Disseminated Intravascular Infection Caused by *Paecilomyces variotii*: Case Report and Review of the Literature

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*Paecilomyces variotii* is a ubiquitous environmental saprophyte with worldwide distribution. Commonly found in soil and decomposing organic material [1, 2], *P. variotii* can also be isolated from drinking water [3] and indoor and outdoor air [4–6]. In immunocompetent hosts, *P. variotii* has been reported as a cause of locally invasive disease including prosthetic valve endocarditis [7, 8], endophthalmitis [9, 10], rhinosinusitis [11, 12], and dialysis-associated peritonitis [13, 14]. In contrast, disseminated infections are more commonly reported in immunocompromised patients, including those with chronic granulomatous disease [15], solid malignancy [16], acute leukemia [17], lymphoma [18], multiple myeloma [19], and after stem cell transplant for myelodysplasia [20]. In 1 case series examining invasive infections by non-*Aspergillus* molds, *P. variotii* was the most common cause after *Fusarium* spp. [21]. Here, we present the case of an immunocompetent patient with extensive intravascular infection involving prosthetic material. We describe successful induction therapy with combination antifungals and extended suppression with posaconazole with clinical quiescence and eventual normalization of serum fungal biomarkers.

**Keywords.** endovascular infection; endovascular mold; invasive mold; *Paecilomyces*.

**CASE REPORT**

A 60-year-old man presented with thoracic aortic dissection 7 years before this presentation. At the time of the dissection, he underwent repair of his ascending thoracic aorta with Hemashield graft and aortic valve resuspension; he was clinically well for the intervening 7 years. He then presented with left flank pain. Computed tomography angiogram (CTA) revealed left renal and splenic infarcts, which were felt to be the result of bland embolization originating from residual dissection flaps. He was discharged with anticoagulation.

He returned 2 months later with left arm numbness and weakness and was found to have occlusion of his left subclavian artery extending to the radial artery. He underwent extensive thromboembolectomy. The excised clot was felt to have an unusual appearance and was sent for routine bacterial culture and pathology. Findings on transthoracic echocardiogram and CTA were concerning for mural thrombus within the false lumen of the distal infrarenal abdominal aorta and adherent to the wall of the ascending aorta at the distal end of the aortic graft. Imaging also demonstrated evidence of mycotic pseudoaneurysms of the left subclavian and middle colic branch of the superior mesenteric artery and of multiple small cerebral mycotic aneurysms with punctate subacute right parietal and chronic right cerebro and precentral gyral infarcts. All of these findings raised concern for infection of his prosthetic aortic graft with septic embolization.

Aerobic and anaerobic cultures of the excised thrombus and routine blood cultures drawn at the time of his initial presentation all had no growth. Histopathology of both the brachial and radial portions of the thrombus revealed numerous fungal hyphal forms (Figure 1). Morphology was consistent with hyalophyphomycosis, as the fungal elements were septate and appeared nonmelanized when stained with hematoxylin and eosin. Both acute- and right-angle branching were present, with neither predominating, and numerous dilations (varicosities) of the hyphae were visible. The latter 2 features are uncommon in *Aspergillus* and raised the suspicion of a non-*Aspergillus* hyaline mold such as *Fusarium* or *Paecilomyces* or a dematiaceous mold with nonpigmented hyphae such as *Scedosporium*. Serum beta-D-glucan and galactomannan were both greater than the upper limit of quantification (Figure 2), supporting a diagnosis of extensive endovascular fungal disease.

Following the return of the pathology report from the excised thrombus, additional samples were taken from a necrotic,
possibly septic area of the left kidney and from a residual left upper extremity hematoma, but cultures including dedi-
cicated fungal cultures were negative. The paraffin-embedded,
formalin-fixed pathology specimen of the excised thrombus
was sent to the Department of Laboratory Medicine at the
University of Washington for amplification and sequencing of
16S and 28S rDNA. No bacterial DNA was detected; sequencing
of 28S rDNA identified *Paecilomyces variotii*.

After the renal and residual hematoma biopsies were
obtained, liposomal amphotericin B 5 mg/kg/d was initiated
pending sequencing results. After 4 days, in response to acute
kidney injury, therapy was switched to micafungin 150 mg in-
travenously (IV) daily and voriconazole 300 mg twice daily
(initially IV, then oral [PO]). When DNA sequencing results
returned, given published data suggesting that posaconazole
has more favorable in vitro activity against *P. variotii* [22, 23]
voriconazole was stopped and delayed release posaconazole
300 mg PO daily was started. Micafungin was continued.

