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Fungal Peritonitis: A Case Series Detailing Clinical Presentations, Diagnostic Challenges, and Management Strategies

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Abstract

Aims: to present the features of fungal peritonitis.

Methodology: The case series comprised three patients: a 52-year-old male with a post-splenectomy subdiaphragmatic abscess, a 64-year-old male with post-operative peritonitis following a perforated duodenal ulcer repair, a 68-year-old female with primary peritonitis. The clinical history, physical examination findings, laboratory and imaging results, surgical reports, and treatment outcomes are presented.

Results: All patients exhibited comorbidities. Non-specific symptoms characterized clinical presentations. Imaging modalities were valuable diagnostic tools. Candida albicans was identified as the causative organism in all cases.

Conclusion: Fungal peritonitis is a clinically challenging condition with non-specific clinical and laboratory manifestations.

Keywords: Peritonitis; Fungal Peritonitis; Comorbidity; Candida albicans.

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Introduction

Fungal peritonitis, a relatively infrequent yet clinically significant complication, poses a substantial challenge to clinicians due to its associated high mortality rates, reported to range from 20% to 30%, and a tendency towards increased morbidity [1-3]. The difficulties in diagnosis and management stem primarily from its frequent occurrence in patients with compromised immune systems [4-6]. This patient population often exhibits atypical clinical presentations, rendering standard diagnostic modalities less informative and therapeutic interventions less effective [7,8]. Consequently, there is a pressing need for improved diagnostic and therapeutic strategies to address this challenging condition. Given the complexity of predicting fungal peritonitis, some authors suggest using specific prognostic tools [9-11].

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In recent years, invasive fungal infections, including peritonitis, have been steadily increasing, thereby augmenting the medical and social significance of this problem [1, 3, 7, 12, 13]. This trend underscores the importance of refining our understanding and managing fungal peritonitis.

While peritoneal dialysis remains a well-recognized risk factor for fungal peritonitis [14, 15], the condition also manifests in patients with various underlying medical conditions, including hepatic, cardiac, renal, and oncologic disorders, as well as in those with metabolic derangements such as obesity [16-20]. Notably, the role of fungal infections in abdominal surgery, particularly in developing postoperative peritonitis, has been increasingly recognized [7, 13, 18, 20]. Postoperative fungal peritonitis is characterized by its diagnostic complexity and often results in suboptimal patient outcomes [21-23]. While diffuse peritonitis is the most commonly encountered presentation, localized fungal intra-abdominal abscesses have also been described [24]. Fungal abscesses can occur in various anatomical locations [25, 26].

Candida albicans is the most frequently identified causative organism in fungal peritonitis [1, 3, 6, 27]. However, other fungal species are also implicated, highlighting the diverse etiology of this condition [28-30].

Patients with diabetes mellitus are susceptible to a wide range of fungal infections [31, 32]. Diabetes is a major risk factor for mycoses affecting the oral cavity, gastrointestinal tract, skin, feet, genitourinary system, and blood, posing significant diagnostic and management challenges and contributing to increased mortality [33, 34]. While fungal infections are commonly reported in these areas, fungal peritonitis in diabetic patients is surprisingly infrequent. Some studies even suggest that diabetes does not elevate the risk of peritonitis during peritoneal dialysis [35]. However, diabetes is generally recognized as a risk factor for peritonitis, particularly postoperative peritonitis [36, 37].

Due to the rarity of fungal peritonitis, large-scale, systematic studies are scarce, making randomization impractical. Consequently, the presentation of individual cases of fungal peritonitis arising from diverse etiologies is crucial for expanding clinicians' knowledge and enhancing their ability to recognize and manage this challenging condition.

Research Problem

The primary objective of this study is to comprehensively assess the clinical and laboratory manifestations of fungal peritonitis, and to analyze patient management strategies and their resulting outcomes.

Research Focus

This study seeks to determine common clinical patterns in the onset and progression of fungal peritonitis.

Research Aim and Research Questions

1. Characterize the clinical presentation of fungal peritonitis, with a focus on identifying common and atypical signs and symptoms across diverse patients.

