

# World Journal of *Clinical Cases*

*World J Clin Cases* 2021 August 6; 9(22): 6178-6581



# REVIEW

- 6178 COVID-19 infection and liver injury: Clinical features, biomarkers, potential mechanisms, treatment, and management challenges

*Sivandzadeh GR, Askari H, Safarpour AR, Ejtehad F, Raeis-Abdollahi E, Vaez Lari A, Abazari MF, Tarkesh F, Bagheri Lankarani K*

- 6201 Gastrointestinal manifestations of systemic sclerosis: An updated review

*Luquez-Mindiola A, Atuesta AJ, Gómez-Aldana AJ*

# MINIREVIEWS

- 6218 Mesenchymal stem cell-derived exosomes: An emerging therapeutic strategy for normal and chronic wound healing

*Zeng QL, Liu DW*

- 6234 Role of autophagy in cholangiocarcinoma: Pathophysiology and implications for therapy

*Ninfolle E, Pinto C, Benedetti A, Marziani M, Maroni L*

# ORIGINAL ARTICLE

## Case Control Study

- 6244 Risk factors for intussusception in children with Henoch-Schönlein purpura: A case-control study

*Zhao Q, Yang Y, He SW, Wang XT, Liu C*

## Retrospective Study

- 6254 Sequential therapy with combined trans-papillary endoscopic naso-pancreatic and endoscopic retrograde pancreatic drainage for pancreatic pseudocysts

*He YG, Li J, Peng XH, Wu J, Xie MX, Tang YC, Zheng L, Huang XB*

- 6268 Retrospective study of effect of whole-body vibration training on balance and walking function in stroke patients

*Xie L, Yi SX, Peng QF, Liu P, Jiang H*

- 6278 Risk factors for preoperative carcinogenesis of bile duct cysts in adults

*Wu X, Li BL, Zheng CJ, He XD*

- 6287 Diagnostic and prognostic value of secreted protein acidic and rich in cysteine in the diffuse large B-cell lymphoma

*Pan PJ, Liu JX*

- 6300 Jumbo cup in hip joint renovation may cause the center of rotation to increase

*Peng YW, Shen JM, Zhang YC, Sun JY, Du YQ, Zhou YG*

**Clinical Trials Study**

- 6308** Effect of exercise training on left ventricular remodeling in patients with myocardial infarction and possible mechanisms  
*Cai M, Wang L, Ren YL*

**Observational Study**

- 6319** Analysis of sleep characteristics and clinical outcomes of 139 adult patients with infective endocarditis after surgery  
*Hu XM, Lin CD, Huang DY, Li XM, Lu F, Wei WT, Yu ZH, Liao HS, Huang F, Huang XZ, Jia FJ*
- 6329** Health-related risky behaviors and their risk factors in adolescents with high-functioning autism  
*Sun YJ, Xu LZ, Ma ZH, Yang YL, Yin TN, Gong XY, Gao ZL, Liu YL, Liu J*
- 6343** Selection of internal fixation method for femoral intertrochanteric fractures using a finite element method  
*Mu JX, Xiang SY, Ma QY, Gu HL*

**META-ANALYSIS**

- 6357** Neoadjuvant chemotherapy for patients with resectable colorectal cancer liver metastases: A systematic review and meta-analysis  
*Zhang Y, Ge L, Weng J, Tuo WY, Liu B, Ma SX, Yang KH, Cai H*

**CASE REPORT**

- 6380** Ruptured intracranial aneurysm presenting as cerebral circulation insufficiency: A case report  
*Zhao L, Zhao SQ, Tang XP*
- 6388** Prostatic carcinosarcoma seven years after radical prostatectomy and hormonal therapy for prostatic adenocarcinoma: A case report  
*Huang X, Cai SL, Xie LP*
- 6393** Pyogenic arthritis, pyoderma gangrenosum, and acne syndrome in a Chinese family: A case report and review of literature  
*Lu LY, Tang XY, Luo GJ, Tang MJ, Liu Y, Yu XJ*
- 6403** Malaria-associated secondary hemophagocytic lympho-histiocytosis: A case report  
*Zhou X, Duan ML*
- 6410** Ileal hemorrhagic infarction after carotid artery stenting: A case report and review of the literature  
*Xu XY, Shen W, Li G, Wang XF, Xu Y*
- 6418** Inflammatory myofibroblastic tumor of the pancreatic neck: A case report and review of literature  
*Chen ZT, Lin YX, Li MX, Zhang T, Wan DL, Lin SZ*
- 6428** Management of heterotopic cesarean scar pregnancy with preservation of intrauterine pregnancy: A case report  
*Chen ZY, Zhou Y, Qian Y, Luo JM, Huang XF, Zhang XM*

