

Spontaneous Fungal Peritonitis Caused by *Pichia norvegensis* in a Patient With Budd-Chiari Syndrome and Noncirrhotic Portal Hypertension: A Case Report

Review began 08/19/2026

Review ended 10/19/2026

Published 10/26/2026

© Copyright 2026

Costa et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118625

Tiago Jorge Costa¹, Bernardo Belchior¹, Diana M. Ferreira¹, Joana C. Costa¹, Arsénio Santos¹

1. Internal Medicine, Hospitais da Universidade de Coimbra, Coimbra, PRT

Corresponding author: Tiago Jorge Costa, tiagojorgecbt@gmail.com

Abstract

Spontaneous fungal peritonitis (SFP) is an uncommon but potentially life-threatening complication of advanced liver disease, predominantly described in patients with cirrhosis. *Pichia norvegensis* (formerly *Candida norvegensis*) is a rare human pathogen for which clinical experience and antifungal susceptibility data remain limited.

We report the case of a 52-year-old man with Budd-Chiari syndrome secondary to polycythemia vera progressing to myelofibrosis, advanced chronic liver disease, severe noncirrhotic portal hypertension, and a transjugular intrahepatic portosystemic shunt (TIPS). He presented with abdominal pain and fever following a recent hospitalization for gastric variceal bleeding, during which he had received ceftriaxone prophylaxis. Ascitic fluid analysis revealed marked neutrocytic peritonitis (11,798 polymorphonuclear neutrophils/ μ L), with negative bacterial cultures. *P. norvegensis* was identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) in two separate ascitic fluid specimens. Antifungal susceptibility testing revealed a micafungin minimum inhibitory concentration of 0.031 mg/L; however, validated species-specific clinical breakpoints were unavailable. Empirical micafungin was administered at 100 mg intravenously daily for 22 days, with favorable clinical evolution, a progressive reduction in ascitic neutrophil counts, and a negative follow-up fungal culture. Concurrent portal hypertensive complications required TIPS revision.

This case illustrates the occurrence of fungal peritonitis associated with an uncommon yeast in a patient with severe portal hypertension without histologically established cirrhosis. Although a secondary intra-abdominal source could not be definitively excluded, repeated fungal isolation supported the clinical significance of the organism. The case highlights the importance of considering fungal infection in patients with neutrocytic ascites and negative bacterial cultures, as well as the need for reliable species identification and cautious interpretation of antifungal susceptibility results.

Categories: Internal Medicine, Infectious Disease, Gastroenterology

Keywords: antifungal susceptibility, budd-chiari syndrome, non-cirrhotic portal hypertension, *pichia norvegensis*, spontaneous fungal peritonitis

Introduction

Spontaneous fungal peritonitis (SFP) is an uncommon but severe infectious complication of advanced liver disease, characterized by fungal infection of ascitic fluid associated with a polymorphonuclear neutrophil count ≥ 250 cells/mm³ in the absence of an identifiable secondary intra-abdominal source. Although substantially less frequent than spontaneous bacterial peritonitis, SFP has been associated with high mortality. Reported in-hospital mortality rates range from 33% to 100%, while one-month mortality rates range from 50% to 73%; however, these estimates are derived predominantly from small, retrospective studies and should be interpreted cautiously [1].

SFP has been described predominantly in patients with cirrhosis and advanced hepatic dysfunction. Previous antibiotic exposure and healthcare-associated or nosocomial infection are frequently reported in affected patients, although their individual contributions to the development of fungal infection remain uncertain [1,2]. *Candida* species account for most reported cases, with *Candida albicans* remaining the most frequently isolated organism, although non-*albicans Candida* species are also increasingly recognized [1-3].

