

Primary Cutaneous Cryptococcosis Presenting as Flexor Tendon Sheath Infection in a Patient With Myelodysplastic Syndrome: A Case Report

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Abstract

Primary cutaneous cryptococcosis (PCC) is a rare clinical presentation, usually associated with traumatic inoculation of cryptococcal spores into the skin, and occurs in both immunocompetent and immunocompromised patients. We report a case of an 81-year-old man with a background of myelodysplastic syndrome (MDS), who developed cellulitis with clinical features of a flexor tendon sheath infection of his right middle phalanx following a splinter. He underwent multiple surgical debridements, including eventual amputation of his finger. *Cryptococcus neoformans* was isolated from culture of debrided tissues. In addition to surgical interventions, he was treated with oral itraconazole for two weeks, followed by fluconazole for nine weeks, resulting in a good clinical outcome. He did not develop respiratory or neurological symptoms and remained systemically well throughout. His serum cryptococcal antigen was also negative on two occasions. This case highlights the need to consider locally invasive PCC in aggressive cellulitis that does not respond to antibiotics or surgical debridement, especially in patients with an impaired immune response.

Categories: Pathology, Orthopedics, Infectious Disease

Keywords: cryptococcus neoformans (c. neoformans), cutaneous cryptococcosis, digital amputation, flexor sheath infections, myelodysplastic syndrome (mds), primary cutaneous cryptococcosis

Introduction

Cryptococcus neoformans and *Cryptococcus gattii* are encapsulated environmental yeasts ubiquitous in the environment. They are well documented to be found in bird excreta, decaying plant matter, and soil, and can cause invasive disease primarily in immunocompromised hosts [1]. Inhalation of *C. neoformans* or *C. gattii* airborne spores can cause local respiratory disease, or may be oligo- or asymptomatic. From the lungs, *C. neoformans* or *C. gattii* can disseminate to multiple other tissues, most notably and lethally to the central nervous system, resulting in cryptococcal meningitis [2]. Secondary cutaneous cryptococcosis can also occur in around 15% of cases as a result of dissemination to the skin [3].

Primary cutaneous cryptococcosis (PCC) represents a distinct yet rare clinical entity from secondary disseminated cryptococcosis, arising from direct inoculation of *C. neoformans* or *C. gattii* spores into the cutaneous tissues from contaminated environmental sources such as wood, soil, or bird droppings [4]. Although no universally validated diagnostic criteria exist for PCC, in 2003, Neuville et al. established a set of key diagnostic features, including the presence of a solitary lesion, a *Cryptococcus*-positive skin culture, and the absence of systemic cryptococcosis [4]. In contrast to secondary disseminated cryptococcosis, PCC occurs more frequently in immunocompetent hosts [5].

The presentation and clinical progression of PCC can vary considerably, creating diagnostic challenges on initial assessment. Lesion morphology is polymorphic, presenting as papules, nodules, plaques, ulceration, abscesses, draining sinuses, cellulitis, and necrotic lesions [6]. Lesions most commonly occur at sites where skin is exposed to environmental trauma such as hands, forearms, and the lower extremities, and there is often but not always a history of an inoculating injury [7]. Unusually, PCC can invade deeper tissues but remain locally contained; rarer still is PCC with systemic dissemination, known as primary disseminated cryptococcosis [8].

Myelodysplastic syndromes (MDSs) are clonal disorders of haematopoietic stem cells, resulting in ineffective haematopoiesis, dysplasia in one or more myeloid cell lineages, and peripheral cytopenias [9]. Patients with MDS have an increased susceptibility to infection, which has traditionally been attributed to neutropenia and immunosuppressive therapy; increasing evidence, however, suggests that aberrant innate and adaptive immunity also contributes to impaired host defence, even in the absence of marked cytopenias [10]. Bacterial infections account for the majority of severe infections in MDS; however, the incidence of fungal infections is not well known [11]. The incidence of PCC in patients with MDS has not been established because of the rarity of this presentation.

