Singapore Med J 2007; 48(3) : 256

Delayed fungal infection following augmentation mammoplasty in an immunocompetent host

Tian H H, Tan S M, Tay K H

ABSTRACT

We report an unusual case of infection of a breast implant with *Trichosporon* spp. in an immunocompetent host. There has only been one other reported case in the published literature. The patient was a healthy 27-year-old woman who presented with pain and swelling 17 months after augmentation mammoplasty with a saline implant. Ultrasound-guided aspiration of the effusion surrounding the implant yielded *Trichosporon* spp. on culture. Oral therapy with fluconazole was commenced, and the implant was salvaged. The nature of this fungi, its mode of transmission and treatment are reviewed. The implications and management of implant infections are also discussed. Physicians should be aware that infection may be delayed, and cultures should be performed for aerobic and anaerobic organisms as well as acid-fast bacilli and fungi. Prompt and appropriate antimicrobial treatment may arrest the infection, sparing the patient the morbidity of a surgical drainage or implant removal.

Keywords: augmentation mammoplasty, fluconazole, fungal infection, mammoplasty complications, saline implant, *Trichosporon* spp

Singapore Med J 2007;48(3):256–258

INTRODUCTION

Implant augmentation mammoplasty is one of the most frequently-performed cosmetic surgical procedures worldwide. Infection remains the most feared, if not the most serious complication of an augmentation, and rates vary from 0% to 6.1%.[1,2] Both bacteria and mycobacteria are the usual organisms implicated.[2] Rarely, the culprit is a fungus. The incidence of nosocomial fungal infections rose from 2.0 to 3.8 infections per 1,000 discharges between 1980 and 1990 in the United States.[1] Accompanying this increase is the recognition that yeasts previously thought innocuous are capable of damaging the human body. The spectrum of infection can range from subclinical to fulminant abscess formation with subsequent implant loss.

We describe a case of an infection of a saline breast implant caused by *Trichosporon* spp. A search of the literature using MEDLINE and the National Library of Medicine databases yielded only one other previously-reported case.[3]

CASE REPORT

A healthy 27-year-old Vietnamese woman underwent bilateral transareolar, submammary saline-implant augmentation while living in Vietnam. She did not experience any postoperative complication. 17 months post-procedure, she presented with progressive swelling of her right breast over a three-week duration. This was associated with a dull discomfort and skin erythema. There was no history of trauma to the chest wall nor were there recent systemic infections. She was given a course of amoxicillin and clavulanate by her physician, but her symptoms worsened.

On examination, the right breast was swollen and tender, with warmth and erythema of the overlying skin. She had a low-grade fever as well as generalised malaise. Ultrasonography (US) demonstrated periprosthetic

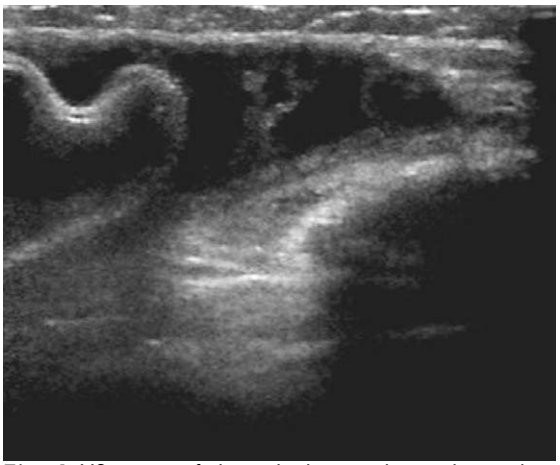

Fig. 1 US image of the right breast shows the implant surrounded by fluid with hyperechoic sediments.