The patient was re-admitted the following month for ulnar
neuropathy caused by brachial plexus compression, caused
by enlargement of his left subclavian pseudoaneurysm.
Posaconazole trough drawn at steady state was in the ther-
peutic range at 1300 ng/mL (serial levels in Table 2). Although
beta-D-glucan levels were still greater than the upper limit of
quantification, galactomannan had decreased to 2.1 (Figure 2).
He underwent resection of the pseudoaneurysm, repaired with
caratid-distal subclavian artery prosthetic bypass graft. The pro-
cedure was complicated by recurrent laryngeal nerve injury and
left posterior cerebral artery stroke. When he presented for out-
patient follow-up 2 months postprocedure, his resulting hoarse-
ness had improved and he was recovering well from his right
homonymous hemianopsia. In the setting of clinical improve-
ment and undetectable galactomannan, micafungin was discon-
tinued even though beta-D-glucan remained greater than assay.

On continued posaconazole therapy, he has achieved clin-
ical stability. Acknowledging the difficulty in correlating serum
fungal biomarker kinetics with clinical outcomes [24], we have
nevertheless been reassured that his beta-D-glucan level gradu-
ally normalized (Figure 2). To date, he has received 40 months
of posaconazole 300 mg daily, with plans for life-long suppres-
sive therapy.

**DISCUSSION**

We present a case of disseminated intravascular infection with
*P. variotii* in an immunocompetent host. *P. variotii* intraocular
lens implant–associated endophthalmitis has been associated
with operating room ventilation repairs [10]. We hypothesize
that in the absence of any other predisposing factors, given this
organism’s environmental ubiquity [3–6], the patient may have
been inoculated at the time that his endovascular graft was
placed, with the long clinical latency explained by the organism’s
low virulence in an immunocompetent host. Discussion with
infection control at the institution where the graft was placed
did not reveal any additional cases.

Our case is also unusual in that the microbiologic diagnosis
was made by 28S rDNA sequencing. A diagnosis of fungal in-
fection was not suspected until the pathology resulted; thus
the thrombus was not sent for directed culture at the time of
thromboembolectomy. Although *P. variotii* can occasionally
be cultured directly from the blood [18, 19, 21, 25], it did not
grow in our case. Besides *Aspergillus* spp., the galactomannan
assay is known to detect other closely related molds in the
family Trichocomaceae, including *Paecilomyces* and *Penicillium*
spp. [26, 27]. Galactomannan can also occasionally cross-react
with more distantly related filamentous fungi such as *Fusarium*
[28], as well as dimorphic fungi such as *Histoplasma* and

![Figure 1. Histopathology from excised thrombus. A, B, Hematoxylin and eosin
stain, 40× (A) and 400× (B). Numerous hyaline fungal hyphae are visible throughout
the thrombus (arrowhead), some with varicosities (arrow). C, D, Gomori’s
methenamine silver stain, 100× (C) and 400× (D). This stain better highlights the
abundant septate hyphae present (arrowhead), with occasional varicosities (arrow).
](https://academic.oup.com/ofid/article-abstract/7/6/ofaa166/5837465/5837465)