- 6435** Manifestation of severe pneumonia in anti-PL-7 antisynthetase syndrome and B cell lymphoma: A case report  
*Xu XL, Zhang RH, Wang YH, Zhou JY*
- 6443** Disseminated infection by *Fusarium solani* in acute lymphocytic leukemia: A case report  
*Yao YF, Feng J, Liu J, Chen CF, Yu B, Hu XP*
- 6450** Primary hepatic neuroendocrine tumor – <sup>18</sup>F-fluorodeoxyglucose positron emission tomography/computed tomography findings: A case report  
*Rao YY, Zhang HJ, Wang XJ, Li MF*
- 6457** Malignant peripheral nerve sheath tumor in an elderly patient with superficial spreading melanoma: A case report  
*Yang CM, Li JM, Wang R, Lu LG*
- 6464** False positive anti-hepatitis A virus immunoglobulin M in autoimmune hepatitis/primary biliary cholangitis overlap syndrome: A case report  
*Yan J, He YS, Song Y, Chen XY, Liu HB, Rao CY*
- 6469** Successful totally laparoscopic right trihepatectomy following conversion therapy for hepatocellular carcinoma: A case report  
*Zhang JJ, Wang ZX, Niu JX, Zhang M, An N, Li PF, Zheng WH*
- 6478** Primary small cell esophageal carcinoma, chemotherapy sequential immunotherapy: A case report  
*Wu YH, Zhang K, Chen HG, Wu WB, Li XJ, Zhang J*
- 6485** Subdural fluid collection rather than meningitis contributes to hydrocephalus after cervical laminoplasty: A case report  
*Huang HH, Cheng ZH, Ding BZ, Zhao J, Zhao CQ*
- 6493** Phlegmonous gastritis developed during chemotherapy for acute lymphocytic leukemia: A case report  
*Saito M, Morioka M, Izumiyama K, Mori A, Ogasawara R, Kondo T, Miyajima T, Yokoyama E, Tanikawa S*
- 6501** Spinal epidural hematoma after spinal manipulation therapy: Report of three cases and a literature review  
*Liu H, Zhang T, Qu T, Yang CW, Li SK*
- 6510** Abdominal hemorrhage after peritoneal dialysis catheter insertion: A rare cause of luteal rupture: A case report  
*Gan LW, Li QC, Yu ZL, Zhang LL, Liu Q, Li Y, Ou ST*
- 6515** Concealed mesenteric ischemia after total knee arthroplasty: A case report  
*Zhang SY, He BJ, Xu HH, Xiao MM, Zhang JJ, Tong PJ, Mao Q*
- 6522** Chylothorax following posterior low lumbar fusion surgery: A case report  
*Huang XM, Luo M, Ran LY, You XH, Wu DW, Huang SS, Gong Q*
- 6531** Non-immune hydrops fetalis: Two case reports  
*Maranto M, Cigna V, Orlandi E, Cucinella G, Lo Verso C, Duca V, Picciotto F*



- 6538** Bystander effect and abscopal effect in recurrent thymic carcinoma treated with carbon-ion radiation therapy: A case report  
*Zhang YS, Zhang YH, Li XJ, Hu TC, Chen WZ, Pan X, Chai HY, Ye YC*
- 6544** Management of an intracranial hypotension patient with diplopia as the primary symptom: A case report  
*Wei TT, Huang H, Chen G, He FF*
- 6552** Spontaneous rupture of adrenal myelolipoma as a cause of acute flank pain: A case report  
*Kim DS, Lee JW, Lee SH*
- 6557** Neonatal necrotizing enterocolitis caused by umbilical arterial catheter-associated abdominal aortic embolism: A case report  
*Huang X, Hu YL, Zhao Y, Chen Q, Li YX*
- 6566** Primary mucosa-associated lymphoid tissue lymphoma in the midbrain: A case report  
*Zhao YR, Hu RH, Wu R, Xu JK*
- 6575** Extensive cutaneous metastasis of recurrent gastric cancer: A case report  
*Chen JW, Zheng LZ, Xu DH, Lin W*

