



Case Report

Prototheca wickerhamii breast implant infection after reconstructive surgery: a new level of complexity

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ABSTRACT

We report the first published case of *Prototheca wickerhamii* breast implant infection. This occurred after mastectomy, chemotherapy, radiotherapy, breast reconstruction, implant revisions and breast seroma aspirations and was preceded by polymicrobial infection. Definitive treatment required implant removal and intravenous liposomal amphotericin B. The management of breast prosthesis infections is discussed.

1. Introduction

Prototheca spp. are saprophytic algae that can colonise the skin, nails and respiratory and gastrointestinal tracts. More than 210 cases of human protothecosis have been reported worldwide since 1964 [1]. *Prototheca wickerhamii* and *Prototheca zopfii* are the most common pathogenic species in humans. Infection requires inoculation into skin or mucous membranes after exposure to environmental reservoirs of *Prototheca* spp. such as contaminated water, soil, vegetation and organic matter. Protothecosis can affect both immunocompetent and immunocompromised hosts. The incubation period is unknown and transmission between humans is not reported [1–3].

Three distinct clinical syndromes can arise from protothecosis, namely, cutaneous infection, local or organ-specific infections and disseminated infection with algaemia. Local or cutaneous infections can be successfully treated with surgical excision and concurrent topical or oral antifungals. Systemic antifungals are required for disseminated or deep-seated infections. The correlation between *in vitro* susceptibility and clinical outcome *in vivo* is uncertain. As resistance to amphotericin B has not been reported, it is regarded as the agent of choice for invasive

protothecosis [1,2]. Topical and oral triazoles are more commonly used for cutaneous and local protothecal infections. Susceptibility to triazoles and echinocandins is isolate-dependent, therefore clinical failure can occur requiring salvage therapy with amphotericin B. As *Prototheca* spp. are regarded as resistant to 5-flucytosine, it is unknown if co-administration with amphotericin B provides any clinical advantage. Synergy *in vitro* has been demonstrated between amphotericin B and antibiotics such as tetracycline, gentamicin and amikacin. However clinical success has only been reported with topical amphotericin B and oral tetracycline in five cases of cutaneous protothecosis and with intravenous (IV) amphotericin B and oral tetracycline for one case of algal peritonitis [3].

Device and prosthesis-associated *Prototheca* infections have included peritonitis with Tenckhoff catheters, meningitis after insertion of ventriculoperitoneal (VP) shunts, nasopharyngeal ulceration with endotracheal intubation, vascular access device-related algaemia and keratitis or endophthalmitis associated with corneal grafts (Refer to Table 1) [1–7]. Breast reconstruction devices including tissue expanders and implants are being used increasingly in the treatment of breast cancer [8]. Breast implant protothecosis has not previously been described.

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Table 1
Device- and prosthesis-associated protothecal infections.