2. Evaluate the diagnostic utility of standard laboratory and imaging modalities, including complete blood counts, biochemical profiles, ultrasound, and computed tomography, in the context of fungal peritonitis.

3. Assess the efficacy and outcomes of current management strategies, including surgical interventions, antifungal therapy, and supportive care, in patients with fungal peritonitis.

4. Identify potential risk factors and comorbidities associated with the development of fungal peritonitis.

Research Methodology

General Background

The infrequency of fungal peritonitis poses a substantial methodological hurdle, hindering the ability to systematically gather and synthesize data required for reliable conclusions.

Sample / Participants / Group

The study population consisted of three patients who were diagnosed with fungal peritonitis and treated at the Chernivtsi Regional Clinical Hospital between January 2015 and December 2024. These patients were selected based on the availability of complete medical records and microbiological confirmation of fungal peritonitis.

Instrument and Procedures

A detailed overview of the data collection methodology is provided in the following table.

Table 1. Retrospective Data Extraction Methodology

Data Category	Specific Data Extracted	Collection Method
Demographic Information	Age, Sex	Retrospective Medical Record Review
Medical History	Comorbidities, Previous Surgical Procedures	Retrospective Medical Record Review
Clinical Presentation	Presenting Symptoms, Physical Examination Findings	Retrospective Medical Record Review
Laboratory Data	Complete Blood Counts, Biochemical Profiles, Microbiological Culture Results	Retrospective Medical Record Review
Imaging Studies	Ultrasound Reports, Computed Tomography Reports	Retrospective Medical Record Review
Operative Reports	Surgical Procedures, Intra-operative Findings	Retrospective Medical Record Review
Post-operative Course	Treatment Modalities, Complications, Outcomes	Retrospective Medical Record Review
Comorbidity Index	Charlson Comorbidity Index Score	Retrospective Medical Record Review
Microbiological Confirmation	Fungal Organism Isolation/Identification	Peritoneal Fluid/Abscess Sample Analysis

Ethical Considerations

This retrospective study was conducted in accordance with ethical guidelines for medical research. Due to the retrospective nature of the study, formal ethical approval was not required. Patient confidentiality was maintained throughout the study.

Data Analysis

The restricted sample size (n=3) prevented the application of statistical methods. Data were analyzed using descriptive case presentations.

Case Presentation

Case 1: A 52-year-old male was admitted with persistent left upper quadrant abdominal pain and a body temperature of 37-37.4°C. His medical history included chronic alcohol abuse. Eight days prior, he had undergone a splenectomy at a district hospital for a traumatic splenic rupture. He was prescribed intravenous fluids and prophylactic cefazolin. The initial post-operative period was uneventful, but on the fourth day, he developed abdominal pain and fever. An ultrasound revealed a fluid collection under the left diaphragm. He was started on increased antibiotic therapy (cefotaxime 1g twice daily, ornidazole 500mg twice daily), but his condition deteriorated.

Physical examination revealed a moist tongue, a non-distended abdomen, and tenderness with guarding in the left hypochondrium. A vague Blumberg's sign (rebound tenderness) was noted. Tissue induration was present along the midline post-operative scar. Bowel sounds were normal, and the passage of flatus and stool was unimpaired. His pulse was 82 beats per minute, and his blood pressure was 120/80 mmHg. Laboratory tests showed a moderate leukocytosis of 8.2x10^9/L (neutrophils 68% (5.58x10^9/L), lymphocytes 21% (1.72x10^9/L), monocytes 9% (0.75x10^9/L), eosinophils 2% (0.16x10^9/L)), and mild anemia (erythrocytes 2.9 x 10^12/L, hemoglobin 102 g/L). The platelet count was elevated at 764 × 10^9/L. Biochemical tests were unremarkable.

