Case Report

Trichosporon Peritonitis Following Duodenal Perforation

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ABSTRACT

Fungal peritonitis, which was once a rare entity, is becoming increasingly common due to various immunocompromised conditions. *Candida* species are considered the common cause of fungal peritonitis in most cases. However, at present, other yeasts and filamentous fungi are replacing the dominance of *Candida albicans* as well as other *Candida* species. *Trichosporon* species are widely distributed in nature and are normal flora in the gastrointestinal tract of humans. Ever since the report of disseminated trichosporonosis in 1970, several cases of infections by various *Trichosporon* species in different clinical patients have been published. Here, we present a patient with *Trichosporon* peritonitis after duodenal perforation. To the best of our knowledge, this is the first case report of its kind from India.

Key Words: Duodenal perforation, fungal peritonitis, Trichosporon

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Fungal peritonitis is an uncommon entity, but it has serious consequences in patients with peritoneal dialysis and intraabdominal pathological conditions.^[1] *Trichosporon*—the basidiomycetous yeast—is a medically important genus, which includes some of the pathogens that cause infections in immunocompetent host and disseminated infections in immunocompromised hosts.^[2] *Trichosporon* species are soil inhabitants and common colonizers of skin and gastrointestinal tract. *Trichosporon* species are widely distributed in nature and are commonly isolated from soil and other environmental sources. They are also commensals in human gastrointestinal and respiratory tracts.^[3]

The first case of disseminated trichosporonosis was reported in 1970 by Watson *et al.*^[4] in a case of leukemia. Since then several reports of Trichosporon infection in immunocompromised patients have been published.^[5] More recently, Ahmad *et al.*, characterized *Trichosporon* species from various clinical samples from Kuwait. This report indicates the ability of this opportunistic fungus in causing infection in a variety of patients by different routes.^[6]

Most commonly, *T. asahii* has been reported, with the other species being *T. inkin*, *T. asteroids*, *T. cutaneum* and *T. mucoides*.^[7] We report a patient with peritonitis that was caused by *T. asahii*. To our knowledge, this is the first case report of peritonitis following soft tissue rupture caused by *T. asahii* from South India.

CASE REPORT

A 50-year-old male agriculturist by occupation was referred to our hospital for ventilatory support and management of renal failure. The patient had undergone exploratory laparotomy for generalized peritonitis and duodenal perforation two days ago in a general hospital; in this hospital, he was admitted with a history of intermittent epigastric pain for 5 years for which he was taking antacids for the last two years on the prescription of a local doctor.

At the time of admission in the general hospital, the patient had renal failure with hypokalemia and complained of severe abdominal pain and a decreased urine output for the last 5 days, with no history of fever or chest pain. He was detected as having hollow viscus perforation for which exploratory laparotomy was performed on him on the same day. After surgery, the patient had oliguria, puffiness of face and breathlessness, severe metabolic acidosis, hypotension, altered sensorium and fever; hence, he was referred to the tertiary care hospital for management of acute crisis. He was put on ventilatory support and antibiotics (aztreonam, metronidazole and intravenous fluids) considering bacterial peritonitis. Despite all these measures, the patient's condition deteriorated.

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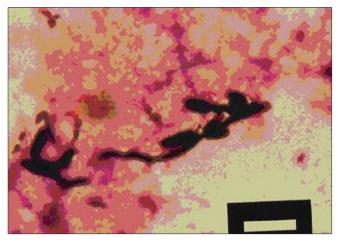


Figure 1: Grams stain, showing arthroconidia and big yeast like fungi

Peripheral blood examination showed normocytic normochromic anemia with elongated cells, stomatocytes and target cells. The results of laboratory examinations were as follows: Neutrophilic leucocytosis (WBC, 16,300/mm³); thrombocytopenia (platelets, 95 lakhs/mm3; ESR, 36 mm in the first hour by Wintrobe's method; serum urea, 204 mg/dl; serum creatinine, 8.2 mg/dl; Na⁺, 135 meq; K⁺, 6.2 meq; total bilirubin, 3.1 mg/dl; conjugated bilirubin, 1.1 mg/dl. Ultra - sonography of the abdomen revealed mild parenchymal renal disease, minimal perisplenic collection in the abdomen and dilated intrahepatic biliary ducts. Under all aseptic precautions, single lumen 16-G, 8-inch (20 cm) catheter was placed in the right inguinal vein under local anesthesia. Urine microscopy showed 2-3 pus cells/HPF. Peritoneal fluid Gram stain smear showed occasional pus cells with few budding yeast cells and many barrel-shaped arthroconidia [Figure 1]. Blood culture was sterile after 24 hours.

Peritoneal fluid culture showed whitish wrinkled colonies with indefinite size and shape on blood agar after 24 hours at 37°C. The growth on MacConkey resembled that on blood agar, but was less luxuriant than growth on blood agar. Gram staining of the colonies showed barrel-shaped arthroconidia with few budding yeast cells.^[1] Sugar fermentation, urease test was (+/-) and slide culture on corn meal agar was performed for further identification [Figure 2].^[2] On the basis of these tests, the isolate was identified as *T. beigelii*.

DISCUSSION

Invasive infections by rare and opportunistic fungi are becoming increasingly common in immunocompromised individuals and the treatment is a challenge to the treating physician. The increasing incidence may be a result of multiple factors including the use of invasive surgical



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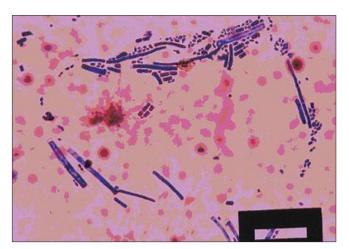


Figure 2: Morphology of the fungus on corn meal agar

procedures and prosthetic materials, such as prosthetic heart valves, central venous catheters and intensive chemotherapy, which exaggerates immunosuppression. Because of immunosuppressed status of the patient, several nonpathogenic fungi prevalent in the environment can cause life-threatening infections.

Fungal pathogens are uncommon isolates in the setting of peritonitis. Peritonitis secondary to perforation of gastrointestinal tract with gross contamination is becoming increasingly common. *Candida* species are the most common culprits of fugal peritonitis; however, other yeasts and filamentous fungi are uncommon.^[8] Almoujahed *et al.*, reported 15 cases of fungal peritonitis after gastrointestinal perforation or anastomotic leakage, of which one was due to *T. pullulans*.^[1] Sanjay Gupta and Robin Kausik had analyzed the site and causes of perforations. They reported the duodenum as the most frequent site of perforation associated with peritonitis.^[9] The causative agents of peritonitis differ at different perforation sites. Gastroduodenal perforations have been reported to be most commonly associated with fugal peritonitis.^[10]

In our patient, there was a long history of epigastric pain and intake of antacids, followed by sudden onset of unbearable pain that was recognized as due to perforation of duodenum, leading to acute peritonitis.

Fungal peritonitis is most commonly caused by *Candida* species, but prophylaxis with fluconazole in patients prone to recurrent gastrointestinal perforations or anastomotic leakages has reduced the incidence of *Candida* peritonitis, because of which the rare opportunistic fungi are becoming increasingly common. Thus, whenever fungal peritonitis is suspected, fungi other than *Candida* species should also be kept in mind.

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