Pichia norvegensis (formerly *Candida norvegensis*) is an uncommon human fungal pathogen, with limited published experience regarding invasive infection. Previous microbiological studies have reported reduced susceptibility to azole antifungals, particularly fluconazole [4,5]. Reliable species-level identification is therefore important when this organism is recovered from a normally sterile site. Interpretation of antifungal susceptibility results also requires caution because validated species-specific clinical breakpoints

How to cite this article

Costa T, Belchior B, Ferreira D M, et al. (October 26, 2026) Spontaneous Fungal Peritonitis Caused by *Pichia norvegensis* in a Patient With Budd-Chiari Syndrome and Noncirrhotic Portal Hypertension: A Case Report. Cureus 18(10): e118625. DOI 10.7759/cureus.118625

are unavailable.

We report a case of SFP associated with *P. norvegensis* in a patient with Budd-Chiari syndrome, advanced chronic liver disease, and severe noncirrhotic portal hypertension. This presentation is particularly noteworthy because the existing literature on SFP predominantly concerns patients with cirrhosis. The case highlights the diagnostic challenges associated with an uncommon fungal pathogen, including species identification, interpretation of antifungal susceptibility testing, and assessment of a possible secondary intra-abdominal source.

Case Presentation

A 52-year-old man presented to the emergency department with a one-day history of diffuse abdominal pain, fever, and anorexia, without vomiting, gastrointestinal bleeding, changes in bowel habits, or respiratory or neurological symptoms. His medical history included Budd-Chiari syndrome associated with polycythemia vera progressing to myelofibrosis, complicated by advanced chronic liver disease, severe noncirrhotic portal hypertension, and refractory ascites. A transjugular intrahepatic portosystemic shunt (TIPS) had been placed 18 years earlier. His calculated Model for End-Stage Liver Disease-sodium (MELD-Na) score on admission was 18, although its interpretation was limited by concomitant warfarin therapy, which could influence the international normalized ratio (INR).

Less than one month before the current admission, he had been hospitalized for nine days following gastric variceal bleeding complicated by hemorrhagic shock, during which he received ceftriaxone prophylaxis. There was no documented recent abdominal surgery, central venous catheter exposure, parenteral nutrition, or previous antifungal treatment. His chronic medication included warfarin, adjusted to a target INR of 2-3, diuretics, and febuxostat.

On admission, the patient was hemodynamically stable and afebrile, despite the reported history of fever. He was anicteric, without signs of hepatic encephalopathy. Abdominal examination revealed distension, collateral venous circulation, marked splenomegaly, and tense ascites, without guarding. Laboratory investigations demonstrated an inflammatory response, with a C-reactive protein level of 14.5 mg/dL (reference range: <0.50 mg/dL), procalcitonin of 5.7 ng/mL (reference range: 0-0.5 ng/mL), and neutrophilia of $8.22 \times 10^9/L$ (reference range: $1.50-7.70 \times 10^9/L$). Hemoglobin was 9.1 g/dL (reference range: 12.0-15.6 g/dL). Additional laboratory results included a white blood cell count of $9.5 \times 10^9/L$, platelet count of $200 \times 10^9/L$, serum sodium of 135 mmol/L, creatinine of 0.95 mg/dL, total bilirubin of 3.2 mg/dL, and INR of 1.67. During hospitalization, acute kidney injury developed, with serum creatinine reaching a peak of 1.83 mg/dL.

Abdominal computed tomography (CT) angiography performed on Day 0 demonstrated chronic liver disease, a patent TIPS, massive splenomegaly measuring approximately 23.1 cm, and multiple splenic infarctions, the largest measuring 14.6 cm. Additional findings included partial splenic vein thrombosis, large-volume ascites, and peritoneal enhancement (Figure 1). No bowel perforation or intra-abdominal abscess was reported. Hepatic nodules were also identified and initially categorized as Liver Imaging Reporting and Data System (LI-RADS) category 4 in the radiological report. Subsequent magnetic resonance imaging demonstrated at least six nodular lesions in segments VII and VIII, measuring up to 17 mm, which were considered possibly dysplastic. Given the underlying Budd-Chiari syndrome and absence of established cirrhosis, the initial LI-RADS categorization was interpreted cautiously. Doppler ultrasonography demonstrated TIPS patency but abnormal portal flow and velocities suggestive of shunt dysfunction.



