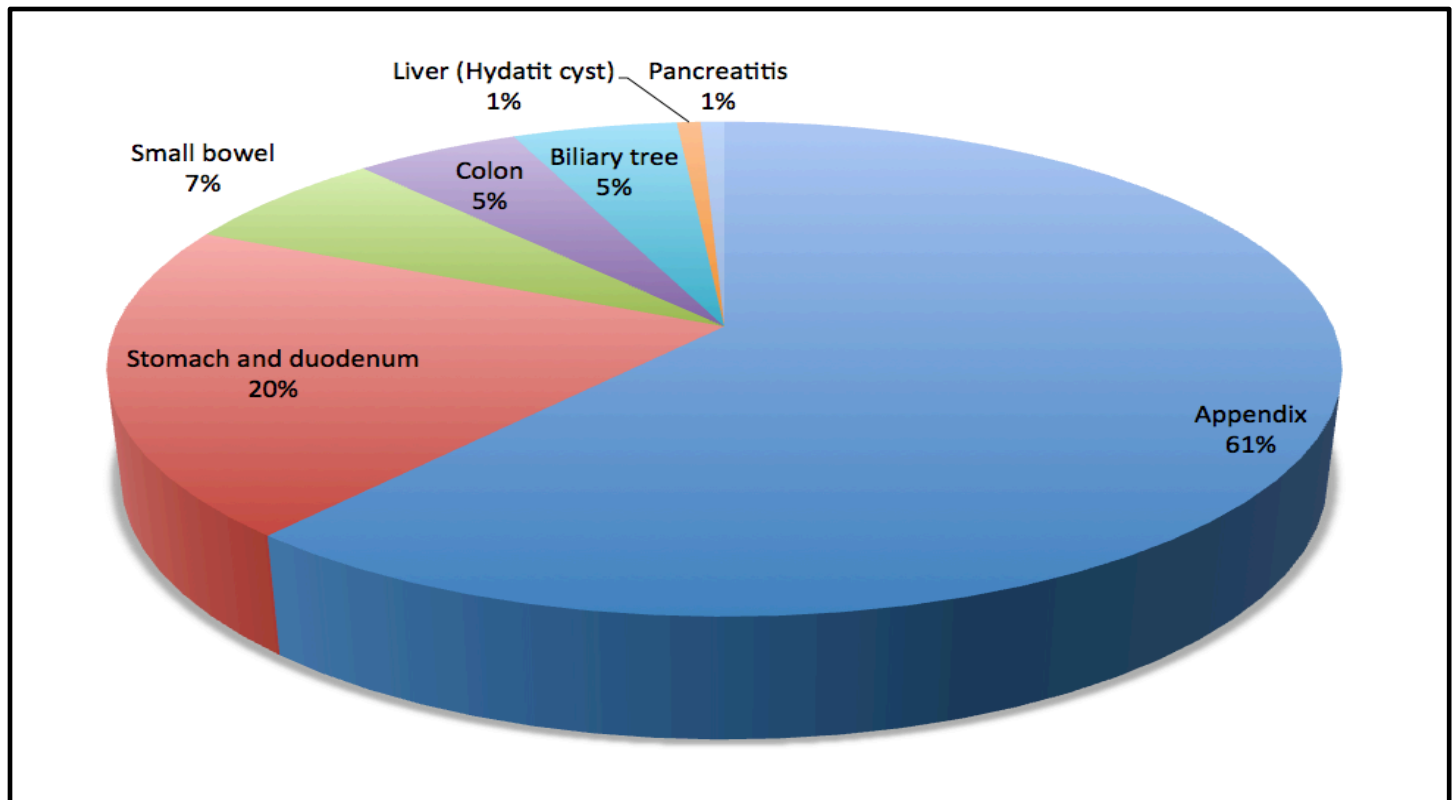


Figure 2. The anatomical locations for the underlying peritoneal perforations.