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The WJCC is now indexed in Science Citation Index Expanded (also known as SciSearch®), Journal Citation Reports/Science Edition, Scopus, PubMed, and PubMed Central. The 2021 Edition of Journal Citation Reports® cites the 2020 impact factor (IF) for WJCC as 1.337; IF without journal self cites: 1.301; 5-year IF: 1.742; Journal Citation Indicator: 0.33; Ranking: 119 among 169 journals in medicine, general and internal; and Quartile category: Q3. The WJCC's CiteScore for 2020 is 0.8 and Scopus CiteScore rank 2020: General Medicine is 493/793.

**RESPONSIBLE EDITORS FOR THIS ISSUE**

**Production Editor:** Yan-Xia Xing; **Production Department Director:** Yun-Jie Ma; **Editorial Office Director:** Jin-Lei Wang.

**NAME OF JOURNAL**

*World Journal of Clinical Cases*

**ISSN**

ISSN 2307-8960 (online)

**LAUNCH DATE**

April 16, 2013

**FREQUENCY**

Thrice Monthly

**EDITORS-IN-CHIEF**

Dennis A Bloomfield, Sandro Vento, Bao-Gan Peng

**EDITORIAL BOARD MEMBERS**

<https://www.wjgnet.com/2307-8960/editorialboard.htm>

**PUBLICATION DATE**

August 6, 2021

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<https://www.wjgnet.com/bpg/GerInfo/288>

**PUBLICATION MISCONDUCT**

<https://www.wjgnet.com/bpg/gerinfo/208>

**ARTICLE PROCESSING CHARGE**

<https://www.wjgnet.com/bpg/gerinfo/242>

**STEPS FOR SUBMITTING MANUSCRIPTS**

<https://www.wjgnet.com/bpg/GerInfo/239>

**ONLINE SUBMISSION**

<https://www.f6publishing.com>

## Disseminated infection by *Fusarium solani* in acute lymphocytic leukemia: A case report

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**Author contributions:** Yao YF and Feng J contributed equally to this work and should be considered joint first authors; Hu XP reviewed the data and coordinated the authors; Yao YF and Feng J wrote the paper and extracted the data; Liu J, Chen CF, and Yu B revised the edited the article.

**Supported by** the Scientific Research Project of Peking University Shenzhen Hospital, No. JCYJ2018011; and the San-Ming Project of Medicine in Shenzhen, No. SZSM201812059.

**Informed consent statement:** Informed written consent was obtained from the patient for publication of this report and any accompanying images.

**Conflict-of-interest statement:** The authors declare that they have no conflict of interest.

**CARE Checklist (2016) statement:** The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE

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### Abstract

#### BACKGROUND

In recent years, the rate of immunosuppressed patients has increased rapidly. Invasive fungal infections usually occur in these patients, especially those who have had hematological malignancies and received chemotherapy. Fusariosis is a rare pathogenic fungus, it can lead to severely invasive *Fusarium* infections. Along with the increased rate of immune compromised patients, the incidence of invasive *Fusarium* infections has also increased from the past few years. Early diagnosis and therapy are important to prevent further development to a more aggressive or disseminated infection.

#### CASE SUMMARY

We report a case of a 19-year-old male acute B-lymphocytic leukemia patient with fungal infection in the skin, eyeball, and knee joint during the course of chemotherapy. We performed skin biopsy, microbial cultivation, and molecular biological identification, and the pathogenic fungus was finally confirmed to be *Fusarium solani*. The patient was treated with oral 200 mg voriconazole twice daily intravenous administration of 100 mg liposomal amphotericin B once daily, and surgical debridement. Granulocyte colony-stimulating factor was administered to expedite neutrophil recovery. The disseminated *Fusarium solani* infection eventually resolved, and there was no recurrence at the 3 mo follow-up.