FIGURE 1: Contrast-enhanced abdominal computed tomography showing marked splenomegaly, splenic infarction, and ascites.

Coronal contrast-enhanced computed tomography image demonstrating marked splenomegaly, measuring approximately 23.1 cm in longitudinal diameter, with a large area of infarction involving the superior splenic pole (green arrow). Abundant ascites is also visible (blue arrows).

Blood cultures obtained on Day 1 were negative. Diagnostic paracentesis performed on Day 2 revealed marked neutrocytic ascites, with 12,744 total cells/ μ L, 12,725 leukocytes/ μ L, and 11,798 polymorphonuclear neutrophils/ μ L. Ascitic fluid biochemical analysis showed a total protein concentration of 2.6 g/dL, glucose of 49 mg/dL, lactate dehydrogenase (LDH) of 806 U/L, and albumin of 1.1 g/dL. The serum albumin concentration measured on the same day was 2.0 g/dL, yielding a serum-ascites albumin gradient (SAAG) of 0.9 g/dL. This value was discordant with the patient's established severe portal hypertension.

Empirical antibacterial therapy with piperacillin-tazobactam was initiated. Bacterial ascitic fluid cultures remained negative, whereas fungal culture yielded *P. norvegensis*, identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS). A repeat paracentesis on Day 5 demonstrated 4,981 total cells/ μ L, 4,973 leukocytes/ μ L, and 4,585 polymorphonuclear neutrophils/ μ L. A second independent ascitic fluid culture again yielded *P. norvegensis*, supporting the clinical significance of the fungal isolation.

Antifungal susceptibility testing was performed using a broth microdilution method based on European Committee on Antimicrobial Susceptibility Testing (EUCAST) methodology. The minimum inhibitory concentrations (MICs) were 1 mg/L for amphotericin B, 16 mg/L for fluconazole, 0.125 mg/L for voriconazole, 0.031 mg/L for micafungin, and 0.125 mg/L for posaconazole. The laboratory reported these results as resistant to amphotericin B, fluconazole, and posaconazole, and intermediate to voriconazole and micafungin. However, these categorical interpretations were based on extrapolated criteria, as validated species-specific clinical breakpoints for *P. norvegensis* were unavailable. Accordingly, the numerical MIC values were considered the primary laboratory findings, and the categorical interpretations were treated with caution. Anidulafungin was not tested.

Micafungin was initiated empirically on Day 5 at a dose of 100 mg intravenously once daily, before antifungal susceptibility testing results became available. Treatment was continued for a total of 22 days in view of the favorable clinical and laboratory evolution. Follow-up paracentesis performed on Day 22 demonstrated 460 total cells/ μ L, 454 leukocytes/ μ L, and 214 polymorphonuclear neutrophils/ μ L, with a negative fungal culture. These findings documented a substantial reduction in ascitic neutrophilia and microbiological improvement during treatment, although they do not independently establish the in vitro susceptibility of *P. norvegensis* to micafungin.

The possibility of secondary peritonitis was considered in view of the markedly elevated ascitic neutrophil count and biochemical findings, including elevated ascitic protein and LDH concentrations and low glucose. Although abdominal CT did not identify a reported bowel perforation or intra-abdominal abscess, there was no recent abdominal surgery, and bacterial ascitic cultures were negative, a secondary intra-abdominal source could not be definitively excluded.

Separately, persistent portal hypertension and abnormal TIPS hemodynamics prompted transjugular portography, which demonstrated a portal pressure of 25 mmHg. TIPS revision was performed through placement of additional stents and balloon angioplasty. A large perigastric varix was also embolized using coils and an Amplatzer Vascular Plug (Abbott, Plymouth, Minnesota, USA) (Figure 2). This intervention was undertaken to address portal hypertensive complications and shunt dysfunction, rather than as treatment for the fungal infection. The procedure was uneventful.