Case	Predisposing Conditions	Site	Device	Antifungal Therapy	Duration of Therapy	Algaemia	Died	Reference	
1	41yo F	ESRF, PD, IP ABX	Peritoneum	Tenckhoff catheter	IV AMB, intra-operative AMB lavage (once)	10 days	N	N	[22]
2	45yo M	ESRF, PD, IP ABX, DM	Peritoneum	Tenckhoff catheter	Induction: IP AMB and 5-FC Maintenance: Oral FLC 200mg/day post-dialysis	Induction: 48hrs Maintenance: 6 weeks	N	N	[23]
3	7yo M	HL, CTX ^b , TPN	Bloodstream	Hickman catheter	IV AMB (1mg/kg/day)	5 days	Y	N	[26]
4	72yo M	ESRF, PD	Peritoneum	Tenckhoff catheter	IV AMB (50mg thrice weekly), oral DOX 100mg/day	30 days	N	Y ^f	[21]
5	24yo F	DM	Nasopharynx	Endotracheal tube	IV AMB (0.5mg/kg/day)	72 days	N	N	[31]
6	32yo M	AML, CTX ^c , febrile neutropaenia, ITC prophylaxis	Bloodstream	CVC	IV AMB	19 days	Y	N	[27]
7	19yo M	HL, alloSCT, GVHD, steroids	Bloodstream	CVC	IV L-AMB (3mg/kg/day)	Not stated	Y	N	[4]
8	36yo M	ESRF, PD, congenital rubella	Peritoneum	Tenckhoff catheter	Empiric: FLC, 5-FC Directed: IV AMB, ITC	Directed: 2 weeks	N	N	[24]
9	61yo M	Liver Tx (TAC, MMF, PRED), resolved HCV, DM, MFG prophylaxis	Bloodstream	CVC	IV AMB	Not stated	Y	Y	[28]
10	54yo M	Fuchs corneal endothelial dystrophy, topical ocular steroids	Cornea	Bilateral keratoplasties	Oral FLC 100mg bd	Not stated	N	N	[5]
11	6mo M	Chronic hydrocephalus	CNS	Extra-ventricular drain, VP shunt	Initial: Oral KTZ 22mg/kg/day Subsequent: IV AMB 1–3mg/kg/day, IV FLC 6mg/kg/day	Initial: 9 days Subsequent: 3 days	N	N	[29]
12	3yo M	ALL, alloSCT, CTX ^d , PD, MFG prophylaxis	Peritoneum	Tenckhoff catheter	Empiric: MFG, CFG, VRC, FLC	54 days	N	Y ^g	[25]
13	23yo M	Chronic meningitis, empiric TB therapy ^e	CNS	Ommaya reservoir, VP shunt	IV AMB (30–35mg/day), oral 5-FC 100mg tds	Initial: 3 months Subsequent: 6 months	N	Y ^h	[30]
14	65yo F	SJS, Sjögren syndrome, keratolimbic allografts	Keratitis, endophthalmitis	Boston Type I keratoprosthesis	Topical 0.15% and intra-vitreous AMB, Topical 0.02% CHX, Topical 1%, intra-vitreous and oral VRC 200mg bd	Intermittent treatment over 26 months with multiple surgical revisions	N	N	[6]
15	12yo M	Eosinophilic meningoencephalitis, steroids	CNS	VP shunt	IV AMB (40mg/day)	9 weeks	N	N	[7]
16 ^a	37yo F	Breast cancer, XRT, BRS	Right breast	Breast implant	IV L-AMB (4mg/kg/day)	14 days	N	N	

Abbreviations – ALL, acute lymphoblastic leukaemia; AlloSCT, allogeneic stem cell transplant; AML, acute myeloid leukaemia; bd, *bis in die*/twice a day; BRS, breast reconstructive surgery; CNS, central nervous system; CTX, chemotherapy; CVC, central venous catheter; DM, diabetes mellitus; ESRF, end stage renal failure; F, female; GVHD, graft versus host disease; HCV, Hepatitis C virus; HL, Hodgkin Lymphoma; IP, intra-peritoneal; IV, intravenous; M, male; mo, months old; MMF, mycophenolate mofetil; PD, peritoneal dialysis; PRED, prednisolone; SJS, Stevens-Johnson Syndrome; TAC, tacrolimus; TB, tuberculosis; tds, *ter die sumendum*/three times a day; TPN, total parenteral nutrition; Tx, transplant; VP, ventriculoperitoneal; yo, years old; XRT, radiotherapy.

Antimicrobials: ABX, antibiotics; AMB, amphotericin B; CFG, caspofungin; CHX, chlorhexidine; DOX, doxycycline; FLC, fluconazole; ITC, itraconazole; KTZ, ketoconazole; L-AMB, liposomal amphotericin B; MFG, micafungin; VRC, voriconazole; 5-FC, flucytosine.

^a Current Case.

^b Chemotherapy included mechlorethamine, vincristine, procarbazine and prednisolone.

^c Chemotherapy included cytosine arabinoside and mitoxantrone.

^d Chemotherapy included fludarabine, treosulphan, thiotepa, anti-thymocyte globulin and cyclosporin A.