![Figure 2. Serum fungal biomarkers over time. Galactomannan and beta-D-
glucan were measured in peripheral blood over time after initiation of therapy.
Galactomannan is reported as an index, with 3.75 being the assay maximum and
0.5 the lower limit of detection. Beta-D-glucan is reported in pg/mL.
](https://academic.oup.com/ofid/article-abstract/7/6/ofaa166/5837465/5837465)
| Year | Infection | Organism Identification | Comorbidities | Treatment | Outcome |
|------|-----------|-------------------------|---------------|-----------|---------|
| 1963 | Prosthetic mechanical mitral valve endocarditis complicated by septic emboli to spleen, kidneys, brain | Growth from blood cultures, identification on pathology | Rheumatic fever | Mycostatin 500000 U Q6H | Treatment failure (death due to heart failure and lack of neurological improvement) |
| 1974 | Prosthetic mechanical aortic valve endocarditis | Growth from blood cultures, identification on pathology | Idiopathic severe aortic insufficiency | AMB 30–50 mg QD, 5FC 2.5 g QD (ultimately discontinued due to toxicity) | Treatment failure (death due to heart failure and septic cerebral emboli complicated by subarachnoid hemorrhage) |
| 1981 | Ventriculo-peritoneal shunt infection | Growth from CSF, identification on pathology of centrifuged CSF | Obstructive hydrocephalus due to basilar artery aneurysm, DM | Shunt exchange, intraperitoneal AMB 50 mg, then 100 mg | Treatment failure (hemorrhage leading to death) |
| 1983 | Pyelonephritis | Growth from stone sample | Nephrolithiasis | Uretero-lithotomy and antibacterials alone | Resolution |
| 1984 | Maxillary sinusitis | Growth from biopsy, identification on pathology | Recent endodontic treatment of tooth 25 | Debridement alone | Resolution |
| 1985 | Pneumonia | Growth from bronchoscopy specimen | Hairy cell leukemia with distant steroids, chlorambucil, and cyclo-phosphamide followed by splenectomy | AMB 60 mg QD | Resolution |
| 1988 | Sphenoid sinusitis | Growth from sphenoidotomy specimen, identification on pathology | | Debridement, 2 doses of AMB | Resolution |
| 1991 | Peritonitis complicated by fungemia | Growth from catheter tip, blood cultures | Chronic interstitial nephritis on PD | Catheter removal, AMB | Resolution (with transition to HD) |
| 1991 | Peritonitis | Growth from dialysate | Wilms' tumor with chemoradiation complicated by CKD on PD | FLC 6 mg/kg QD, then 3 mg/kg QD (failure) leading to catheter removal, AMB, FLC 3 mg/kg after TiW HD | Resolution with latter regimen (with transition to HD) |
| 1992 | Pneumonia | Growth from bronchoscopy specimen | DM | KTC 400 mg QD (failure) leading to AMB | Resolution with latter regimen |
| 1992 | Purulent cellulitis | Growth from debridement sample | Autosomal recessive CGD on IFN-γ | AMB 0.8 mg/kg/d for 7 wk, then ITC 100 mg QD for 1 y | Resolution |
| 1993 | Peritonitis (4 cases) | Growth from dialysate | PD | AMB intraperitoneal with failure leading to catheter removal in 2 cases, with 1 of those cases followed by total AMB 1480 mg over 4 wk; KTC 400 mg TID for 10 d, catheter removal in 1 case; KTC 200 mg QD with catheter removal in another | Resolution (with transition to HD) in all cases |
| 1995 | Chronic suppurative otitis media | Growth from biopsy specimen | Chronic amoebic dysentery | Debridement, KTC 200 mg PO QD for 1 mo, complicated by relapse, then topical TFC cream | Resolution |
| 1995 | Multifocal osteomyelitis, pneumonia | Growth from biopsy specimen | CGD | AMB 1.5 g/kg total dose, IFN-γ then ITC 200 mg QD for 1 y | Resolution |
| 1995 | Saline breast implant contamination | Growth from implant fluid | | Implant removal without reimplantation | Resolution |
| 1996 | Peritonitis | Growth from dialysate | Hepatitis B, PD | Catheter removal, ITC and 5FC for 4 wk | Resolution (with resumption of PD) |
| 1996 | Deep SSI (complicating cesarean section) | Growth from percutaneous drainage fluid | Gestational diabetes | Debridement, antibiotics alone | Resolution |
| 1996 | Fungemia | Growth from blood cultures | Allogeneic BMT, CVC | CVC removal, AMB total of 641 mg, ITC 100 mg QD for 3 mo | Resolution |
| 1998 | Peritonitis | Growth from dialysate | Chronic pyelonephritis complicated by CKD on PD | AMB 1 mg/kg/d for total dose of 2500 mg IV followed by 1 mg/L IP catheter removal, then ITC 400 mg QD for 5 wk, then ITC 200 mg QD for 11 mo | Resolution (with transition to HD) |
| 1999 | Endogenous endophthalmitis with altered mental status | Growth from vitreous aspirate | AML on cytotoxic chemotherapy | AMB (25 mg/d IV, intravitreal 5 mg/d for 3 injections, topical 2% hourly), vitrectomy | Resolution (with preservation of remaining vision) |
### Table 1. Continued