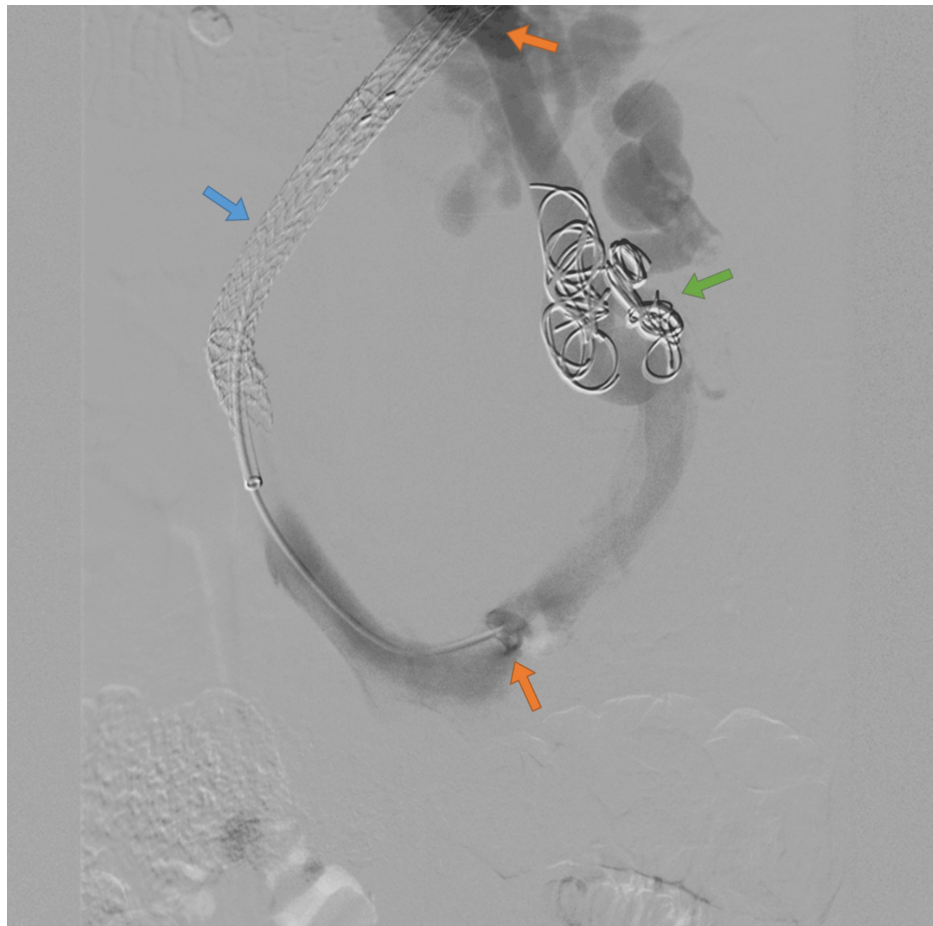


FIGURE 2: Transjugular intrahepatic portosystemic shunt (TIPS) revision and embolization of portosystemic collaterals.

Fluoroscopic image obtained during TIPS revision, demonstrating the revised shunt with additional stent placement (blue arrow) and embolization coils within a large portosystemic collateral (green arrow). Contrast-opacified vascular structures are also visible (orange arrows). The procedure included balloon angioplasty of the TIPS and embolization of a large perigastric varix using coils and an Amplatzer Vascular Plug (Abbott, Plymouth, Minnesota, USA).

Histological examination of a transjugular liver biopsy demonstrated chronic congestive hepatopathy associated with Budd-Chiari syndrome, with septal and perisinusoidal/pericellular fibrosis, regenerative nodularity, and extramedullary hematopoiesis involving all three hematopoietic lineages. There was no histological evidence of cirrhosis or malignancy.