#### CONCLUSION

Our case illustrates the early detection and successful intervention of a systemic invasive *Fusarium* infection. These are important to prevent progression to a more aggressive infection. Disseminate *Fusarium* infection requires the systemic use of antifungal agents and immunotherapy. Localized infection likely benefits from surgical debridement and the use of topical antifungal agents.

Checklist (2016).

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**Manuscript source:** Unsolicited manuscript

**Specialty type:** Dermatology

**Country/Territory of origin:** China

**Peer-review report's scientific quality classification**

Grade A (Excellent): 0  
Grade B (Very good): B, B, B  
Grade C (Good): 0  
Grade D (Fair): 0  
Grade E (Poor): 0

**Received:** March 9, 2021

**Peer-review started:** March 9, 2021

**First decision:** April 24, 2021

**Revised:** May 7, 2021

**Accepted:** May 25, 2021

**Article in press:** May 25, 2021

**Published online:** August 6, 2021

**P-Reviewer:** Jameel PZ, Nwabo  
Kamdje AH

**S-Editor:** Zhang H

**L-Editor:** Filipodia

**P-Editor:** Xing YX



**Key Words:** Acute lymphocytic leukemia; Invasive fungal infection; *Fusarium sp.*; Neutropenia; Skin lesions; Case report

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**Core Tip:** *Fusarium* as a rare pathogenic fungus can lead to severely invasive fusariosis and is associated with high morbidity and with up to 70% mortality. The clinical manifestations of invasive *Fusarium* infection are varied; early diagnosis and proper therapies are essential. In such infections, the identification of fungal etiology is very important. Histopathological examination, microbial cultivation, antifungal susceptibility testing, and molecular biological identification are helpful for the diagnosis and treatment of this disease. In this case, the diagnosis was clear and the patient was successfully treated with positive efforts.

**Citation:** Yao YF, Feng J, Liu J, Chen CF, Yu B, Hu XP. Disseminated infection by *Fusarium solani* in acute lymphocytic leukemia: A case report. *World J Clin Cases* 2021; 9(22): 6443-6449

**URL:** <https://www.wjgnet.com/2307-8960/full/v9/i22/6443.htm>

**DOI:** <https://dx.doi.org/10.12998/wjcc.v9.i22.6443>

## INTRODUCTION

Fusariosis is the second common mold infection after aspergillosis[1]. *Fusarium* species are important plant pathogens causing a broad spectrum of infections, including superficial infections (keratitis and onychomycosis) and invasive or disseminated infections[2]. Agents of any type of fusariosis are mainly found in the following three species complexes: *Fusarium solani* (*F. solani*) complex (FSSC), *Fusarium oxysporum* complex, and *Fusarium fujikuroi*. Members of the FSSC cause the majority fusariosis cases of humans and are responsible for approximately two-thirds of all cases of fusariosis[1]. Disseminated *Fusarium* infection is a rare and serious fungal infection in immunocompromised patients, and clinical manifestations vary considerably[3]. The early diagnosis and treatment are quite difficult, and the mortality rate is estimated to be between 50% and 70% in adult patients. The remarkable intrinsic resistance of *Fusarium* species to most antifungal agents makes treatment more difficult. However, the optimal treatment for disseminated fusariosis has not been established[4]. Here, we describe the case of a 19-year-old male acute B-lymphocytic leukemia patient who had multiple skin lesions, endophthalmitis, and septic arthritis caused by *F. solani* infection during the course of chemotherapy.

## CASE PRESENTATION

### Chief complaints

A 19-year-old male presented to the Department of Dermatology of our hospital complaining of multiple skin lesions of the right neck, right calf, and left scrotum, which showed ulcerated painful nodules with a necrotic center.

### History of present illness

A 19-year-old male presented with swelling of the parotid gland and superficial lymphadenopathy for 2 wk and was admitted to our hospital. Through comprehensive examination, a diagnosis of acute B-lymphocytic leukemia was made. He was treated with BMF95 chemotherapy regimen (vindesine, daunorubicin, L-asparaginase, and prednisone) according to the National Comprehensive Cancer Network guidelines and achieved complete remission. The major adverse event of the treatment was myelosuppression. He received regular injections of granulocyte colony-stimulating factor (G-CSF) to increase the neutrophil count.