How to cite this article

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We report a rare case of PCC with rapid progression from a superficial inoculation lesion to aggressive local invasion of the flexor tendon sheath and adjacent soft tissues in a man with transfusion-dependent MDS.

Case Presentation

An 81-year-old man presented to the emergency department with pain, swelling, and blistering of the right middle finger. He was systemically well, with no fever or features of systemic infection. His medical history included red blood cell transfusion-dependent myelodysplastic/myeloproliferative neoplasm with ring sideroblasts and thrombocytosis (MDS/MPN-RS-T), type 2 diabetes mellitus (T2DM) controlled with metformin and saxagliptin, hypertension controlled with ramipril and tadalafil, and actinic keratosis. His haematological disorder was diagnosed two years before the current presentation and was characterised by the absence of excess blasts, normal cytogenetics, and the presence of SF3B1 and TET2 variants on molecular testing. He was classified as having low-risk disease and had been managed with supportive care since diagnosis. He was not receiving any immunosuppressive agents, chemotherapy, or corticosteroid therapy.

The patient reported sustaining a splinter injury to the finger while on holiday in France several days earlier. On returning to the United Kingdom one week later, he noticed a small bruise over the palmar aspect of the right middle phalanx. The following morning, the patient awoke with significant pain and swelling of the finger. He denied any other trauma or injury to the site. Three days later, the patient presented to the hospital, where a review by the orthopaedic team revealed a grossly swollen right third finger with overlying erythema that was hot to the touch. There was associated swelling of the right hand, mainly over the dorsal aspect. Superficial blood-filled blisters were present on both sides of the skin overlying the third middle phalanx, with serous fluid discharging from a small open area. There was pain on flexion of the finger and on palpation of the flexor sheath. His sensation was normal, with a strong radial pulse and normal capillary refill time.

Full blood count (FBC) on admission revealed mild leukocytosis ($13.8 \times 10^9/L$), with a neutrophil count of $12.2 \times 10^9/L$. Haemoglobin was approximately at his expected baseline for myelodysplasia at 76 g/L, and his platelet count was normal. Two sets of blood cultures obtained at admission were negative. Further baseline investigations from admission are presented in Table 1. The patient's creatinine, urea, electrolytes, and liver function tests were all normal. Hand radiography showed marked soft tissue swelling of the right middle finger, mild degenerative changes in the interphalangeal joints, and no evidence of bony destruction. Systemic examination revealed no other abnormalities.

Parameter	Value and units	Normal range
White blood cell count	13.8 x 10 ⁹ /L	3.0-11.0
Haemoglobin	76 g/L	130-166
Red blood cell count	2.23 x 10 ¹² /L	4.25-5.55
Mean cell volume	100.4 fL	80.0-100.0
Haematocrit	0.224 (ratio)	0.390-0.490
Mean cell haemoglobin	34.1 pg	28.1-32.1
Platelets	351 x 10 ⁹ /L	150-400
Neutrophils	12.2 x 10 ⁹ /L	1.8-8
Lymphocytes	0.5 x 10 ⁹ /L	0.6-4.4
Monocytes	1.1 x 10 ⁹ /L	0.0-0.8
Eosinophils	0.0 x 10 ⁹ /L	0.0-0.5
Basophils	0.0 x 10 ⁹ /L	0.0-0.3
Nucleated red blood cells	0.06 x 10 ⁹ /L	0.0-0.10
Serum ferritin	2,684 µg/L	30-400
Serum iron	5 µmol/L	11-30
C-reactive protein	96 mg/L	0-5
HbA1c	41 mmol/mol	20-41
Glucose	10.9 mmol/L	Critical low 2; critical high 30
Blood culture - set 1	No growth after five days of incubation	-
Blood culture - set 2	No growth after five days of incubation	-

TABLE 1: Baseline full blood count, serum ferritin, serum iron, C-reactive protein (CRP), glycosylated haemoglobin (HbA1c), blood glucose, and blood cultures on admission to the hospital.