The patient's clinical and laboratory parameters progressively improved, and he was discharged after 23 days of hospitalization. He was referred for hepatology follow-up and liver transplantation evaluation. At approximately two years of follow-up, he remained clinically stable, without documented recurrence of fungal peritonitis.

Discussion

SFP is an uncommon but potentially life-threatening complication of advanced liver disease. Most available evidence comes from small retrospective studies involving patients with cirrhosis, which have reported substantial short-term mortality. A systematic review including 82 patients described in-hospital mortality ranging from 33.3% to 100% and one-month mortality ranging from 50% to 73.3% [1]. Bremmer et al. reported a 28-day mortality rate of 56% [3], while another small case series documented acute kidney injury in six of 10 patients and six deaths within 28 days [6]. Although these findings highlight the severity of SFP, the heterogeneity and limited size of the available studies preclude precise estimates of mortality or direct extrapolation to patients with noncirrhotic portal hypertension.

The present case is noteworthy because fungal peritonitis developed in a patient with Budd-Chiari syndrome, advanced chronic liver disease, and severe portal hypertension despite the absence of histologically established cirrhosis. Published studies of SFP have predominantly focused on patients with

cirrhosis [1,3,7], and evidence concerning its occurrence in noncirrhotic portal hypertension remains limited. This case therefore suggests that fungal peritonitis should also be considered in patients with severe portal hypertension and advanced hepatic dysfunction without established cirrhosis, while recognizing that a single observation cannot define the incidence or clinical characteristics of SFP in this population.

Several clinical features may have increased the patient's vulnerability to infection, including advanced hepatic dysfunction, recent gastric variceal hemorrhage, hospitalization, and prior antibiotic exposure. Previous antibiotic therapy and healthcare-associated or nosocomial infection have been frequently reported among patients with SFP [1,2,7]. In this case, ceftriaxone was administered during a recent hospitalization, whereas piperacillin-tazobactam was initiated only after the onset of the current episode. Although antibiotic exposure may be relevant to the clinical context, its contribution to the development of fungal peritonitis cannot be established. Similarly, the coexistence of several potential predisposing conditions prevents identification of any single causal factor.

The isolation of *P. norvegensis* is particularly unusual. This yeast is rarely implicated in invasive human infection, and published clinical experience remains limited [8]. Peritoneal infection caused by *P. norvegensis* has previously been described in a patient undergoing continuous ambulatory peritoneal dialysis [9], a setting that differs substantially from ascites associated with advanced liver disease. In the present case, *P. norvegensis* was identified by MALDI-TOF MS and recovered from two independently collected ascitic fluid specimens. Repeated isolation in the presence of marked neutrocytic ascites supports the clinical significance of the organism and makes incidental contamination less likely. However, species identification was not independently confirmed by molecular sequencing or a reference laboratory.

An important diagnostic consideration is the distinction between spontaneous and secondary peritonitis. The initial ascitic fluid analysis demonstrated 11,798 polymorphonuclear neutrophils/ μ L, a total protein concentration of 2.6 g/dL, glucose of 49 mg/dL, and LDH of 806 U/L. The elevated protein concentration, reduced glucose level, and increased LDH raise concern for a possible secondary intra-abdominal source. Although abdominal CT did not identify bowel perforation or an intra-abdominal abscess, there was no recent abdominal surgery, and bacterial cultures remained negative, these findings do not definitively exclude secondary peritonitis. Accordingly, the diagnosis of SFP should be interpreted in the context of this residual uncertainty. Nevertheless, the recovery of *P. norvegensis* from two separate ascitic fluid specimens, together with the pronounced inflammatory response, provides substantial evidence of clinically significant fungal peritonitis.