Five months later, he presented with unremitting fever during the fifth course of treatment. The highest body temperature was 41 °C. The result of blood culture was *Klebsiella pneumoniae*. His chest computed tomography (CT) scan was negative. He received moxifloxacin, vancomycin, and teicoplanin to treat septicemia. After treatment, he still had intermittent fever. Considering the high possibility of fungal infection in this patient, he started prophylactic anti-fungal therapy with caspofungin. Twenty-five days later, he developed multiple skin lesions, including ulcerated painful nodules with a necrotic center of the right neck, right calf, and left scrotum (Figure 1). Blood culture and galactomannan (GM) test of serum samples were negative. At the same time, he underwent surgical resection of infected skin tissues. Additionally, the lesions on his neck and scrotum underwent pathological examination and microbial culture, and the results supported *Fusarium spp.* infection (Figures 2 and 3). Then, he was treated with oral voriconazole (200 mg) twice daily and intravenous administration of AmB (amphotericin B) liposome (100 mg) once daily. His fever promptly disappeared on the 2<sup>nd</sup> day of treatment. During the therapy, he received regular injections of G-CSF. Upon re-examination of the complete blood count, his absolute neutrophil count recovered to  $0.9 \times 10^9/L$ . On the 6<sup>th</sup> day of antifungal treatment, he developed left eyeball pain and conjunctival hemorrhage with blurred vision. A plain CT scan of the orbit showed that the lateral wall was slightly thicker and the lacrimal gland was slightly swollen. *Fusarium* was cultured from the vitreous drainage fluid of the left eyeball, and AmB local eye drops were added to the treatment. However, the pain in the left eyeball worsened, and he experienced gradual blindness. Vitrectomy of the left eye was performed on the 15<sup>th</sup> day of anti-fungal treatment. At the same time, the skin lesion gradually subsided. On the 18<sup>th</sup> day of anti-fungal treatment, he developed pain in the right knee joint, and ultrasound showed knee joint effusion. We performed joint puncture and the surgeon extracted 70 mL of yellow turbid liquid, and the fungus cultured was *Fusarium*. The articular cavity was continuously washed with saline (1000 mL) and amphotericin B liposome (10 mg), and after treatment, the arthritis resolved. Moreover, his final neutrophil count recovered to  $1.7 \times 10^9/L$ . He had no recurrence after 3 mo of follow-up.

### History of past illness

The patient was diagnosed as acute B-lymphocytic leukemia 5 mo ago.

### Personal and family history

Patient denied family history of hereditary diseases.

### Physical examination

Physical examination showed that there were multiple skin lesions in the right neck, right calf, and left scrotum, including ulcerated painful nodules with a necrotic center (Figure 1).

### Laboratory examinations

Complete blood count showed a hemoglobin level of 6.4 g/dL, white blood cell count of  $0.21 \times 10^9/L$  (neutrophil:  $0.02 \times 10^9/L$ ), and platelet count of  $82 \times 10^9/L$ . The result of blood culture was *Klebsiella pneumoniae*. Serum GM test was negative.

Biopsy of the neck skin lesion and microscopic examination for fungus showed the presence of fungal elements and confirmed *Fusarium sp.* infection (Figures 2 and 3). Fungal susceptibility assays showed an AmB minimum inhibitory concentration of 4 µg/mL and resistance to terbinafine, micafungin, posaconazole, and voriconazole. Thereafter, polymerase chain reaction followed by sequencing of the internal transcribed spacer region confirmed that the fungus belonged to the FSSC.

Vitreous drainage fluid of the left eyeball cultured *Fusarium sp.* Right knee joint liquid also cultured *Fusarium sp.* Blood culture and GM test of serum samples were negative.

### Imaging examinations

Chest CT scan was negative. Plain CT scan of the orbit showed that the lateral wall was slightly thicker, and the lacrimal gland was slightly swollen.