| Year | Infection | Organism Identification | Comorbidities | Treatment | Outcome |
|------|-----------|-------------------------|---------------|-----------|---------|
| 2000 | Peritonitis | Growth from dialysate | 14-mo-old with congenital bilateral renal hypoplasia on PD | FLC 5 mg/kg/d and 50 mg/L intraperitoneally for 4 wk | Resolution (with continuation on PD) |
| 2002 | Deep sternal SSI | Growth from sternal debridement tissue | Idiopathic bronchiectasis leading to bilateral lung transplantation | AMB for total dose of 1500 mg, debridement, then ITC 400 mg QD for 1 y | Resolution |
| 2003 | Meningo-encephalitis | Growth from CSF | Metastatic breast cancer on cytotoxic chemotherapy, DM | AMB 100, then 150, then 200 mg QD | Treatment failure (worsening mental status and gram-negative bacteremia leading to death) |
| 2003 | Peritonitis | Growth from dialysate | Hypertension and DM leading to CKD on PD | Catheter removal, AMB 50 mg QD, then ITC 200 mg QD | Resolution |
| 2004 | Exogenous endophthalmitis | Growth from vitrectomy specimen | DM, IOL for cataract | Vitrectomy, intravitreal AMB 5 mcg, KTC PO | Resolution (but with remaining visual acuity only finger counting at 2 m) |
| 2005 | Splenic abscess | Growth from abscess cultures | X-linked CGD | Drainage partial splenectomy, AMB 1–1.5 mg/kg/d for 1 wk then FLC 10 mg/kg/d, 5FC 100 mg/kg/d for 14 mo | Resolution |
| 2005 | Fungemia | Growth from blood cultures | MM leading to autologous BMT, CVC | AMB for 6 wk | Resolution |
| 2007 | Pyelonephritis | Growth from suprapubic urine culture and left ureteral stent | DM, nephrolithiasis with ureteral stents in place | AMB 1 mg/kg/d for 4 wk | Resolution |
| 2010 | Pneumonia | Growth from broncho-atelectatic lavage fluid | NHL treated with chemotherapy and allogeneic BMT complicated by presumed Aspergillus pneumonia, CMV esophagitis | AMB | Treatment failure (persistently elevated galactomannan with death from esophageal hemorrhage from CMV disease) |
| 2013 | Purulent nodular cellulitis | Growth from skin biopsy | DM | ITC 200 mg BID for 6 mo | Resolution |
| 2014 | Peritonitis (3 cases) | Growth from dialysate | PD, 1 also with DM | AMB in all cases (with 800 mg, 750 mg, 900 mg cumulative doses), additional ITC in 1 case | Resolution (but 1 with pneumonia leading to death and the others with transition to HD) |
| 2015 | Pneumonia | Growth from associated pleural effusions | DM | ITC 200 mg BID for 4 wk | Resolution |
| 2015 | Peritonitis | Growth from peritoneal fluid | Wison's disease necessitating liver transplant | AMB 3 mg/kg/d for 10 d combined with VRC 7 mg/kg BID ultimately for 4 additional wk | Resolution (with preservation of graft function) |
| 2016 | Pan-sinusitis | Growth from sinus tissue | AML treated with chemotherapy, haploidentical BMT | Debridement, ITC 200 mg BID for 3 mo | Resolution |
| 2016 | Pneumonia | Growth from broncho-atelectatic lavage fluid culture | NHL, chemotherapy complicated by HBV reactivation and liver failure requiring transplant | VRC 16 mg/kg/d then 4 mg/kg BID (with infusion reaction), then POS 300 mg BID to QD | Resolution |
| 2017 | Peritonitis | Growth from dialysate | PD | AMB 3 mg/kg/d, ITC 400 mg QD for 4 wk | Resolution (with transition to HD) |
| 2017 | Fungemia | Growth from blood cultures | NHL, chemotherapy complicated by HBV reactivation and liver failure requiring transplant | AMB 5 mg/kg/d, VRC 200 mg BID for 8 d, then AFG 100 mg QD for 3 wk, then POS 200 mg suspension QID for 10 wk | Resolution |
| 2018 | Cutaneous ulcers | Growth from biopsy specimens | Renal transplant, DM | VRC | Resolution (but with re-admission with presumed bacterial pneumonia leading to death) |
| 2019 | Pulmonary mycetoma | Growth from broncho-atelectatic lavage fluid culture | Interstitial lung disease on prednisone | POS | Resolution |

Only reports with full-text articles available were included. Drug dosages were included when available.