A differential diagnosis of severe third-finger cellulitis with flexor tendon sheath infection was made. The patient was subsequently started on empiric intravenous antibiotics and taken to the theatre the following morning (day 1) for urgent surgical exploration. Dorsal and volar incisions were made in the finger. On blunt dissection of the flexor tendon sheath, murky 'dishwater' fluid was immediately expressed, with additional superficial fluid released through multiple incisions over the dorsum of the finger. No frank pus or tissue necrosis was evident. The flexor tendon compartment was irrigated; no tissue debridement was required at that stage. Fluid samples were sent for microbiological analysis, with no growth after 48 hours of routine bacterial culture.

On day 4, the patient returned to the theatre due to worsening swelling of the finger. Dusky discolouration of the skin and a wet, necrotic finger extending to the level of the proximal interphalangeal joint (PIPJ) were evident. Copious amounts of pus were released through multiple incisions in the finger, and thrombosis of the digital arteries was noted. The finger was therefore disarticulated through the PIPJ, followed by washout and tissue debridement back to the bleeding point. Tissue samples were again sent for routine bacterial culture and, given the progression to necrosis, were also inoculated onto Sabouraud dextrose agar for fungal culture. Seventy-two hours later, fungal culture of tissue from the amputated finger demonstrated creamy, yeast-like colonies (Figure 1). Fungal culture of tissue from the initial debridement also yielded the same findings. As definitive identification was likely to be delayed due to the slow-growing nature of the fungus, empirical oral itraconazole 200 mg every 12 hours was commenced initially to cover a broader range of fungi, such as *Sporothrix* species. Ongoing inflammation at the amputation site necessitated further debridement and bone shortening on day 7. The patient remained systemically well throughout and was discharged home on day 8 while awaiting further culture results.



FIGURE 1: Yeast-like colonies cultured from the patient's finger tissue following inoculation onto Sabouraud dextrose agar.

The colonies are smooth and uniform, with a creamy, milk-like colour.

The yeast-like colonies cultured from tissue obtained during both debridement and right finger amputation were provisionally identified as *C. neoformans* using the VITEK® MS system (bioMérieux SA, Marcy-l'Étoile, France). The patient was therefore recalled to the hospital for further review and investigations. Clinical review found no evidence of respiratory or central nervous system involvement. The patient was alert, with a normal neurological examination. No headache or fever was present. He denied any respiratory symptoms, and auscultation of his chest was normal. Chest X-ray was also normal. HIV-1/2 antibodies and p24 antigen were not detected. Serum cryptococcal antigen (CrAg) testing was performed to assess for disseminated infection and was subsequently confirmed to be negative. As the patient was systemically well, with no features of neurological dissemination and a negative serum CrAg result, a lumbar puncture was not performed. Histological analysis of the patient's finger tissue subsequently revealed numerous spores and narrow-based budding (Figures 2–4).

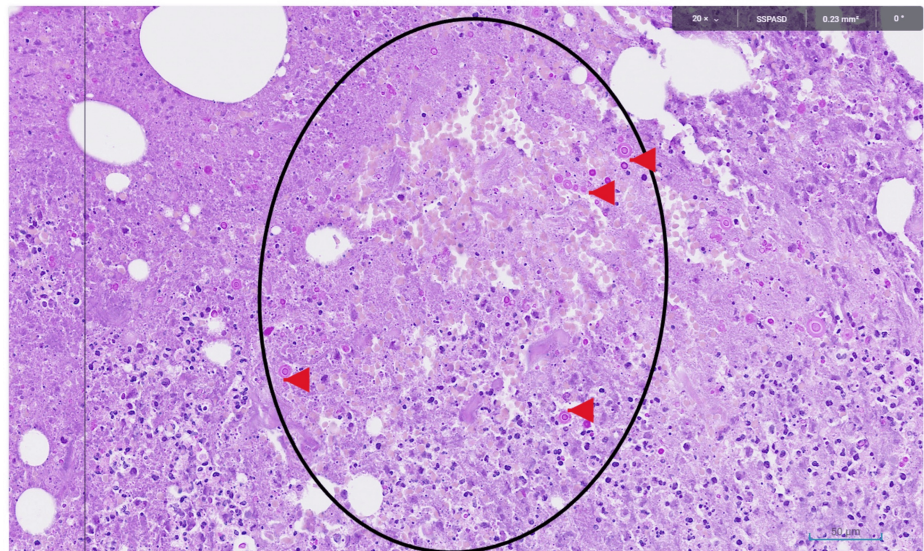


FIGURE 2: Periodic acid-Schiff diastase stain of right finger tissue.