A CT scan revealed partial subcutaneous eventration of small bowel loops, intra-abdominal adhesions, and a 170x89mm fluid collection under the left diaphragm, encapsulated by the greater omentum, stomach wall, and colon wall. A diagnosis of left-sided subdiaphragmatic abscess was made. A relaparotomy was performed. The skin was incised along the previous scar, and the tissues were dissected. A loop of small bowel was found in the subcutaneous tissue. The aponeurosis and peritoneum were incised. Soft adhesions between the small bowel loops and the abdominal wall were lysed. The small bowel loops appeared pink and non-dilated. The greater omentum

defined the left subdiaphragmatic space. The abscess cavity was opened, yielding 70mL of turbid grey pus under pressure. The lower wall of the abscess was formed by the infiltrated, dense wall of the left colic flexure. The moderately infiltrated stomach wall formed the right wall.

The abscess cavity was lavaged repeatedly, and two drainage tubes were inserted. Due to the eventration, the aponeurosis was sutured with a duplication technique. The surgical wound was closed. The patient was started on intravenous meropenem 500mg three times daily, along with saline infusions. Pus culture revealed Candida albicans (10^6 colony-forming units/mL). Meropenem was discontinued, and fluconazole 200mg twice daily was initiated. The patient had an uneventful post-operative course and was discharged. One year later, he underwent surgery for an incisional ventral hernia, which was repaired with a prolene subway mesh. The patient underwent follow-up examinations at one and two years post-hernioplasty without recurrence. No surgery-related health disorders were identified.

Case 2: A 64-year-old male was admitted with persistent moderate abdominal pain and a body temperature of 37.2-37.7°C. His medical history included type 2 diabetes mellitus and ischemic heart disease. Twelve days prior, he had undergone a repair of a perforated duodenal ulcer at a district hospital with abdominal lavage and drainage. After surgery, he developed persistent low-grade fever and abdominal pain. Repeated ultrasound revealed increasing fluid in the lateral gutters and between bowel loops. Conservative management failed, and his condition worsened.

Physical examination revealed a moist tongue and a slightly distended abdomen. The anterior abdominal wall was moderately tender and soft. A vague Blumberg's sign (rebound tenderness) was present throughout the abdomen. Bowel sounds were diminished. Passage of flatus and stool was unimpaired. His pulse was 86 beats per minute, and his blood pressure was 125/85 mmHg. Laboratory tests showed a moderate leukocytosis of 9.8x10^9/L (neutrophils 64% (6.27x10^9/L), myelocytes 2% (0.2x10^9/L), lymphocytes 26% (2.55x10^9/L), monocytes 10% (0.75x10^9/L), eosinophils 2% (0.16x10^9/L)). Erythrocyte count was 3.1 x 10^12/L, haemoglobin 112 g/L, and platelet count was 284 × 10^9/L. Total plasma protein was 59 g/L, urea 9.2 mmol/L, creatinine 110 μmol/L, total bilirubin 23 μmol/L, and glucose 8.2 mmol/L. A CT scan revealed moderate dilatation of small and large bowel loops and free fluid in the lateral gutters, between bowel loops, and in the pelvis. A diagnosis of diffuse post-operative peritonitis was made. A relaparotomy was performed. A moderate infiltrative-adhesive process was found in the abdominal cavity, with small bowel loops adherent to each other and the anterior abdominal wall. Adhesiolysis was performed. The bowel loops were slightly dilated, and the bowel walls were moderately edematous. The greater omentum was moderately swollen. Turbid fluid with fibrin (1L) was aspirated between the bowel loops, lateral gutters, subdiaphragmatic spaces, and pelvis. The stomach was slightly dilated. The duodenal sutures were intact. A pneumocompression test revealed no leakage. The abdominal cavity was lavaged repeatedly with saline. Four drainage tubes were inserted. The surgical wound was closed. The patient was started on intravenous meropenem 500mg three times daily and ornidazole 500mg twice daily, along with saline infusions. Peritoneal fluid culture revealed Candida albicans (10^4 colony-forming units/mL). Meropenem and ornidazole were discontinued, and fluconazole 200mg twice daily was initiated. The patient had an uneventful post-operative course and was discharged. He was followed up for four years without recurrence. No health disorders related to the surgery were found.

Case 3: A 68-year-old female was admitted with diffuse abdominal pain, nausea, and vomiting. She reported a two-day history of progressive abdominal discomfort, initially localized to the epigastric region before becoming generalized. There was also a delay in the passage of flatus and stool. Her past medical history was significant for ischemic heart disease, atrial fibrillation, and chronic hepatitis.