^e TB therapy (rifampicin, isoniazid, ethambutol and pyrazinamide) was ceased at 2 weeks after the patient was diagnosed with protothecal meningitis.

^f Protothecal peritonitis had resolved 3 months prior and was not associated with the patient's death from enterococcal sepsis.

^g Tenckhoff catheter placed 1–2 days beforehand and was not removed prior to patient's death.

^h VP shunt was not removed prior to patient's death.



Fig. 1. Wrinkled cream-coloured colonies of *Prototheca wickerhamii* after 6 weeks of incubation on Sabouraud dextrose agar at 30°C.

2. Case

A 37-year-old woman had a right mastectomy with insertion of a tissue expander (MENTOR® CPX™4) for local recurrence of breast cancer, on a background of excision, axillary clearance, radiotherapy and chemotherapy nine years earlier. After eight months, a contralateral tissue expander was inserted following prophylactic left mastectomy. At twelve months, both expanders were replaced with breast implants (MENTOR® CPG™ 330cc textured silicone gel). Five months later, nipple reconstructions were performed and the right breast implant was repositioned due to rotation.

Two weeks post-operatively (day 0), right breast erythema developed with associated fever, leucocytosis with a white cell count of $11.3 \times 10^9/L$ (normal range, $4.0\text{--}11.0 \times 10^9/L$) and elevated C-reactive protein (CRP) of 134 mg/L (normal range <3 mg/L). Immediate surgical exploration to obtain diagnostic samples was performed. As a new implant was not immediately available, to preserve the reconstruction, the right breast implant was removed, scrubbed with single-use povidone-iodine, washed with sterile normal saline and re-implanted after capsular debridement and pulsed lavage with sterile normal saline. Operative specimens of breast tissue, nipple eschar and peri-implant fluid were obtained. Intravenous ceftriaxone and vancomycin were administered. The closed wound was irrigated for 16 hours post-operatively with sterile normal saline, draining via two drain tubes which were removed on day +5.

Moderate growth of *Klebsiella oxytoca* and scant growth of *Staphylococcus epidermidis* accompanied by a few polymorphs on the Gram stain were cultured from breast tissue. The nipple eschar showed heavy growth of *K. oxytoca*, *S. epidermidis* and *Pseudomonas fluorescens* with no polymorphs seen on the Gram stain. Heavy growth of *K. oxytoca*, with numerous polymorphs seen on the Gram stain, was obtained from peri-implant fluid. *K. oxytoca*, *P. fluorescens* and *S. epidermidis* were all susceptible to ciprofloxacin. *S. epidermidis* was additionally susceptible to rifampicin. Oral ciprofloxacin and rifampicin were administered. These agents were selected for their superior biofilm activity and tissue penetration given the presence of the breast prosthesis. The patient recovered well and the leucocytosis and elevated CRP level normalised. Subsequent wound dehiscence on day +38 was managed with further debridement and wound closure. *Candida albicans* was cultured from an intra-operative swab of peri-implant fluid and oral fluconazole was

added two days after debridement.

While on antimicrobial therapy with rifampicin, ciprofloxacin and fluconazole, the patient had ultrasound-guided aspirations of seroma fluid on day +41, day +44 and day +53. Microbiological culture of seroma fluid was not requested on day +41. However, scant growth of *C. albicans* was cultured from the breast aspirate specimen collected on day +44 and was associated with a few polymorphs on Gram stain. Culture of seroma fluid aspirated on day +53 did not grow any organisms.

The CRP and white cell count remained normal and on day +97, the patient returned for definitive surgery. The right breast implant was removed and curettage and washout with single-use povidone-iodine and pulsed lavage with sterile normal saline were performed. No macroscopic features of active infection were seen. Operative specimens were collected before a new right breast implant was inserted (MENTOR® 330cc Smooth Round Implant). After four days of incubation on chocolate agar at 35°C in CO₂, pure growth of an organism was seen from both the prosthesis and surrounding breast tissue. The cream-coloured colonies were suggestive of a yeast. A saline wet preparation showed cells of various sizes with thick refractile walls and sporangia resembling *Prototheca* spp. No hyphae or budding were seen. On sub-culture and further incubation the colonies appeared dry and wrinkled (Fig. 1).