Magnification: 20×, 0.23 mm². The skin is inflamed, with a background of necrotic debris and an inflammatory infiltrate. Multinucleated histiocytes are present. Scant, variably sized, round-to-oval yeast forms are present within the inflamed and necrotic tissue, exhibiting cell walls and subtle narrow-based budding; the field of interest is indicated by the black circle. Numerous yeast forms with surrounding ring-like capsules are demonstrated (red arrows). Scale bar: 50 μm (bottom-right corner).

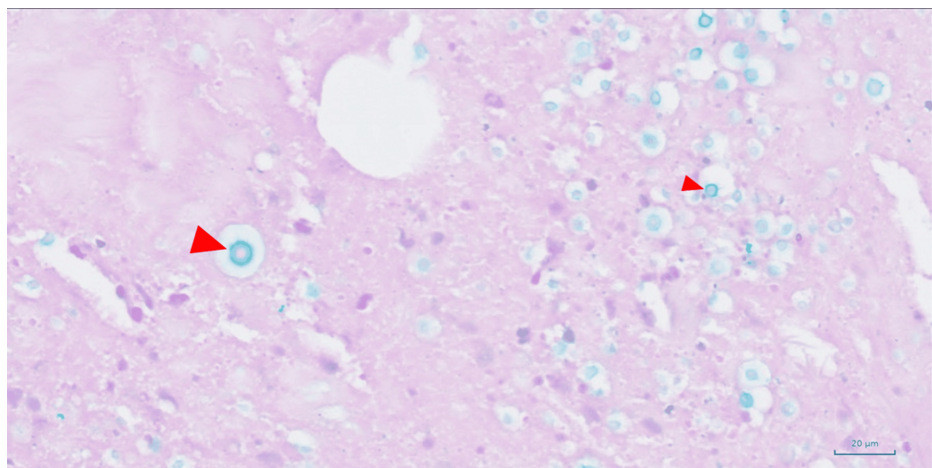


FIGURE 3: Alcian blue stain of right finger tissue.

Magnification: 60×, 0.050 mm². Yeast forms with mucoid capsules are demonstrated in blue (red arrows). Scale bar: 20 μm (bottom-right corner).

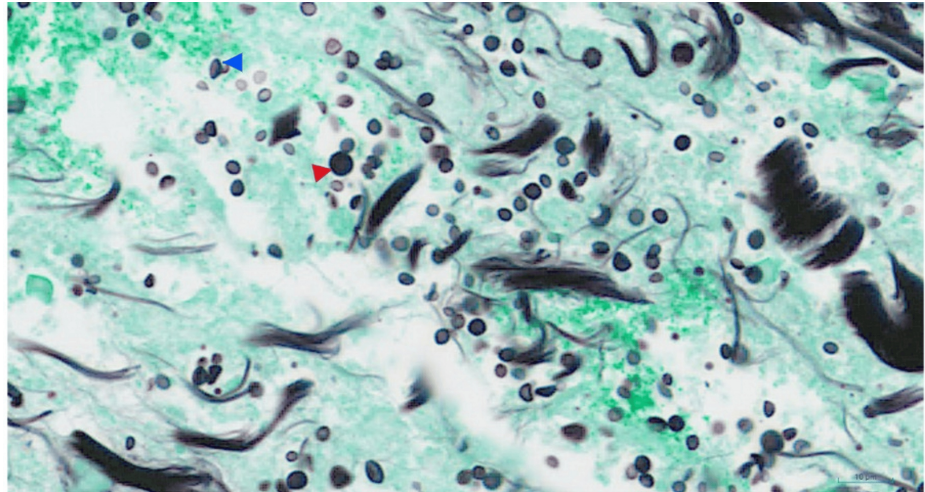


FIGURE 4: Grocott silver stain of right finger tissue.