Department of General Surgery, Changi General Hospital, 2 Simei Street 3, Singapore 529889

Tian HH, MBBS, MMed, MRCSE Resident

Tan SM, MMed, FRCSE, FAMS Consultant

Tay KH, FRCSE, FICS, FAMS Senior Consultant and Head

Correspondence to:
Dr Roger Tian Ho Heng
Tel : (65) 6850 3551
Fax : (65) 6260 1709
Email: roger_tian@cgh.com.sg

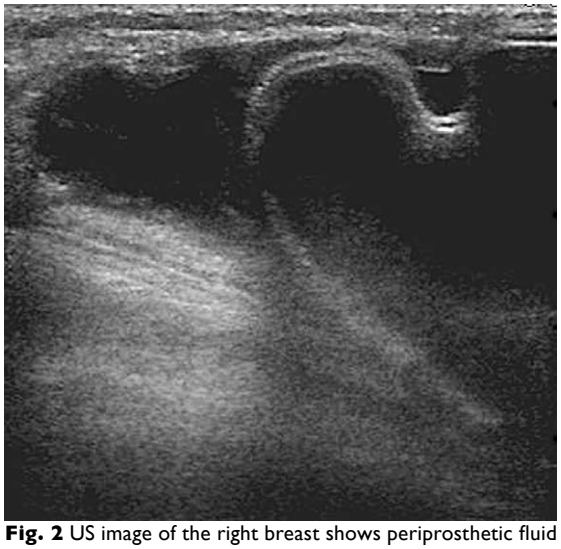

Fig. 2 US image of the right breast shows periprosthetic fluid around the lateral edge of the implant.

fluid in her right breast (Figs. 1 & 2). There was no evidence of implant rupture. An ultrasound-guided needle aspiration was performed, and the cloudy-yellow, odourless aspirate was sent for aerobic, anaerobic and acid-fast cultures. She was given an empirical course of ciprofloxacin and cloxacillin.

Blood markers of infection and immunocompromise were also despatched. These were all within normal ranges (white cell count, 7,900 × 10^3/μL; T-cell CD4, 39.2%; T-cell CD8, 20.2%; CD4/CD8 ratio, 1.90) except for a raised C-reactive protein of 14.2 mg/L. Tests for diabetes mellitus were also negative. Initial microbiological cultures were negative until three weeks later, when *Trichosporon* spp. was isolated. She was promptly started on oral fluconozole 400 mg once daily. After two weeks of treatment, her breast swelling and pain resolved. The amount of periprosthetic fluid detected on US also decreased over the next few weeks. C-reactive protein levels had normalised to 3 mg/L one month after initiation of antifungal therapy. She tolerated the therapy well and the implant was not removed. She remained well on follow-up at six months.

DISCUSSION

Trichosporon is a common skin contaminant and is generally non-pathogenic.[3] It also inhabits the respiratory and gastrointestinal tract and has been reported to cause both superficial and deep infections.[4] In the immunocompetent, it causes innocuous infections such as white piedra and onychomycosis. In immunocompromised patients, it can be life-threatening, causing deep localised or disseminated infection.[4-6] Predisposing conditions include haematological malignancies, neutropenia, treatment with corticosteroids, diabetes mellitus, human immunodeficiency virus infection, and the presence of prosthetic heart valves and central venous catheters.[3,4] Cell-mediated immunity is suppressed in these patients, thus increasing the potential for infection with otherwise less pathogenic organisms.

Histologically, *Trichosporon* spp. infection is characterised by the presence of hyphae, pseudophyphae, blastoconidia, and arthroconidia. Arthroconidia are the most typical microscopical feature of this genus; they are unicelluar and usually cubical, barrel-shaped or elongated. As with our case, the illness is usually insidious, and a fungal aetiology is seldom suspected until it is identified in culture or biopsy specimens.[3] Cytological examination of the aspirate, which was not performed in our patient as a fungal aetiology was not suspected initially, would have helped in identifying the causative organism. It would also show the presence of multinucleated giant cells, which usually accompany fungal infections.

The pathogenesis of the infection in our patient was not well understood. Although she was immunocompetent and did not have any predisposing medical conditions, the presence of a foreign body such as an implant could have lowered the threshold for infection.[7] Possible portals of entry include the skin, respiratory or gastrointestinal tract. Both in vivo and in vitro studies, using saline implants, have demonstrated that bacteria and fungi can survive and reproduce in a restricted implant environment for extended periods of time.[8] Implant contamination either before or during the procedure could also have occurred. Cross-contamination of surgical and anaesthetic equipment have also been reported by various authors.[5,6] After transient fungaemia, the organism may have localised to the site of the implant. As the aspiration was performed under aseptic conditions on two separate occasions, it is unlikely that the culture results were due to skin contamination.