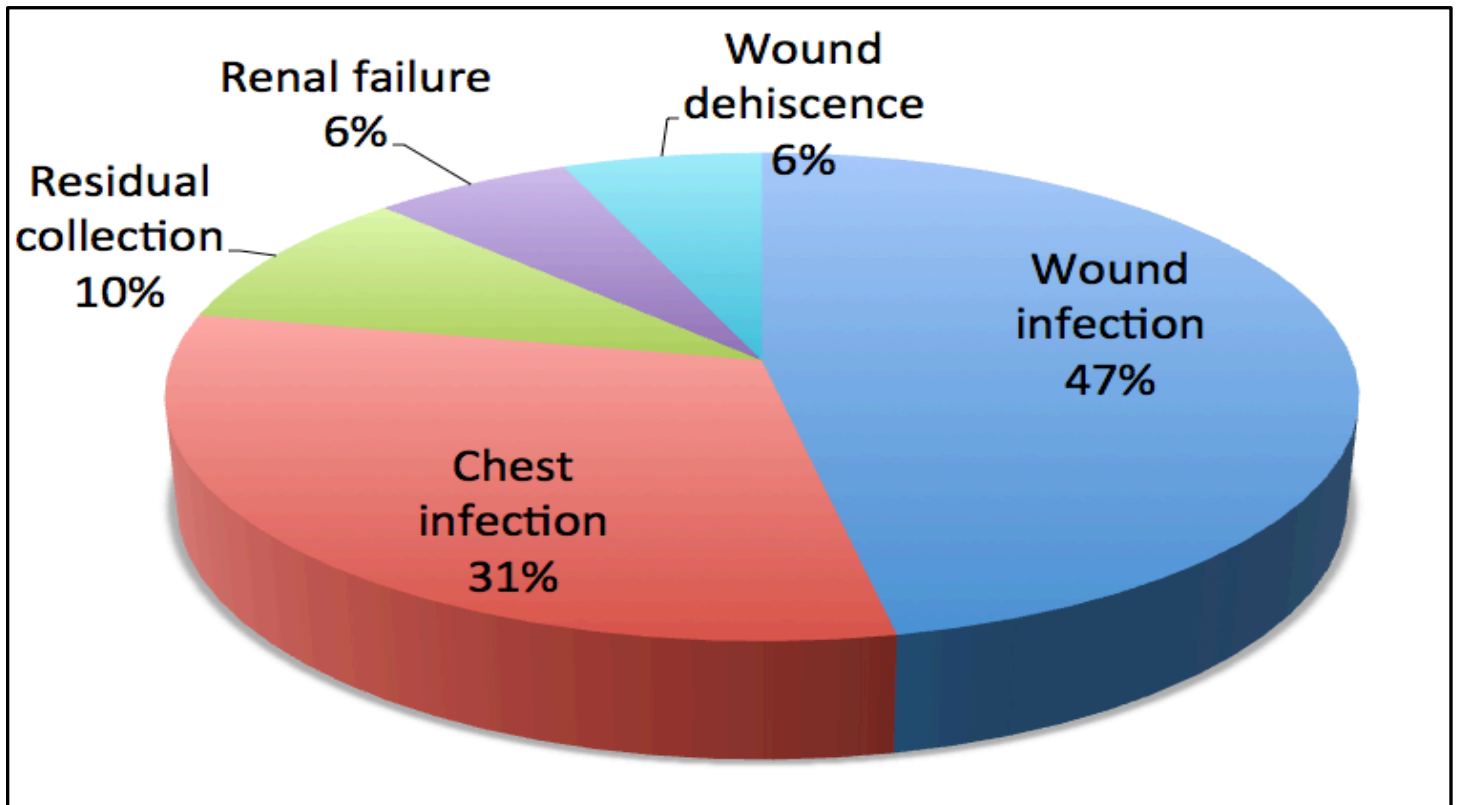


Figure 3. Frequency of complications of secondary peritonitis in 23 out of the 137 patients studied.

The treatment principles of intra-abdominal infections include: 1) restoration of fluid and electrolyte imbalances; 2) administration of appropriate empiric antimicrobial therapy; 3) control of the source of infection; and 4) physiologic support of organ systems. Failure to address any of these important areas can lead to increased mortality, an increased incidence of organ failure, and prolonged hospital stay (4)

In a prospective study from Ghana, appendicitis and typhoid ileal perforation were the commonest causes, occurring in 43.1% and 35.1% of patients, respectively (5). Other conditions such as gastroduodenal perforations, ruptured abscesses, traumatic bowel perforations and amoebic colonic perforations together accounted for fewer than quarter of the cases (5). In another study of 480 cases of peritonitis, cases in New Mexico, 58.5% were secondary to perforation of appendix and 27% of patients had perforated colonic lesions from either diverticular disease or other colonic pathology (6). Results of the present study concur with both of these studies (5,6); thus confirming that perforated appendicitis remains the most common cause of secondary peritonitis.

A number of other studies showed different results by demonstrating that colonic perforation was the most common cause of secondary peritonitis. In the study by Gauzit et al, peritonitis originated in the colon (32%), appendix (31%), stomach/duodenum (18%), small bowel (13%) or biliary tract (6%) (3). Sotto et al reported that perforation of the colon was the most common cause of peritonitis in their series (40%) followed by perforation of the stomach (19.1%) whereas perforation of the appendix accounts only for 11.1% (7). Riché et al (8) showed colonic perforations (38.3%), gastroduodenal perforations (21.6%), perforated appendicitis (7.7%) and biliary tract perforations (7.7%). In study by Van Ruler et al., the most frequent cause of secondary peritonitis was perforation of the colon (42%) followed by perforation of small intestine (24%), biliary perforations (9%) and gasroduodenal perforations (9%). Only 4% patients were included with peritonitis due to perforated appendicitis (9). In the present study, the most common cause of secondary peritonitis was perforated appendicitis in agreement with two previous series (5,6) but at variance with others (3,7-9) reflecting difference in disease patterns in the West from our region.

Postoperative peritonitis was a cause of secondary peritonitis in only 3% of cases in our series. The most common bacteria identified were *E. coli* and *B. fragilis*. The identified bacterial species were consistent with other reports of peritonitis cultures in the literature (6,10). The morbidity rate was 23%. Wound complication rate was lower than the other studies (6). The overall mortality rate was 15% in a study performed by Gauzit et al. and morbidity and mortality were significantly lower in patients with appendiceal peritonitis [3]. Sotto et al, found higher mortality rate (25%) that was explained by the high percentage of colonic perforation (40%) in his study compared with appendicular one (11%) as a source of secondary peritonitis [8].

There are studies that show better outcome for patients with secondary peritonitis such as the study reported by Mosdell et al, where the mortality rate for all 480 patients was 6% which is reflected to the high number of appendicular perforation (58%) in the study (7). In our study the overall mortality rate was 4.4% which is very low compared with the other studies (3,6,7). This may be attributed to the high appendicitis in our country, thereby the high percent of perforated appendix as origin of secondary peritonitis. Additionally, the fact that our patients were younger, more often had peritonitis due to appendicitis, and had fewer bacterial species per culture are all possible factors for better outcome.

In conclusion, appendiceal peritonitis was the most common cause of secondary peritonitis in our study. This may explain the lower mortality when compared with reports from elsewhere as appendiceal peritonitis tends to be less severe and has a better prognosis than peritonitis originating in other sites. The rates of severe peritonitis, morbidity, and mortality were significantly lower in patients with appendiceal peritonitis.

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