Magnification: 110 $\times$, 0.015 mm². Numerous round and oval-shaped yeast forms are shown with thick outer capsules (blue arrow) and narrow-based budding (red arrow). Scale bar: 10 μ m (bottom-right corner).

Identification and sensitivity testing performed at the Bristol mycology reference laboratory confirmed the yeast to be *C. neoformans*. Testing was performed on the following agents, with minimum inhibitory concentrations (MICs) all deemed to be sensitive as per the reference laboratory: amphotericin (MIC 0.5 μ g/mL), voriconazole (MIC <0.03 μ g/mL), and fluconazole (MIC <2 μ g/mL). Following discussion with a medical mycologist, antifungal therapy was switched to oral fluconazole 800 mg daily due to the generally unpredictable serum concentration of itraconazole. The decision to proceed with a higher dose of fluconazole than normally recommended for PCC was made due to the aggressive nature of the presentation, requiring multiple surgical debridements.

Four weeks after his initial presentation, the patient unfortunately required a third surgical debridement due to poor wound healing. Repeat tissue samples cultured *C. neoformans*. At the six-week outpatient review, after 17 days of targeted fluconazole therapy, further minor debridement in clinic was required. No further samples were sent for culture at this time, and serum CrAg remained negative. The patient remained clinically well throughout, with no evidence of systemic dissemination. The fluconazole dose was halved from 800 to 400 mg daily after seven weeks of treatment and continued for a further two weeks. Fluconazole therapy was stopped after nine weeks due to intolerable gastrointestinal side effects and evidence of clinical resolution.

At the 12-week review, the wound had healed with scarring and no signs of active infection. The patient completed a total of two weeks of empirical oral itraconazole therapy followed by nine weeks of targeted oral fluconazole therapy. He remained well six months after treatment completion, with no reported wound breakdown or systemic upset. A timeline of the patient's clinical course, investigations, and treatment is shown in Figure 5.

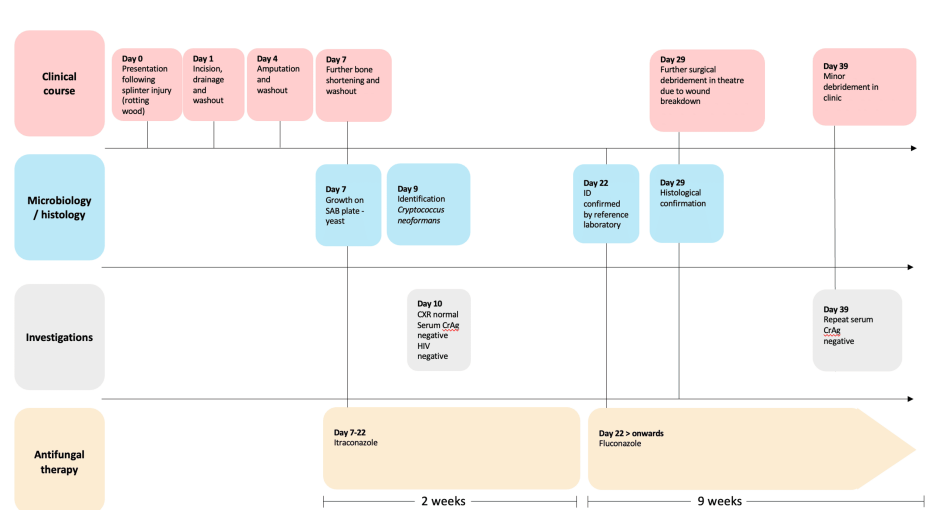


FIGURE 5: Clinical course and management of primary cutaneous cryptococcosis in a patient with myelodysplastic syndrome.

Clinical course (red panel), microbiology/histology (blue panel), other relevant investigations (grey panel), and empirical (itraconazole) and definitive (fluconazole) antifungal therapy (yellow panel) are displayed.