Upon physical examination, her tongue was moist, and her abdomen was slightly distended. The anterior abdominal wall exhibited tenderness and rigidity, with a weakly positive Blumberg's sign (rebound tenderness) elicited throughout the abdomen. Bowel sounds were diminished. Her pulse was 112 beats per minute and irregular, and her blood pressure was 115/80 mmHg. Laboratory investigations revealed a leukocytosis of 10.2x10^9/L, with a differential count showing neutrophils 62% (6.32x10^9/L), lymphocytes 24% (2.45x10^9/L), monocytes 11% (1.12x10^9/L), and eosinophils 1% (0.10x10^9/L). Her erythrocyte count was 3.3 x 10^12/L, haemoglobin 110 g/L, and platelet count 267 × 10^9/L. Biochemical analysis showed total plasma protein 62 g/L, urea 8.3 mmol/L, creatinine 121 μmol/L, total bilirubin 20 μmol/L, and glucose 6.2 mmol/L. An abdominal ultrasound revealed dilated small bowel loops, hepatomegaly, and free fluid in all abdominal quadrants. A plain abdominal radiograph showed dilated bowel loops with air-fluid levels but no evidence of free air. Based on these findings, a provisional diagnosis of mesenteric thrombosis and acute diffuse peritonitis was made.

An exploratory laparotomy was performed. Intraoperatively, a diffuse, turbid exudate with an unpleasant odour was observed throughout the abdominal cavity. The parietal and visceral peritoneum were hyperemic. The oesophagus, stomach, and duodenum appeared grossly normal. The liver was enlarged, edematous, and

homogeneous, with a scarred capsule. The gallbladder, spleen, and pancreas were unremarkable. The small and large bowels were moderately distended and filled with fluid. The uterus, ovaries, and fallopian tubes were unremarkable. A thorough reexploration revealed no signs of organ perforation or other identifiable source of peritonitis. Therefore, a diagnosis of acute primary diffuse peritonitis was established. The abdominal cavity was extensively lavaged with saline, and four drainage tubes were inserted. Due to the absence of a clear source, the surgical wound was closed with temporary sutures. A planned relaparotomy was performed the following day. This revealed a small amount of clear fluid and a reduction in peritoneal hyperemia. The condition of the intra-abdominal organs has improved. The abdominal cavity was again lavaged with saline, and the surgical wound was closed definitively. The patient was started on intravenous cefotaxime 1g twice daily and ornidazole 500mg twice daily, along with saline infusions. Cultures of the peritoneal exudate subsequently grew Candida albicans (10^7 colony-forming units/mL). In response, cefotaxime and ornidazole were discontinued, and fluconazole 200mg twice daily was commenced. The patient experienced an uneventful postoperative course and was discharged from the hospital. One year later, she underwent surgery for an incisional ventral hernia, which was repaired with a sublay prolene mesh. She did not return for follow-up examinations post-hernioplasty.

Discussion

The three cases presented in this series, while diverse in their primary etiologies, share several commonalities that highlight the challenges and complexities of managing fungal peritonitis. Notably, all patients exhibited comorbidities, suggesting that comorbidity plays an important role in the development of this condition. In this study, Charlson Comorbidity Index (CCI) scores varied among patients with different abdominal infections. A male with a subphrenic abscess had a CCI score of 1. A male with postoperative peritonitis scored 4, while a female with primary peritonitis scored 3. Thus, while there was some variation, all CCI scores were relatively low. However, it's important to note that many patients with similar or higher CCI scores do not develop fungal peritonitis.

The male patient with postoperative diffuse peritonitis presented with diabetes mellitus, a recognized risk factor for fungal infections associated with immune dysregulation [32]. Additionally, the literature suggests a potential association between gastroduodenal ulcer perforation and fungal flora [9]. The female patient with primary peritonitis exhibited multiple comorbidities, including chronic hepatitis, which is also implicated in fungal infection susceptibility due to its impact on immune function [16]. The male patient with a subdiaphragmatic abscess reported chronic alcohol consumption, a known immunosuppressant [38]. Furthermore, this patient's history of trauma, splenectomy, blood loss, and erythrocyte transfusions likely contributed to immune compromise [39]. However, a definitive causal link between these conditions and fungal infections remains unclear, with only isolated case reports documented in the literature.