Treatment strategies for implant infection are evolving and depend largely on the severity of the infection. The aim of treatment is to eradicate the infection, prevent its progression with subsequent abscess formation, soft-tissue loss and implant extrusion. Repeated bouts of infection may cause a change in capsular biology, eventually increasing the risk of capsular contracture. Recommendations include antimicrobial treatment alone and/or device removal with delayed replacement of the implant.[9] Systemic antifungal agents that are available for treatment of yeast infections include the polyenes (amphotericin B, 5-fluorocytosine) and azoles (fluconazole, itraconazole, ketoconazole). Their efficacy against unusual yeasts is unknown because insufficient cases have been reported to provide useful guidelines.[10] Many strains

of *Trichosporon* spp. are resistant to both amphotericin B and 5-fluorocytosine.[4-8,10,11] For susceptible strains, high doses are often required. The activity of the azoles against *Trichosporon* spp. is also variable, but appears to be superior to amphotericin B.[4] Fluconazole has been used effectively in the treatment of trichosporonosis,[2,3,9] although reports of clinical resistance have been documented.[10] Both itraconazole and posaconazole have lower minimal inhibitory concentrations than fluconazole, and currently, the most active agent appears to be voriconazole.[4] The optimal duration of antifungal therapy for trichosporon infection is as yet unknown. In the current literature, recommendations vary from several weeks to six months. The toxicity of the agent, the pathogenicity of the organism, as well as the immune status of the individual need to be considered when deciding on treatment length. Combination therapy has also been advocated, with amphotericin B and fluconazole demonstrating enhanced antifungal activity in vivo.[4]

Physicians managing patients with breast implants should be aware that infection can occur even after the immediate postoperative period. When such patients present, specimens should be collected and dispatched for aerobic, anaerobic, fungal and acid-fast cultures. Pending a definitive microbiological diagnosis, broad spectrum antibiotics should be started, as the commonest aetiology is bacterial. If the causative organism is mycobacteria or fungi, the patients should also undergo screening for immunosuppression and underlying medical conditions such as diabetes mellitus. Prompt and appropriate antimicrobial treatment can salvage the implant and spare the patients the morbidity associated with surgical drainage or implant removal.

REFERENCES

1. Beck-Sague C, Jarvis WR. Secular trends in the epidemiology of nosocomial fungal infections in the United States, 1980-1990. J Infect Dis 1993; 167:1247-51.
2. Freedman A, Jackson IT. Infections in breast implants. Infect Dis Clin North Am 1989; 3:275-87.
3. Reddy BT, Torres HA, Kontoyiannis DP. Breast implant infection caused by Trichosporon beigelii. Scand J Infect Dis 2002; 34:143-4.
4. Fleming RV, Walsh TJ, Anaissie EJ. Emerging and less common fungal pathogens. Infect Dis Clin North Am 2002; 16:915-33.
5. Cawley MJ, Braxton GR, Haith LR, et al. Trichosporon beigelli infection: experience in a regional burn center. Burns 2000; 26:483-6.
6. Walsh TJ. Trichosporonosis. Infect Dis Clin North Am 1989; 3:43-52.
7. Jennings DA, Morykwas MJ, Burns WW, et al. In vitro adhesion of endogenous skin microorganisms to breast prostheses. Ann Plast Surg 1991; 27:216-20.
8. Young VL, Hertl MC, Murray PR, et al. Microbial growth inside saline-filled breast implants. Plast Reconstr Surg 1997; 100:182-96.
9. Spear SL, Howard MA, Boehmler JH, et al. The infected or exposed breast implant: management and treatment strategies. Plast Reconstr Surg 2004; 113:1634-44.
10. Hazen KC. New and emerging yeast pathogens. Clin Microbiol Rev 1995; 8:462- 78.
11. Warnock DW. Fungal infections in neutropenia: current problems and chemotherapeutic control. J Antimicrob Chemother 1998; 41 Supp D:95-105.