CrAg, cryptococcal antigen; CXR, chest X-ray; HIV, human immunodeficiency virus; SAB, Sabouraud agar

Discussion

This case represents an unusually aggressive presentation of PCC, with rapid extension from a presumed inoculation site into the flexor tendon sheath and adjacent tissues. Our patient's presentation aligns with the principal features of PCC described by Neuville et al.: *C. neoformans* was cultured from serial tissue samples, encapsulated budding yeasts were demonstrated on histological analysis, serum cryptococcal antigen remained negative, and no features of pulmonary or central nervous system disease were present throughout the clinical course [4]. Additionally, the patient reported sustaining a splinter injury after handling rotting wood shortly before the lesion developed, providing a plausible route of acquisition. Decaying wood and plant material are well-recognised environmental reservoirs of *C. neoformans*, supporting the theory of a direct inoculation event [12]. These features all favour a diagnosis of true primary disease rather than secondary disseminated cryptococcosis.

There is no single diagnostic criterion with established sensitivity and specificity for reliably distinguishing PCC from secondary cutaneous cryptococcosis, with diagnosis relying on a combination of clinical history, lesion characteristics, microbiological findings, and investigations to exclude systemic dissemination [4]. A history of preceding trauma or direct inoculation, particularly when followed by a localised lesion at the site of injury, supports a diagnosis of PCC. Marques et al. reported a sensitivity of 95% and specificity of 98% for cryptococcal antigen detection by latex agglutination and suggested that negative testing in at least serum and cerebrospinal fluid (CSF) supports the absence of systemic involvement with 98% certainty [7]. However, these figures relate to cryptococcal antigen detection rather than a validated diagnostic tool for distinguishing PCC from secondary cutaneous cryptococcosis. In our case, the history of penetrating trauma, localised infection, negative blood cultures, repeatedly negative serum cryptococcal antigen tests, and absence of systemic symptoms strongly supported PCC. Nevertheless, we acknowledge that lumbar puncture was not performed to obtain CSF for cryptococcal antigen testing and that chest CT imaging was not undertaken, limiting our ability to definitively exclude occult dissemination.

PCC has a markedly heterogeneous presentation according to the literature. Reported manifestations include, but are not limited to, papules, nodules, plaques, blistering, ulceration, abscesses, granulomas, draining sinuses, cellulitis, and necrotising soft tissue infection [6]. Its clinical course is similarly variable, ranging from acute or subacute onset to lesions reported several months after a proposed environmental trauma [7,13]. The presentation described in our case began as a small bruise-like lesion, rapidly progressing over a few hours to a severe cellulitis picture with flexor tendon sheath infection. Within 72 hours, operative findings demonstrated tissue necrosis of the finger, necessitating amputation. Continued local infection required repeated debridement despite initiation of antifungal therapy. This rapid tissue destruction contrasts with the more nodular, plaque-like, and ulcerative lesions that are more commonly described in PCC in immunocompetent individuals [5].

Cellulitis is a recognised but relatively uncommon presentation of PCC. Almatroud et al. reviewed 22

reported cases of PCC in immunocompetent patients and found that only a minority presented with cellulitic features or required surgical intervention [5]. Inoue et al. described cutaneous cryptococcosis in a patient with MDS; interestingly, this case also followed an aggressive course requiring tissue debridement [14]. Although no pulmonary or neurological involvement was evident in their case, multiple factors made it difficult to rule out secondary cutaneous infection following asymptomatic pulmonary inhalation, namely, an exceptionally high cryptococcal antigen titer, the absence of an inoculation event, and the location of the lesion on the chest wall, a site unlikely to have been exposed to environmental trauma. Shalom and Horev also reported a case of PCC in a patient with MDS. They described a cellulitis-like macule adjacent to a deeper ulcer resembling pyoderma gangrenosum, located at erythropoietin derivative injection sites [15]. No systemic dissemination was evident in this case, and the patient improved with antifungal therapy alone, without the need for tissue debridement [15]. These case reports may suggest that immune dysfunction in MDS could contribute to a more aggressive inflammatory presentation of cellulitis in PCC, rather than slower-progressing granular or nodular lesions. However, the heterogeneity of PCC presentation and the very small number of reported cases in patients with MDS preclude any firm association between MDS and this PCC phenotype. Further evidence and more comprehensive reviews comparing features of PCC in immunocompetent versus immunosuppressed hosts are required to explore this further.