The cause of peritonitis in a female remained unclear. Primary peritonitis is peritonitis that occurs without evidence of its cause in the abdominal cavity [40]. Most often, primary peritonitis occurs in patients with liver cirrhosis and ascites [41]. Medical instruments can also introduce primary infection to your peritoneal cavity. Dialysis and tube feeding are two ways this can happen. However, the patient did not have any of these reasons. During pre-surgical examination, during the surgery for an incisional hernia, signs of liver cirrhosis and ascites were not found.

Following inpatient treatment for peritonitis, all patients underwent immunological consultation, resulting in a diagnosis of D83.9, unspecified common variable immunodeficiency (CVID). However, pre-hernioplasty evaluations and serial examinations of the male patient with diffuse postoperative peritonitis revealed no clinical or laboratory evidence of immunodeficiency. This discrepancy suggests a transient immune dysfunction. The observed immunological changes may have been multifactorial, potentially stemming from pre-existing chronic conditions and the physiological stress of surgical interventions. Furthermore, the immunologist's prescribed treatment may have facilitated the resolution of these transient immune abnormalities.

A consistent clinical presentation of fungal peritonitis was notably absent across all patients, characterized by vague symptomatology. Patients with prior surgical interventions received prolonged conservative management due to the insidious nature of postoperative complications. The female patient with primary peritonitis presented late, delaying medical intervention by two days due to initially mild symptoms. Upon admission, clinical findings remained subtle. None of the patients exhibited hemodynamic instability typically associated with acute peritonitis.

While typical haematological findings in peritonitis include hyperleukocytosis (>12 x 10^9/L) and marked neutrophilia (>70%) [36,37], these features were not consistently observed in our patient cohort. Leukocyte counts were not sufficiently elevated to definitively diagnose peritonitis, and a significant increase in neutrophils was absent. Myelocytes, indicative of stress and potential granulocytopoietic insufficiency [42], were detected in the male patient with diffuse postoperative peritonitis. Notably, a trend towards neutropenia was observed across all patients, potentially reflecting underlying immune dysregulation [42].

Biochemical analysis in the male patient with a subphrenic abscess revealed no significant abnormalities. In the male patient with diffuse postoperative peritonitis, biochemical alterations (total plasma protein 59 g/L, urea 9.2 mmol/L, creatinine 110 μmol/L, total bilirubin 23 μmol/L) were observed; however, these findings were not diagnostic for peritonitis, as they could be attributed to pre-existing conditions and surgical trauma. In the female patient, elevated urea (8.3 mmol/L) and creatinine (121 μmol/L) suggested potential renal dysfunction, while other parameters (total plasma protein 62 g/L, total bilirubin 20 μmol/L) remained within normal limits.

When clinical manifestations are ambiguous, and laboratory findings are inconclusive, imaging modalities become paramount for diagnosis, with ultrasound and computed tomography being primary tools [36,37]. However, the efficacy of these modalities is often diminished in patients with prior surgical interventions. In males with postoperative peritonitis, the presence of abdominal fluid detected by ultrasound following initial surgery could represent a sequela of the primary pathology (trauma, ulcer perforation) or a consequence of the surgical procedure itself. Close clinical observation and the surgeon's expertise are critical in these instances. In the female patient with primary peritonitis, imaging diagnostics were instrumental in establishing the diagnosis and guiding appropriate management.

A shared characteristic among the presented patients was the development of incisional hernias in two cases. It is well-established that the risk of incisional hernia increases with repeat abdominal surgeries [43]. Notably, all patients in this series underwent at least two surgical procedures. In the male patient with a subphrenic abscess, eventration was identified via computed tomography and during re-operation. Despite aponeurosis duplication, an incisional hernia developed. While mesh reinforcement is generally recommended for incisional hernia prevention in high-risk patients [44], its application in the setting of peritonitis remains controversial. Mesh placement may increase the risk of early wound complications. Therefore, alternative preventive strategies should be considered [45].

