Multidisciplinary Treatment of Persistent Nontuberculous Mycobacterial Spinal Hardware Infection with a Pedicled Superior Gluteal Artery Perforator Flap

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Abstract

Nontuberculous mycobacterial hardware infections are extremely challenging to treat. Multidisciplinary care involving removal of infected hardware, thorough debridement, and durable soft tissue coverage in conjunction with antibiotic therapy is essential for successful management. This case report presents a patient with chronic mycobacterial spinal hardware infection that underwent successful treatment with aggressive serial debridements and reconstruction with a large pedicled superior gluteal artery perforator flap coverage.

Keywords
► SGAP flaps
► implant-associated infections
► Mycobacterium massiliense
► debridement

Introduction

Nontuberculous mycobacterial (NTM) infections are defined as infections of mycobacterial species other than Mycobacterium tuberculosis complex and Mycobacterium leprae. Increasing incidence of NTM infections has been reported globally. These infections are initially thought to be environmentally acquired, but recent studies suggest local spread potential with fomite and aerosols.1 We presented a case of chronic NTM infection of spinal hardware, which failed the initial wide local excision (WLE) procedure and eventually underwent successful treatment with multidisciplinary approaches.

Case

This is the case of a 77-year-old male patient with a history of chronic Mycobacterium abscessus and later Mycobacterium massiliense infection of L2 to L5 posterior spinal fusion and L2 to L3 lateral interbody cage hardware. The patient’s past medical history was significant for coronary artery disease treated with percutaneous transluminal coronary angioplasty, hyperlipidemia, chronic renal disease, nephrolithiasis, and hypertension. The initial posterior spinal fusion and interbody cage procedure were performed outside the United States. The patient presented 3 months later with pruritus...
and a draining sinus tract in the posterior wound (►Fig. 1). This failed to improve with oral cephalexin and ciprofloxacin, and the patient had a brief course of IV antibiotics which were discontinued due to the development of drug-induced hepatitis and pancytopenia. The patient underwent WLE and debridement of the deep tissue. Unable to be closed due to the persistent mycobacterial infection on deep tissue biopsies, his wound was managed with negative pressure wound therapy.

With limited expertise locally, the patient presented to our multidisciplinary mycobacterial soft tissue team. After extensive discussion, he elected to have his spinal hardware removed, the wound managed with multiple debridements to obtain source control of the infection, and definitive closure with a soft tissue flap. Throughout this process, he was treated with systemic antimycobacterial drugs. The patient underwent a total of 16 irrigation and debridement procedures with amikacin antibiotic bead placement and negative pressure wound therapy. Since establishing care at our institution, serial wound cultures have been intermittently negative. However, due to failure to obtain consistently negative cultures and having reached the limits of safe debridement, we elected to reconstruct the wound after a new appliance was placed. The patient would be maintained on suppressive antibiotics for a year.

The combined procedure was performed by the orthopaedic spine and plastic surgery teams. The spine team removed the L2 and 3 interbody spacer, judiciously debrided the area, and performed a lateral fusion of L2 and 3 and L4 and 5, using tricortical structural allograft and bone morphogenic protein (BMP) via a lateral approach. The posterior wound was debrided further with revision of the posterior instrumentation with pedicle screws placed at L1 and S1 and bilateral iliac bolts, and posterior instrument spinal fusion of L1 to the ilium with BMP, demineralized bone cortical fibers, and allograft cancellous bone. Antibiotic beads were placed into the wound bed prior to closure.

We then defined the extent of the defect and evaluated the reconstructive options. The defect was full thickness down to the level of bone and fascia and measured 196 cm². A preoperative CT scan with a specific focus on the area of the gluteal vasculature was performed. It showed a prominent right-side perforator (►Fig. 2) outside the zone of injury and close enough to serve as the pivot point and nutrient vessel. The flap was designed long enough to reach after transposition. This would be a 10 × 35 cm superior gluteal artery perforator (SGAP) flap (►Fig. 3). If the 180-degree transposition compromised the flap, the flap could be harvested as a free perforator flap and anastomosed to the superior gluteal artery.

The flap was incised and elevated in the subfascial plane to the perforator identified on the CT scan and confirmed with Doppler signal. Once the perforator was identified, the fibers of the gluteus muscle were split to facilitate rotation, but the distal end of the flap did not reach the defect. The muscle fibers were split further (►Fig. 4), and the perforator was brought closer to the defect. At this point, the flap easily covered the defect, and the distal end was deepithelialized and inset to fill the dead space laterally. The perfusion to the flap was confirmed with SPY angiography, and the flap showed no clinical evidence of venous compromise (►Fig. 5).