Prototheca wickerhamii was identified on the VITEK® 2 Yeast identification card (bioMérieux, France). The isolate was referred to the National Mycology Reference Centre where it was confirmed as *Prototheca wickerhamii* by 18S rRNA sequencing. Minimum inhibitory concentrations (MIC) determined by broth microdilution using a Sensititre™ YeastOne™ YO10 antifungal susceptibility testing plate incubated at 35°C in ambient air for 72 hours (Thermo Fisher Scientific, United States) were: amphotericin B, MIC 0.5 mg/L and fluconazole, MIC >256 mg/L. There are no clinical breakpoints for interpreting *Prototheca* spp. susceptibility results.

For definitive management of the infection the right breast implant was removed on day +109 (12 days post-insertion) and liposomal amphotericin B at a dose of 4 mg/kg/day was administered for 14 days post-removal. *Prototheca wickerhamii* was not re-isolated however one colony of *Candida parapsilosis* was isolated from an intra-operative swab at removal of the implant. There has been no clinical relapse at 36 months post-implant removal.

3. Discussion

Breast reconstructive surgery post-mastectomy (BRSPM) often requires a two-stage process where a temporary tissue expander is exchanged for a breast implant and the nipple-areola complex is restructured. The Australian Breast Device Registry data from 2012 to 2018 included 41,921 procedures of which 19% were implant-based BRSPM [8]. Over this time period, 3,589 prosthesis revisions were performed for the following reasons: capsular contracture (40%), malpositioning (34%), haematoma or seroma (4%) and deep wound infection (3%) [8].

Worldwide, the reported incidence of BRSPM infection has wide variation at 1–35% [9–13]. BRSPM procedures have a higher incidence of infections compared with cosmetic breast augmentation [9]. Sources of infection include environmental contamination of the implant or surgical milieu, breaches in skin integrity and haematogenous or contiguous spread [12–14]. Risks for infection have not been formally quantified, however mastectomy and previous radiotherapy are thought to predispose to poorer reconstructive outcomes and surgical site infection [12–14].

Coagulase negative staphylococci, *Staphylococcus aureus* and *Cutibacterium acnes* are common causes of breast implant infections, however the range of potential pathogens is broad including Gram-negative bacteria, non-tuberculous mycobacteria and yeasts [9,12,14]. Prosthetic devices predispose to formation of biofilms which are three-dimensional structures comprised of organisms and exopolysaccharides that are resistant to phagocytosis and antimicrobials. *In vitro* studies demonstrate that *Prototheca* spp. have the ability to form single-species biofilms that can be resistant to systemic antifungals [15]. Given the polymicrobial nature of our patient's infection, including saprophytic algae, we presume environmental contamination of the breast implant. Strict adherence to recommendations for prevention of surgical site infection can minimise the risk of environmental contamination [16].

There are no current randomised controlled trials or definitive diagnostic or treatment guidelines endorsed by Infectious Diseases societies for breast prosthesis infections. Imaging-guided aspiration and/or intra-operative tissue sampling to confirm the causative organisms followed by empiric and then targeted antimicrobial therapy based on culture results, is advised [12–14]. Definitive treatment has traditionally involved prosthesis explantation, however this is associated with poorer cosmesis and significant emotional and financial costs [9,10,17]. Salvage strategies, with or without prosthesis exchange, have evolved in selected situations to avoid delayed reconstruction. Irrigation of the wound and prosthesis with saline, povidone-iodine and topical antibiotics is an established practice, however efficacy is unknown and limited to case reports [9,11,14,18]. Selected studies in breast augmentation surgery and the World Health Organization support use of povidone-iodine in wound irrigation, however it should be noted that the manufacturer instructions recommend this agent for external use only and the United States' Food and Drug Administration recommends single-use sterile iodine formulations for wound cavity lavage [14,16,18,19]. Potential adverse events associated with wound irrigation with topical antimicrobials include hypersensitivity reactions, impaired healing, environmental contamination, unanticipated drug interactions and antimicrobial resistance.