A critical consideration in the management of fungal peritonitis is antibiotic therapy. The established principle is to initiate broad-spectrum antibiotics postoperatively, tailoring the regimen based on microbiological culture results [reference]. However, the use of potent antibiotics can promote the proliferation of fungal microflora. Intraoperative diagnosis of fungal peritonitis is challenging due to the nonspecific nature of abdominal cavity findings (exudate, hyperemia, oedema, bowel dilatation), which are largely indistinguishable from other forms of peritonitis regardless of microbial aetiology. Therefore, timely and accurate microbiological diagnostics, particularly utilizing automated rapid testing, is crucial for prompt result acquisition and appropriate treatment modification.

In summary, comorbidity leading to immune dysfunction is a significant risk factor for fungal peritonitis. Clinical presentation is often vague, and immune-related haematological changes may be subtle, complicating diagnoses, particularly in postoperative cases. While imaging can be helpful, clinical observation and surgical expertise remain paramount in postoperative fungal peritonitis. Patients are at increased risk for incisional hernias. Standard antibiotic therapy may paradoxically promote fungal proliferation. Effective management and prevention of postoperative complications require a robust prognostic scale, which is currently lacking. However, it's crucial to acknowledge that no prognostic tool can replace clinical judgment and experience.

Conclusions and Implications

Fungal peritonitis remains a rare but clinically challenging condition, primarily affecting individuals with compromised immune systems due to comorbidities or post-surgical states. Its insidious presentation, characterized by non-specific clinical and laboratory findings, necessitates a high index of suspicion, particularly in post-operative settings. Definitive diagnosis relies on microbiological confirmation, obtained post-operatively, contributing to delays in appropriate antifungal therapy and increased morbidity. The consistent development of incisional hernias in these patients highlights the need for meticulous surgical technique and post-operative care. Further research is warranted to develop rapid diagnostic tools and optimize management strategies for this complex condition.

Limitations of the study

This study is subject to several limitations. First, the retrospective nature of the case series design may introduce bias, as data collection relies on existing medical records. Second, the small sample size (n=3) limits the generalizability of the findings and precludes statistical analysis. Third, the study is conducted at a single center, which may further limit its external validity. Fourth, the lack of a control group prevents comparative analysis with other forms of peritonitis or non-infected patients. Fifth, the diagnosis of Common variable immunodeficiency was done after the treatment of peritonitis, so we cannot know the exact role of this diagnosis in the development of the peritonitis.

Suggestions for Future Research

Future research should prioritise several critical areas to advance the understanding and management of fungal peritonitis. Firstly, efforts should be directed towards the development of rapid intra-operative diagnostic tools that can facilitate timely initiation of antifungal therapy. Additionally, large-scale, multi-centre studies are necessary to validate the findings of this case series and enhance the generalisability of the results. It is also essential to investigate the role of immunodeficiency in the pathogenesis of fungal peritonitis, particularly focusing on the prevalence of undiagnosed immunodeficiencies within this patient population. Furthermore, evaluating the efficacy of various antifungal regimens will be key to optimising treatment protocols. Developing risk stratification models to accurately identify patients at high risk of fungal peritonitis is another important area of inquiry. Lastly, research should explore effective strategies for the prevention of incisional hernias in individuals affected by this condition.

Declarations

Author Contributions

All authors contributed to the examination and management of the presented patients, conception and design of the study, data collection and analysis, and manuscript preparation.

Data Availability Statement

The data supporting the findings of this study are not publicly available due to ethical and legal constraints related to patient privacy and medical record confidentiality.

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Institutional Review Board Statement

This retrospective case series was conducted in accordance with ethical guidelines for medical research. Formal ethical approval was not required due to the retrospective nature of the study and the use of de-identified patient data.

Informed Consent Statement

Informed consent for diagnostic and therapeutic procedures was obtained from all patients after they received comprehensive information about the procedures and potential outcomes. Informed consent for publication was not obtained because the data presented are depersonalized.

Conflicts of Interest

The authors declare that there is no conflict of interests regarding the publication of this manuscript. In addition, ethical issues, including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, and redundancies have been completely observed by the authors.

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