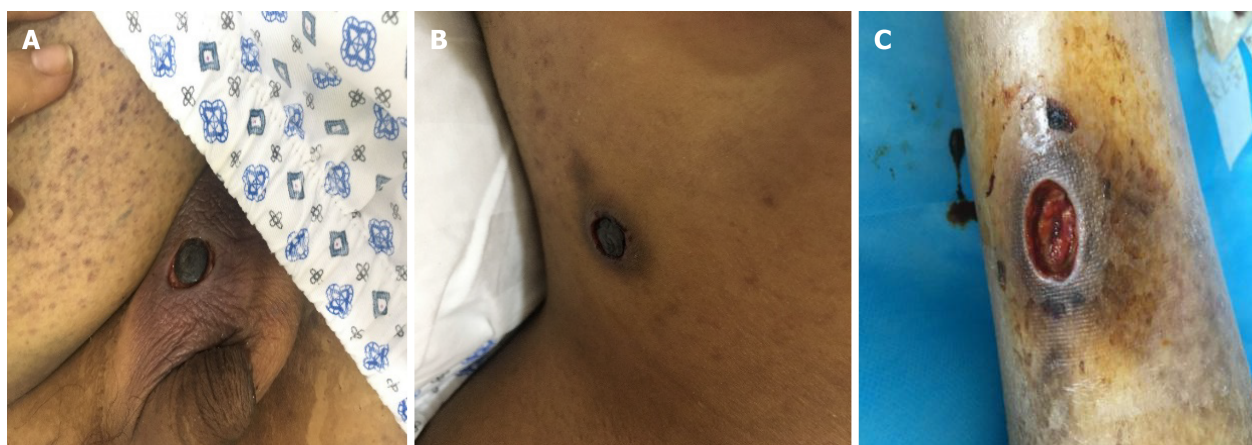
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## FINAL DIAGNOSIS

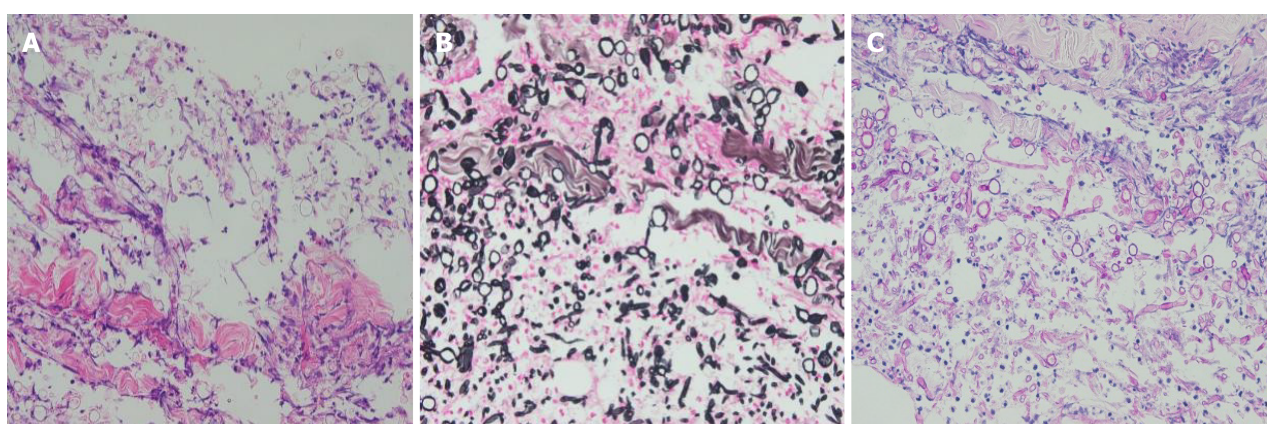
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Disseminated *F. solani* infection.