The salvage surgery strategy frequently advocated in severe infections includes debridement with or without capsulectomy, lavage and one-stage device exchange [9,11]. More recently, some centres have reported successful outcomes without prosthesis exchange, using antimicrobials with or without debridement. Spear and Seruya reported 100% success rate with a defined course of antimicrobials alone for mild

infection [11]. Franchelli et al. and Song et al. reported 57% and 58.6% success rates respectively with antimicrobials alone [9,20]. Viola et al. reported a 76% success rate using a regimen of antimicrobials with anti-biofilm activity [10].

We identified fifteen cases of device- and prosthesis-associated protothecal infections published between 1986 and 2017 in addition to our patient (Refer to Table 1). The patients involved were aged between 6 months and 72 years, were mostly male (12 patients, 80%) and included four paediatric patients (age range: 6 months to 12 years). With respect to patient characteristics, three had a background of haematological malignancy (acute myeloid leukaemia, acute lymphoblastic leukaemia and Hodgkin lymphoma) and three cases occurred in transplant recipients (liver, allogeneic hematopoietic stem cells and a keratolimbal ocular allograft). Three patients were receiving chemotherapy, four patients were prescribed corticosteroids and three others were on anti-rejection medication when protothecosis was diagnosed. Diabetes mellitus was a co-morbidity in three patients.

Devices and prostheses implicated in protothecosis included Tenckhoff catheters (5 cases) [21–25], Hickman lines or central venous catheters (4 cases) [4,26–28], VP shunts (3 cases) [7,29,30], keratoplasties (2 cases) [5,6] and an endotracheal tube (1 case) [31]. In most patients the source of infection was unknown, however in two cases of protothecal peritonitis involving Tenckhoff catheters, water exposure at a municipal swimming pool [22] and a river [23] were environmental risk factors. Nine patients (60%) presented with a fever and local features consistent with infection at the time of diagnosis however specific details with respect to inflammatory markers were omitted. Prior to *Prototheca* spp. being cultured, thirteen patients (87%) received intra-peritoneal, IV and/or topical antibiotics, two received micafungin and one received itraconazole prophylaxis. Pure growth of *Prototheca* spp. was cultured from normally sterile sites such as blood, tissue, cerebrospinal fluid and peritoneal fluid in eleven patients (73%). Co-pathogens that were isolated and treated concurrently in four patients included bacteria (*S. epidermidis*, *Enterococcus faecalis*, *Stenotrophomonas maltophilia*, *Leuconostoc* spp.) and yeast (*Candida glabrata*).

The optimal agent, dose, route of administration and duration of antimicrobial therapy for protothecosis is unclear. Clinical success has been reported with use of amphotericin B and triazoles for infections caused by *Prototheca* spp. [2,3]. Removal of the infected device or prosthesis and biofilm eradication is an important adjunct to antimicrobial therapy. Apart from one patient who died after the insertion of a Tenckhoff catheter for emergency peritoneal dialysis [25] and another who had a VP shunt retained at the time of death [30], all other cases had removal of the infected device or prosthesis as part of the treatment strategy for protothecosis. Due to the invasive nature of the device- and prosthesis-associated protothecal infections, IV amphotericin B was used in most cases (12 patients, 80%). Intra-peritoneal and intra-vitreous/cameral amphotericin B were used for Tenckhoff catheter-associated peritonitis and corneal graft-associated keratitis respectively. Duration of therapy varied considerably between cases. Median duration of therapy was 30 days (range: 3 days to 26 months). Mortality was 20% (3 patients). Two cases of fatal protothecosis involved immunocompromised patients with line-related bacteraemia (liver transplant) [28] and peritonitis (allogeneic stem cell transplant) [25]. One case involved an immunocompetent patient with chronic meningitis from an infected Ommaya reservoir and VP shunt [30]. In addition, death occurred three months after a resolved episode of protothecal peritonitis in one patient who died from a cardiac arrest after enterococcal sepsis [21]. Three of the four patients with line-related bacteraemia survived. Vascular access devices were removed for all four patients.