Extension beyond the dermis and subcutaneous tissues while remaining locally confined has been reported in PCC but appears rare. Mason and Keith described chronic suppurative cryptococcal extensor tenosynovitis in a patient with Castleman disease; the lesion in this case formed a necropurulent mass possibly arising from the extensor tendon sheath [16]. However, because the patient had no history of inoculating trauma or evidence of infection at a distant site, a diagnosis of localised cryptococcal tenosynovitis was made rather than confirmed PCC. In contrast, our case provides a rare illustration of rapidly progressing, contiguous extension of PCC to deeper tissue structures, with clinical and intraoperative findings consistent with flexor tendon sheath involvement. Although *C. neoformans* was isolated from the affected finger tissue, the specimens submitted for microbiological and histopathological analysis were not specifically identified as originating from the flexor tendon sheath; thus, direct microbiological or histopathological confirmation of tendon sheath infection was not established. Despite this, no other organisms were identified, and confirmation of *C. neoformans* in adjacent tissue, together with clinical and surgical evidence of flexor tendon sheath infection, supports the diagnosis of cryptococcal tenosynovitis. Additionally, the degree of extension and tissue necrosis warranted extensive debridement and disarticulation of the third finger at the proximal interphalangeal joint. Infection of the bone could not be confirmed because no bone samples were submitted for microbiological or histological analysis, but it is plausible given the rapid destruction of surrounding tissues.

Invasive cryptococcosis is generally considered an opportunistic infection in those with advanced immunosuppression. In contrast, PCC occurs more frequently in immunocompetent individuals [5]. Although our patient had transfusion-dependent MDS, he was not receiving corticosteroids, cytotoxic chemotherapy, or other immunosuppressive therapy and had normal peripheral white cell counts before presentation. While infection susceptibility in MDS has traditionally been associated with neutropenia, more recent evidence suggests that the complex interplay of abnormally active innate and adaptive immune responses, dysregulation of T-cell, B-cell, natural killer cell, and dendritic cell populations, and overexpression of inflammatory cytokines such as TNF-alpha all contribute to increased infection risk [10]. Functional granulocyte impairment has been described; thus, a preserved circulating neutrophil count does not necessarily imply normal granulocyte function [17]. Indeed, our patient had MDS with suspected trilineage dysplasia, which may have resulted in peripheral neutrophil dysfunction despite normal neutrophil counts at baseline. However, as neutrophil function and cellular immunity were not specifically assessed in our patient, the contribution of the underlying MDS to the severity of infection in this case remains speculative.

Finally, due to the rarity of the condition, the optimal treatment of PCC is guided largely by case series, reports, and expert recommendations. Current global guidance recommends fluconazole 400 mg daily for three to six months as a first-line antifungal agent for PCC without systemic dissemination [18]. In this case, growth on the fungal culture plates was initially faint, with Gram stain demonstrating yeast-like organisms after 72 hours. As definitive identification was likely to be delayed due to the slow-growing nature of the fungus, itraconazole was initially chosen for broader antifungal coverage. Although a recent review suggests itraconazole as an appropriate alternative therapy for PCC, the initial two-week course was considered empirical and was therefore excluded from the duration of definitive antifungal treatment [5]. While itraconazole achieves high concentrations in the epidermis and other keratinised tissues, its oral absorption and serum concentrations can be variable [19]. Treatment was subsequently changed to fluconazole following confirmation of *C. neoformans* and susceptibility testing, providing more predictable systemic exposure. Potential drug interactions should be an important consideration when choosing appropriate antifungal therapy for PCC. Of particular relevance to this case, itraconazole is a more potent inhibitor of CYP3A4 than fluconazole and may therefore increase systemic exposure to concomitant medications, including tadalafil and saxagliptin. However, the clinical significance of these interactions should be assessed on a case-by-case basis, with specialist pharmacy input where appropriate.