Antifungal susceptibility testing presents additional challenges for uncommon yeast species. Previous microbiological studies have reported reduced susceptibility of *P. norvegensis* to azole antifungals, particularly fluconazole [4,5]. In our case, broth microdilution testing based on EUCAST methodology yielded MICs of 16 mg/L for fluconazole, 1 mg/L for amphotericin B, 0.125 mg/L for voriconazole, 0.125 mg/L for posaconazole, and 0.031 mg/L for micafungin. Although the laboratory assigned resistant or intermediate categories using extrapolated interpretative criteria, validated species-specific clinical breakpoints were unavailable. These categorical results should therefore not be regarded as established clinical susceptibility classifications. In particular, the present findings do not justify conclusions regarding intrinsic antifungal resistance in *P. norvegensis*.

Micafungin was initiated empirically on Day 5 before the susceptibility results became available, and was continued at 100 mg intravenously daily for 22 days. Infectious Diseases Society of America guidelines recommend echinocandins as initial treatment for several forms of invasive candidiasis [10], although these recommendations are not specific to *P. norvegensis* or to the precise clinical circumstances of this case. The decision to continue micafungin was supported by the patient's favorable clinical evolution and progressive reduction in ascitic neutrophilia. Follow-up paracentesis on Day 22 showed a polymorphonuclear neutrophil count of 214 cells/ μ L and a negative fungal culture. However, the follow-up sample was obtained during antifungal treatment, and the observed improvement cannot establish that the isolate was susceptible to micafungin or that the drug alone accounted for the favorable outcome.

Another unusual finding was the low SAAG of 0.9 g/dL, despite well-established severe portal hypertension. Although a SAAG ≥ 1.1 g/dL is generally associated with portal hypertensive ascites, its diagnostic performance is not absolute, and a lower value does not necessarily exclude portal hypertension [11]. The discordance observed in this case should therefore be interpreted cautiously. There is insufficient evidence to attribute the low SAAG specifically to the fungal infection. The diagnosis of peritonitis was primarily supported by the markedly elevated ascitic polymorphonuclear neutrophil count and repeated isolation of *P. norvegensis*.

The extensive splenic infarctions, partial splenic vein thrombosis, and TIPS dysfunction further illustrate the complexity of the patient's underlying portal vascular disease. Although these abnormalities formed part of the clinical context, their contribution to the development of fungal peritonitis cannot be determined. TIPS revision and embolization of portosystemic collaterals were undertaken to address portal hypertensive complications and abnormal shunt hemodynamics, independently of antifungal treatment.

This report has several limitations. As a single case, it cannot establish causal relationships, define risk factors for SFP in noncirrhotic portal hypertension, or demonstrate the efficacy of a particular antifungal regimen. Although repeated isolation of *P. norvegensis* supports a clinically significant infection, a secondary intra-abdominal source could not be definitively excluded. Species identification was based on MALDI-TOF MS without documented independent molecular confirmation, and antifungal susceptibility interpretation was constrained by the absence of validated species-specific clinical breakpoints. Despite these limitations, the case emphasizes the importance of considering fungal infection in patients with neutrocytic ascites and negative bacterial cultures, obtaining reliable species-level identification, and integrating microbiological findings with the broader clinical assessment.

Conclusions

This case highlights the diagnostic challenges of SFP caused by the uncommon organism *P. norvegensis* in a patient with Budd-Chiari syndrome and severe noncirrhotic portal hypertension. It suggests that fungal infection should be considered in patients with neutrocytic ascites, particularly when bacterial cultures are negative, even in the absence of histologically established cirrhosis. Repeated isolation of *P. norvegensis* from ascitic fluid supported its clinical significance, although a secondary intra-abdominal source could not be definitively excluded. The favorable clinical and microbiological evolution observed during micafungin treatment does not establish antifungal efficacy or susceptibility in the absence of validated species-specific clinical breakpoints. This case emphasizes the importance of reliable species-level identification, cautious interpretation of antifungal susceptibility testing, and individualized clinical assessment.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Tiago Jorge Costa, Diana M. Ferreira, Joana C. Costa