Abbreviations: 5FC, flucytosine; AFG, anidulafungin; ALL, acute lymphocytic leukemia; AMB, amphotericin B; AML, acute myeloid leukemia; BID, twice daily; BMT, bone marrow transplant; CGD, chronic granulomatous disease; CKD, chronic kidney disease; CMV, cytomegalovirus; CSF, cerebrospinal fluid; CVC, central venous catheter; DM, diabetes mellitus; FLC, fluconazole; HD, hemodialysis; IFN-γ, interferon gamma; IOL, intraocular lens implantation; ITC, itraconazole; IV, intravenous; KTC, ketoconazole; MM, multiple myeloma; NHL, non-Hodgkin lymphoma; PD, peritoneal dialysis; PO, oral; POS, posaconazole; QD, once per day; QID, four times daily; SSI, surgical site infection; TID, three times daily; TIW, three times weekly; VRC, voriconazole.
Blastomyces [26, 29]. Though we find it unlikely, it remains possible that our patient’s infection was caused by a more fastidious galactomannan-positive mold that also responded to posaconazole, and the 28S result reflects environmental contamination of the pathology specimen. However, the referral lab that performed the PCR-based identification advised us that they have not previously identified *P. variotii* on PCR-based tissue testing, suggesting that this is not a common contaminant. Hopefully, as fungal DNA amplification and sequencing become more common, as available reference databases expand, and as protocols for DNA extraction become more standardized, there will be more data on the sensitivity and specificity of this approach. To aid in this endeavor, consensus definitions of invasive fungal infections have recently been updated to allow classification as “proven invasive mold infection” cases in which molds are seen on pathology and fungal DNA is successfully amplified [30].

Review of the literature is complicated by frequent microbiologic identification only to the genus level and previous taxonomic grouping of *P. variotii* with the generally more triazole-resistant (*now Purpureocillium* lilacinum) [31, 32]. Previous reports have mainly described treatment with amphotericin B formulations, often in combination with or with transition to an extended-spectrum triazole (Table 1). European guidelines endorse this practice [33], but in our case, kidney injury limited duration of liposomal amphotericin B therapy to 4 days. Though in vitro activity is difficult to correlate with clinical efficacy in non-*Aspergillus* mold infections [34] several in vitro studies of *P. variotii* have demonstrated low minimum inhibitory concentrations of echinocandins (with micafungin minimum inhibitory concentrations more favorable than those of caspofungin or anidulafungin) as well as triazoles (with posaconazole and itraconazole more active than voriconazole) [21, 35–38]. Data are scant for newer agents such as isavuconazole, but 1 study generated promising data for ibrexafungerp [35]. In vitro synergy has not been demonstrated between echinocandins and triazoles for *P. variotii* [36, 39] but given the extent of the infection and our inability to facilitate surgical debulking, our patient was initially treated with both micafungin and posaconazole. The patient remains well, now >3 years after transition to posaconazole monotherapy. His excellent outcome is striking in its contrast to those of patients with hematologic malignancy and non-*Aspergillus* mold infections [34] and perhaps reflects his preserved immune system more than the treatment strategy used.

Increasingly sophisticated molecular diagnostic approaches facilitate definitive organism identification for a growing number of unusual or difficult-to-diagnose infections. This increase in microbiologic diagnoses, in turn, offers the opportunity to expand our understanding of the spectrum of infections caused by individual organisms. Together with previously reported cases, our case suggests that *P. variotii* may have a predisposition for causing endovascular infection associated with prosthetic material in immunocompetent hosts. The case additionally illustrates that infection with low-virulence organisms can become extensive before causing symptoms that drive a clinical presentation. Failure to culture the organism despite a significant endovascular burden underlines the critical role that molecular diagnostics can play for both diagnosis and management. In this case, although our suspicion of fungal infection was very high based on pathology, sequencing results directed a change in antifungal agent. Our case additionally provides supportive evidence for the successful early use of posaconazole for endovascular *P. variotii* infection; given the substantial potential side effects of amphotericin formulations, an early change to alternate agents may have overall long-term benefit to patients.

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**Potential conflicts of interest.** J. A. B. reports receiving consulting fees from Roche Diagnostics, DiaSorin, Inc., and T2 Biosystems. M. B. B. reports being a stockholder of Pfizer Inc. A. K. B. reports being a co-inventor on US patent 9885088, Rapid phenotypic diagnosis of transcriptional expression signatures.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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**Table 2. Delayed-Release Posaconazole Therapeutic Drug Monitoring**

| Date       | Dose      | Level       | Trough | Steady State |
|------------|-----------|-------------|--------|--------------|
| 08/31/2016 | 300 mg daily | 1570 ng/mL | Yes    | Yes          |
| 09/09/2016 | 300 mg daily | 1940 ng/mL | Yes    | Yes          |
| 09/24/2016 | 300 mg daily | 1300 ng/mL | Yes    | Yes          |
| 10/04/2016 | 300 mg daily | 3420 ng/mL | Yes    | Yes          |
| 03/18/2017 | 300 mg daily | 3630 ng/mL | Yes    | Yes          |
| 08/29/2017 | 300 mg daily | 2990 ng/mL | Yes    | Yes          |
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