The patient’s postoperative recovery was uneventful, and the patient was discharged on postoperative day 19. Initial
intraoperative cultures were positive for *M. abscessus*. However, multiple subsequent isolates were positive for subspecies *M. massiliense*, with phenotypic susceptibility to macrolides. The patient was, therefore, started on imipenem, amikacin, bedaquiline, and clofazimine. Amikacin was discontinued, and the patient was started on azithromycin. Eravacycline was prescribed later but discontinued secondary to drug-induced hepatitis, edema, and nausea. This was replaced with a course of ceftaroline.

Before discharge, the patient was extensively counseled regarding the range of motion limitations, including off-loading, turning frequently, no direct pressure on the flap, laying on his left side, and maintaining 45 degrees of flexion when sitting or in bed. He began physical therapy, focusing on upper back strengthening, core strengthening, and overall reconditioning at 3-month postoperation.

Twenty-four months after his reconstructive surgery, the patient is doing well. He has been off antibiotics for at least 1 year and shows no signs of infection. Recently, the patient had painful iliac bolts removed, and cultures obtained in the operating room showed no infection. His SGAP flap continues to provide well-vascularized, durable, and aesthetic coverage (*Fig. 6*). Most importantly, the patient has returned to his preoperative level of activities of daily living without significant debility.
Discussion

Defects in the lumbosacral region are common but challenging conditions that are frequently treated with local muscle flaps like the paraspinous or gluteal flaps by reconstructive surgeons. In this case, these flaps were not options due to the invasive nature of the infection and the need for aggressive debridement, which compromised these local muscle flaps. Recently, perforator flaps have been used for pressure ulcers, pseudomeningoceles, and exposed hardware. The SGAP flap is considered an ideal option, as it preserves muscle function in the gluteal region while maintaining adequate perfusion via a single perforator. This permits a greater arc of rotation in comparison to other local flap options.

Nontuberculous mycobacterial infections involving hardware have been reported in the literature, and they pose a significant treatment challenge. To treat these infections adequately, patients often require prolonged antibiotics with risks of adverse reaction, prolonged hospitalization, periods of debility, multiple operative interventions, and need for rehabilitation. In most cases, patients ultimately require hardware removal for successful treatment of their infection. NTM infections have been reported to affect both immunocompetent and immunocompromised patients. They should be considered when patients present with prolonged infections, chronic wounds or draining sinuses, hardware failure, and persistent systemic symptoms of infection.

Chronic hardware infection is a challenging condition to treat. Successful treatment often requires a multidisciplinary approach with multiple surgical specialties or subspecialties, along with infectious disease, radiology, and physical and occupational therapy. In cases such as ours, biofilms inherent in implant-associated infections result in the persistence of bacteria, despite aggressive serial debridements. Therefore, the infected hardware is ideally explanted and replaced in order to facilitate successful extirpative surgery and antibiotic penetration.

Obtaining antimicrobial drug sensitivity is also crucial in the treatment of mycobacterial infections. Different combination therapies for NTM infection have been described, with a median duration of 14 months. In our case, 3 months of continued combination antibiotic therapy after flap coverage and a year total of suppressive antibiotics proved successful.

Flap coverage can be instrumental in infection control and promoting chronic wounds to heal. Flap coverage has long been viewed as critical in open fracture treatment and deep wound infection treatment, as they can eliminate dead space and provide vascularization to poorly perfused areas. In the case presented above, SGAP flap coverage provided definitive, durable vascularized tissue to a deep, large wound in an anatomically challenging region, while allowing for antibiotic penetration and eradication of long-standing NTM hardware infection.

Chronic mycobacterial infections of spinal hardware can be challenging to treat and often require a multidisciplinary approach, with long-term antibiotics, extirpative surgery, hardware removal and/or replacement, and flap coverage for definitive treatment. In this case, the patient underwent all of the above. This patient has now been NTM infection free for more than 2 years without sacrificing mobility or function.

Authors’ Contributions

Conceptualization: T.W.C.
Project administration: K.R.T., T.W.C.
Writing – original draft: K.R.T., J.H.Y.

Ethical Approval

Patient consent was obtained for publication.

Conflict of Interest

The authors declare no conflict of interest for this article and none of the authors are members of the editorial board.

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