Acquisition, analysis, or interpretation of data: Tiago Jorge Costa, Bernardo Belchior, Diana M. Ferreira, Joana C. Costa, Arsénio Santos

Drafting of the manuscript: Tiago Jorge Costa, Joana C. Costa

Critical review of the manuscript for important intellectual content: Tiago Jorge Costa, Bernardo Belchior, Diana M. Ferreira, Joana C. Costa, Arsénio Santos

Supervision: Diana M. Ferreira, Joana C. Costa

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Tariq T, Irfan FB, Farishta M, Dykstra B, Sieloff EM, Desai AP: Spontaneous fungal peritonitis: micro-organisms, management and mortality in liver cirrhosis - a systematic review. *World J Hepatol*. 2019, 11:596-606. [10.4254/wjh.v11.i7.596](https://doi.org/10.4254/wjh.v11.i7.596)
2. Fiore M, Leone S: Spontaneous fungal peritonitis: epidemiology, current evidence and future prospective. *World J Gastroenterol*. 2016, 22:7742-7. [10.3748/wjg.v22.i34.7742](https://doi.org/10.3748/wjg.v22.i34.7742)
3. Bremmer DN, Garavaglia JM, Shields RK: Spontaneous fungal peritonitis: a devastating complication of cirrhosis. *Mycoses*. 2015, 58:387-93. [10.1111/myc.12321](https://doi.org/10.1111/myc.12321)
4. Pérez-Hansen A, Lass-Flörl C, Lackner M: Antifungal susceptibility profiles of rare ascomycetous yeasts. *J Antimicrob Chemother*. 2019, 74:2649-56. [10.1093/jac/dkz231](https://doi.org/10.1093/jac/dkz231)
5. Sandven P, Nilsen K, Digranes A, Tjade T, Lassen J: *Candida norvegensis*: a fluconazole-resistant species. *Antimicrob Agents Chemother*. 1997, 41:1375-6. [10.1128/AAC.41.6.1375](https://doi.org/10.1128/AAC.41.6.1375)
6. Li B, Yang C, Qian Z, Huang Y, Wang X, Zhong G, Chen J: Spontaneous fungal ascites infection in patients with cirrhosis: an analysis of 10 cases. *Infect Dis Ther*. 2021, 10:1033-43. [10.1007/s40121-021-00422-w](https://doi.org/10.1007/s40121-021-00422-w)
7. Hwang SY, Yu SJ, Lee JH, et al.: Spontaneous fungal peritonitis: a severe complication in patients with advanced liver cirrhosis. *Eur J Clin Microbiol Infect Dis*. 2014, 33:259-64. [10.1007/s10096-013-1953-2](https://doi.org/10.1007/s10096-013-1953-2)
8. Guitard J, Angoulvant A, Letscher-Bru V, et al.: Invasive infections due to *Candida norvegensis* and *Candida*

inconspicua: report of 12 cases and review of the literature. *Med Mycol.* 2013, 51:795-9.
[10.3109/13693786.2013.807444](https://doi.org/10.3109/13693786.2013.807444)

9. Nielsen H, Stenderup J, Bruun B, Ladefoged J: *Candida norvegensis* peritonitis and invasive disease in a patient on continuous ambulatory peritoneal dialysis. *J Clin Microbiol.* 1990, 28:1664-5.
[10.1128/jcm.28.7.1664-1665.1990](https://doi.org/10.1128/jcm.28.7.1664-1665.1990)
10. Pappas PG, Kauffman CA, Andes DR, et al.: Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. *Clin Infect Dis.* 2016, 62:e1-50.
[10.1093/cid/civ933](https://doi.org/10.1093/cid/civ933)
11. Cervantes Pérez E, Cervantes Guevara G, Cervantes Pérez G, et al.: Diagnostic utility of the serum-ascites albumin gradient in Mexican patients with ascites related to portal hypertension. *JGH Open.* 2020, 4:838-42.
[10.1002/jgh3.12404](https://doi.org/10.1002/jgh3.12404)