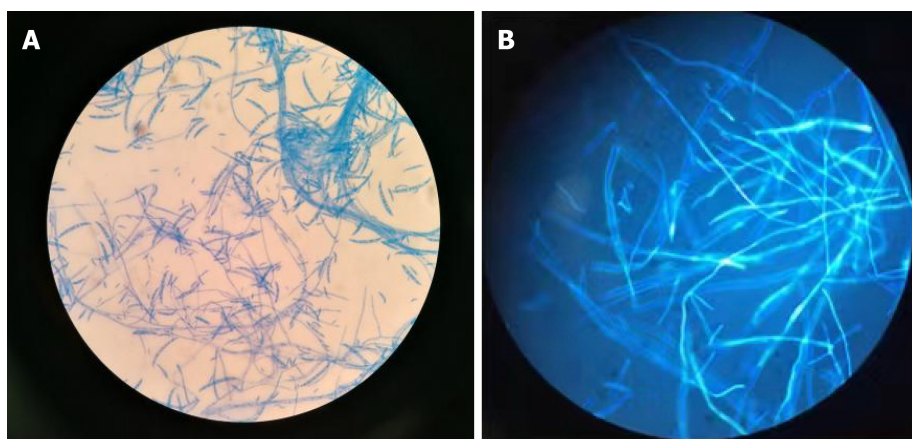




**Figure 1 Images of the nodules.** A: Left scrotum; B: Right neck; C: Right lower leg. Painful nodules are seen.



**Figure 2 Staining images.** A: Hematoxylin and eosin staining; B: Methenamine silver staining; C: Periodic acid-Schiff staining. *Fusarium* hyphae and spores are seen in the right neck lesion (magnification  $\times 400$ ).



**Figure 3 Microscopic examination of fungus.** A: Ordinary microscope; B: Fluorescence microscopy.

## TREATMENT

The patient was given systemic use of antifungal agents, oral voriconazole (200 mg) twice daily, intravenous administration of AmB liposome (100 mg) once daily, and regular injections of G-CSF to increase the neutrophil count. After surgical removal of skin and left eyeball lesions, local antifungal therapy was given. Extracted joint effusion was performed, and the articular cavity was continuously washed with saline (1000 mL) and AmB liposome (10 mg).



## OUTCOME AND FOLLOW-UP

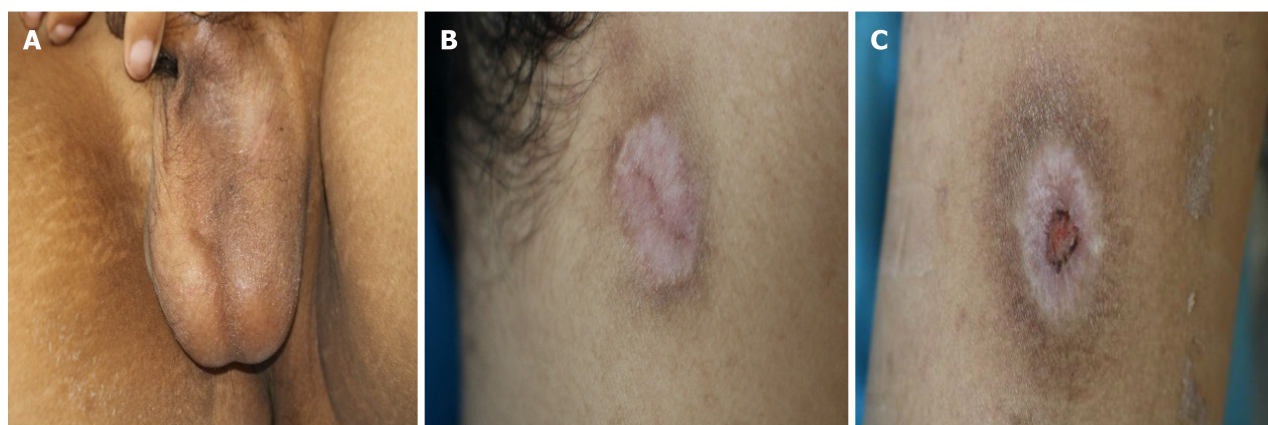
The skin lesions resolved, with pigmentation and crust (Figure 4). He had no recurrence after 3 mo of follow-up.

## DISCUSSION

The most common risk factors of disseminated *Fusarium* infection in acute lymphocytic leukemia are prolonged neutropenia (absolute neutrophil count  $\leq 0.5 \times 10^9/\text{L}$ ) due to intensification of cytotoxic chemotherapy and the wide spread use of corticosteroids and other immunosuppressive agents[5]. Clinical manifestations of invasive *Fusarium* infection vary considerably and often involve skin as well as lung or sinus lesions[6]. The most frequent pattern of disseminated disease is a combination of cutaneous lesions and positive blood cultures with or without involvement at other sites (the sinuses, lung, joint, and others)[7]. In many patients, skin lesions may be the first sign of a disseminated infection and are commonly seen in the early stages of the disease. It has been reported that 70% of patients often present with characteristic skin lesions involving multiple nodular and painful lesions, frequently with a necrotic center[8]. Pulmonary nodules have been reported in 80% of patients with respiratory symptoms, and nodules greater than 1 cm should raise the suspicion of invasive fungal infection [9]. Although some patients received broad-spectrum antifungal prophylaxis and underwent the most active antifungal agent treatment against this fungus, their clinical condition remained serious[10]. The worst disseminated fungal infections can trigger multiple organ failure and lead to patient death.