While the subsequent course of fluconazole was shorter than initially intended, treatment discontinuation was considered appropriate given that the wound had fully healed and the patient was experiencing

intolerable adverse effects despite dose reduction. Indeed, current global guidelines recommend fluconazole for three to six months, or until the wound is fully healed. We therefore considered the duration of definitive fluconazole treatment reasonable in the context of complete clinical resolution and treatment-limiting adverse effects. Successful outcomes following treatment courses shorter than three months have also been reported in the literature; however, evidence for this remains limited to individual case reports [15,20]. Although our patient achieved a successful outcome with a shorter-than-recommended course of fluconazole, antifungal treatment alone would probably have been insufficient in the presence of necrotic tissue, vascular thrombosis, and tendon sheath involvement. The successful outcome in this case supports a combined medical and surgical approach when PCC results in extensive necrosis or invades deeper local compartments. Treatment for PCC should therefore be based on current guidelines, with individualisation of treatment duration according to clinical response and tolerability.

Conclusions

In conclusion, although rare, PCC should be considered in progressive cellulitis or flexor sheath infection that fails to respond to conventional antibacterial therapy, particularly following environmental trauma. It can cause rapid, destructive extension through contiguous soft tissues without pulmonary, neurological, or haematogenous dissemination. Although we found no clinical or laboratory evidence of disseminated cryptococcosis in our case, the lack of lumbar puncture and chest CT imaging limits our ability to definitively exclude occult dissemination, particularly in the context of underlying MDS.

Another important limitation of this case report is its limited generalisability. Our case represents a single patient with a specific underlying haematological disorder; the findings cannot necessarily be extrapolated to the wider population of patients with MDS, other haematological malignancies, or other immunocompromised states. Furthermore, the degree and nature of immune dysfunction in MDS are heterogeneous and may vary according to disease subtype, severity, and treatment. Although it is biologically plausible that the underlying MDS contributed to the aggressive clinical course, no specific investigations of neutrophil or cellular immune function were performed. Consequently, a causal relationship between the patient's haematological disorder and the severity of infection cannot be established. Other patient-specific factors, including the site and mechanism of inoculation, extent of tissue involvement, and timing of presentation and diagnosis, may also have influenced the clinical course. The successful outcome following combined surgical and antifungal management cannot establish the relative contribution of either intervention in this case or determine the optimal treatment approach for other patients. Nevertheless, this case highlights several potentially generalisable practice points, including the importance of early deep-tissue sampling for fungal culture and histopathology, assessment for disseminated infection, and timely multidisciplinary management. Combined antifungal and surgical treatment may be appropriate in selected patients with extensive or progressive local disease. The role of early surgical assessment in immunocompromised patients with PCC may warrant further consideration in future clinical guidance, particularly where deep tissue involvement is suspected.

Other preventive measures should also be considered. Although complete avoidance of exposure to *Cryptococcus* species is impractical owing to their ubiquitous nature in the environment, simple protective measures may help reduce the risk of infection, such as wearing appropriate protective clothing when gardening, handling decaying vegetation, or handling materials likely to be contaminated with bird excreta. Patients with MDS and other conditions associated with impaired host immunity may benefit from advice regarding these precautions, particularly when undertaking activities that increase the risk of skin injury or environmental fungal exposure. Such measures may also reduce exposure to other environmental fungal pathogens.

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: Julia Gleeson, Kavya Mohandas

Acquisition, analysis, or interpretation of data: Julia Gleeson, Krishna Gumparthy, Meher Khan

Drafting of the manuscript: Julia Gleeson, Meher Khan

Critical review of the manuscript for important intellectual content: Julia Gleeson, Kavya Mohandas, Krishna Gumparthy

Supervision: Kavya Mohandas, Meher Khan

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. N/A issued approval N/A. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** Case was presented by Julia Gleeson at the Fungal Update Mycology Conference in London (Royal College of Physicians), March 2026. Funding was provided for travel, hotel stay and conference fees. No funding was provided for preparation of any part of the design or writing of the manuscript. **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.

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