Breast implant infections can be due to a diverse range of pathogens, including as in this case, environmental algae. The optimal management and antimicrobial therapy for breast implant infections are yet to be established. Biofilm formation on prostheses has significant implications for both medical and surgical management strategies. As for any device- or prosthesis-related infection, removal of an infected implant should always be considered [9,12–14,17]. Our patient's breast implant infection caused by the algae *Prototheca wickerhamii* is the first reported case of breast implant protothecosis. Our patient was immunocompetent and did not participate in any water-based activities to predispose to protothecosis, however introduction of environmental flora through surgical revisions, aspirations or drain tubes is plausible. *Prototheca* spp. have been described as difficult to eradicate with antifungals alone due to their biofilm-forming potential. In our case, *P. wickerhamii* was regarded as a clinically significant pathogen as it was isolated from breast tissue in addition to the prosthesis. These microbiological features were supportive of an early, invasive, deep-seated tissue infection. Although our patient remained afebrile with normal inflammatory markers and did not have clinical features of breast cellulitis, implant retention was inappropriate once *P. wickerhamii* was cultured given the absence of guidelines for managing breast implant protothecosis. Current literature does not support breast implant salvage for persistent and incurable biofilm-based infections. Early surgical intervention and removal of the infected breast implant as soon as protothecosis was confirmed contributed towards clinical cure and treatment success for our patient. Biofilm active antimicrobial agents will likely play an increasing role adjunctive to surgical management of breast implant infections, guided by future clinical research.

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Declaration of competing interest

There are none.