An early and definitive diagnosis requires isolation of *Fusarium* sp. from infected sites (the skin, sinuses, lungs, blood, and others) by direct microscopic examination or microorganism culture. There is a relatively high frequency of positive blood cultures (about 80%) for *Fusarium* sp.[11]. In this patient, the blood culture and GM test of serum samples were negative, probably because of the continuous prophylaxis antifungal treatment. Lung involvement is common in invasive fusariosis. The clinical presentation is nonspecific and includes dry cough and shortness of breath[12]. Chest CT is the imaging method in patients with pulmonary *Fusarium* infection[3]. The most common findings are nodules or masses but are nonspecific. Etiological diagnosis is critical, and until reliable non-invasive diagnostic approaches become available, invasive procedures, such as bronchoscopy with broncho-alveolar lavage and lung nodule biopsy, will continue to be necessary[1]. Skin biopsy of a nodule is necessary in patients with multiple skin lesions, and histopathological findings include branching septate hyaline hyphae with sporulation. Microorganism culture identification is important because *Fusarium* can produce hyaline, crescent or banana-shaped, multicellular macroconidia[13]. However, different species may present the same structure, or the outcome may be negative. Given these difficulties, new approaches have been pursued[4]. Polymerase chain reaction and ribosomal RNA internal transcribed spacer sequencing can identify the species of *Fusarium*. These technologies hasten the identification of fungi and are more accurate[14]. Multilocus sequence typing is currently viewed as a promising new approach to diagnose fusariosis. Matrix-assisted desorption/ionization time of flight mass spectrometry is a promising new tool for rapid identification and classification of culture microorganisms based on their protein spectra[15].

Therapy for invasive fusariosis is a challenge, mainly because *Fusarium* shows high minimum inhibitory concentrations to antifungal agents[16]. Additionally, owing to the lack of clinical trials, there is no proven effective treatment regimen. In reported cases, the treatment of these diseases often depended on a combination of antifungals. In some cases, treatment should include surgical debridement. Voriconazole, AmB, and various combinations have been reported with varying success. Data on combination therapy for fusariosis, such as caspofungin plus AmB, voriconazole plus AmB, and voriconazole plus terbinafine, have been reported[17]. For immunocompromised patients, treatment should include voriconazole or AmB as initial therapy and posaconazole as salvage therapy. Localized infection, such as keratitis, is usually treated with topical antifungal treatment, and natamycin is the drug of choice. Skin lesions may be the source for disseminated and life-threatening *Fusarium* infections. Fungal growth might occur in compartments where insufficient antifungal drug concentrations are achieved, such as the joint and eyeball[5]. Local debridement should be performed, and topical antifungal agents should be used. Reversal of immunosuppression, especially the restoration of the neutrophil count, is essential for



**Figure 4** Images of the lesions after treatment. A: Left scrotum; B: Right neck; C: Right lower leg. The skin lesions have gradually subsided.

a successful therapeutic outcome[15].

Among acute lymphocytic leukemia patients, invasive fungal infections are very common. *Candida* and *Aspergillus* are the most common invasive fungal infection pathogens[5]. *Fusarium*, *Zygomycetes sp.*, *Alternaria sp.*, and *Exserohilum sp.* are less prevalent. The most frequent site of infection is the respiratory tract, including pulmonary, sinus, or nasopharyngeal infections[18]. *Fusarium* infection has its own characteristics. Empiric antifungal treatment is usually AmB due to its broad spectrum coverage, then switched to other antifungals based on susceptibility results.

## CONCLUSION

Our case illustrates the early detection and successful intervention of an invasive *F. solani* infection. Identifying the risk factors of invasive *Fusarium* infection and actively looking for the pathogen can help early diagnosis and treatment, which are important to prevent progression to a more aggressive or disseminated infection. In general, disseminated *Fusarium* infection requires the use of systemic agents and immunotherapy. Localized infection likely benefits from surgical debridement and the use of topical antifungal agents.

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