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References

- [1] J. Todd, T. Matsumoto, R. Ueno, et al., Medical mycology 2017, *Med. Mycol.* 56 (2018) S188–S204.
- [2] J. Todd, J. King, A. Oberle, et al., Protothecosis: report of a case with 20-year follow-up, and review of previously published cases, *Med. Mycol.* 50 (2012) 673–689.
- [3] C. Lass-Flörl, A. Mayr, Human protothecosis, *Clin. Microbiol. Rev.* 20 (2007) 230–242.
- [4] H. Torres, G. Bodey, J. Tarrand, et al., Protothecosis in patients with cancer: case series and literature review, *Clin. Microbiol. Infect.* 9 (2002) 786–792.
- [5] A. Solky, N. Laver, J. Williams, et al., *Prototheca wickerhamii* infection of a corneal graft, *Cornea* 30 (2011) 1173–1175.
- [6] J. Ng, D. Minckler, T. Walsh, et al., An intractable case of *Prototheca* keratitis and chronic endophthalmitis in Stevens-Johnson Syndrome with Boston Type 1 keratoprosthesis, *Cornea* 35 (2016) 1257–1260.
- [7] A. Anh, Y.-J. Choe, J. Chang, et al., Chronic eosinophilic meningoencephalitis by *Prototheca wickerhamii* in an immunocompetent boy, *Pediatr. Infect. Dis. J.* 36 (7) (2017) 687–689.
- [8] I. Hopper, E. Parker, B. Pellegrini, et al., The Australian Breast Device Registry 2018 Annual Report, Monash University, Department of Epidemiology and Preventive Medicine, 2019.
- [9] S. Franchelli, M. Pesce, I. Baldelli, et al., Analysis of clinical management of infected breast implants and of factors associated to successful breast pocket salvage in infections occurring after breast reconstruction, *Int. J. Infect. Dis.* 71 (2018) 67–72.
- [10] G. Viola, J. Selber, M. Crosby, et al., Salvaging the infected breast tissue expander: a standardised multi-disciplinary approach, *Plast Reconstr Surg Glob Open* 4 (2016) e732–e743.
- [11] S. Spear, M. Seruya, Management of the infected of exposed breast prosthesis: a single surgeon's 15-year experience with 69 patients, *Plast. Reconstr. Surg.* 125 (4) (2010) 1074–1084.
- [12] T. Lalani, Breast implant infections: an update, *Infect. Dis. Clin.* 32 (4) (2018) 877–884.
- [13] L. Washer, K. Gutowski, Breast implant infections, *Infect. Dis. Clin.* 26 (26) (2012) 111–125.
- [14] B. Pittet, D. Montandon, D. Pittet, Infection in breast implants, *Lancet Infect. Dis.* 5 (2005) 96–106.
- [15] J. Kwiecinski, Biofilm formation by pathogenic *Prototheca* algae, *Lett. Appl. Microbiol.* 61 (6) (2015) 511–517.
- [16] World Health Organization, Global Guidelines for the Prevention of Surgical Site Infection, second ed., World Health Organization, 2018.
- [17] S. Spear, M. Howard, J. Boehmler, et al., The infected or exposed breast implant: management and treatment strategies, *Plast. Reconstr. Surg.* 113 (2004) 1634–1644.
- [18] A. Deva, W. Adams, K. Vickery, The role of bacterial biofilms in device-associated infection, *Plast. Reconstr. Surg.* 132 (2013) 1319–1328.
- [19] H. Hu, K. Johani, A. Almatroudi, et al., Bacterial biofilm infection detected in breast implant-associated anaplastic large-cell lymphoma, *Plast. Reconstr. Surg.* 137 (6) (2016) 1659–1669.
- [20] J. Song, Y. Kim, B. Jung, et al., Salvage of infected breast implants, *Arch. Plast. Surg.* 44 (6) (2017) 516–522.
- [21] M. Sands, D. Poppel, R. Brown, Peritonitis due to *Prototheca wickerhamii* in a patient undergoing chronic ambulatory peritoneal dialysis, *Rev. Infect. Dis.* 13 (1991) 376–378.
- [22] J. O'Connor, G. Nimmo, R. Rigby, et al., Algal peritonitis complicating continuous ambulatory peritoneal dialysis, *Am. J. Kidney Dis.* 8 (1986) 122–123.
- [23] A. Gibb, R. Aggarwal, C. Swainson, Successful treatment of *Prototheca* peritonitis complicating continuous ambulatory peritoneal dialysis, *J. Infect.* 22 (1991) 183–185.
- [24] C. Pérez Melón, M. Camba, A. Tinajas, et al., Peritonitis por *Prototheca wickerhamii* en pacientes en diálisis peritoneal [Spanish], *Nefrología* 27 (1) (2007) 81–82.
- [25] T. Sykora, J. Horakova, D. Buzzasyova, et al., Protothecal peritonitis in child after bone marrow transplantation: case report and literature review of paediatric cases, *New Microbes New Infect.* 2 (2014) 156–160.
- [26] C. Heney, M. Greeff, V. Davis, Hickman catheter-related protothecal algemia in an immunocompromised child, *J. Infect. Dis.* 163 (1991) 930–931.
- [27] A. Kunová, T. Kollár, S. Spáňik, et al., First report of *Prototheca wickerhamii* algemia in an adult leukemic patient, *J. Chemother.* 8 (2) (1996) 166–167.
- [28] M. Narita, R. Muder, T. Cacciarelli, et al., Protothecosis after liver transplantation, *Liver Transplant.* 14 (2008) 1211–1215.
- [29] I. Żak, T. Jagielski, S. Kwiatkowski, et al., *Prototheca wickerhamii* as a cause of neuroinfection in a child with congenital hydrocephalus. First case of human protothecosis in Poland, *Diagn. Microbiol. Infect. Dis.* 74 (2) (2012) 186–189.
- [30] Y. Li, Y. Zheng, G. Ning, et al., A case report of prototheca meningitis in China, *Pharmacoeconomics* 1 (2) (2016) 108–110.
- [31] V. Iacoviello, P. DeGirolami, J. Lucarini, et al., Protothecosis complicating prolonged endotracheal intubation: case report and literature review, *Clin. Infect. Dis.* 15 (1992) 959–967.