

Surgical site infection following spine surgery

Kshitij Chaudhary¹, Gautam Zaveri¹

Abstract

Surgical site infection in spine surgery is a devastating complication not only from a medical perspective but also from an economic standpoint. Early detection and debridement are necessary, especially if one has to retain the instrumentation. In late infection, if the fusion is confirmed, implant exit can be performed to better treat the infection. The current review focuses on the decision making in Surgical site infections in Spine Surgery

Keywords: Surgical site infection, Spine Surgery, implant

Introduction

Surgeons have always dreaded surgical site infection (SSI). Not only does an SSI compromise the outcome of surgery, but it can result in significant morbidity, long term disability and even death.

Although SSIs are uncommon events following routine spine surgery, the incidence is increased manifold with revision surgeries and surgeries that involve spinal instrumentation. As our surgical horizons expand, we are operating on more complex pathologies, even in the elderly and the immunocompromised, who until a couple of decades ago, would not have been considered suitable for surgery. This review article briefly summarizes the problem of surgical site infection following spine surgery. A few case illustrations are presented along with this review.

Definition

SSIs are defined as infections occurring

within 30 days of surgery or within one year of insertion of any foreign bodies, like spinal instrumentation[13]. SSIs are categorized by the depth of layers involved as 1) superficial, 2) deep incisional, or 3) organ and surrounding space[14]. Early SSI (<3 months) are typically due to wound contamination from the index procedure while late SSI (>3 months) usually results from hematogenous seeding of bacteria from a distant site, like UTI or rarely it can result from local contamination of the wound by a low virulence organisms like *Propionibacterium acnes*. The rate of postoperative SSI in spine surgery average 2.1% [1]. However, the rate varies depending on several risk factors that could be patient, pathology or surgery-related.

3. Risk factors

3.1. Patient-related

1) Diabetes: Diabetes mellitus (DM) is a prevalent condition amongst elderly patients undergoing spine surgery. Postoperative blood glucose of > 200 mg/dL and preoperative HbA1C more than 7% are risk factors for SSI [15].

2) Obesity: The skin-to-lamina distance rather than BMI have been found to directly correlate

with postoperative wound infection. A thicker layer of fat increases the dead space and also requires more forceful retraction of tissues which can lead to necrosis [16]. On the other hand, some studies have found a positive correlation between BMI and SSI rates. A BMI of more than 30 puts the patient at higher risk of SSI [17].

3) Smoking: In addition to other negative effects, smoking doubles the risk of SSI [18]. Smoking cessation should be strongly encouraged and many surgeons are hesitant to offer elective spine surgery to patients who refuse to quit.

4) Malnutrition: Albumin levels of < 3.5 g/dL and total lymphocyte count < 1500 cells/mm³ are risk factors for postoperative SSI [19]. Adequate preoperative nutrition is vital for successful wound healing. Immunocompromised patients would also be at a higher risk of SSI [2].

5) MRSA carrier: Nasal MRSA carriers are at a higher risk for SSI. It is important to identify such patients preoperatively and treat them with a decolonization protocol using 2% chlorhexidine and 2% intranasal mupirocin. This protocol has been shown to significantly lower SSI rates [20]. A patient who has had long hospital admissions in the past or those undergoing instrumentation should have

¹Sir HN Reliance Foundation Hospital and Research Center, Mumbai

²Department of Spine Surgery, Jaslok Hospital & Research Centre

Address of Correspondence

Dr. Kshitij Chaudhary

Sir HN Reliance Foundation Hospital and Research Center, Mumbai

E-mail: chaudhary.kc@gmail.com



Figure 1: a) Preop MRI, b) 7 days later following wound infection, c) 1 month later after a month of empirical antibiotics d) 3.5 months after organism-specific antibiotics



Figure 2: Four years after debridement and fusion.

nasal swab testing done to identify MRSA carrier status.

3.2. Surgery-related risk factors

The risk of SSI varies greatly depending on the surgical time and the complexity or invasiveness of the surgical procedure [5]. Minimally invasive surgery carries a lower risk, while multiple levels of surgery with instrumentation carry a higher risk of wound infection. Posterior spinal approaches have a relatively higher risk of wound infection compared to anterior spinal approaches. Other risk factors include increased operating room traffic, poor draping techniques, C-arm contamination and inadequate intraoperative irrigation [1]. Preoperative antibiotics have to be given within 60 minutes before the incision. Failure to

identify MRSA carrier status would lead to a wrong choice of preoperative antibiotic prophylaxis.

3.3. Pathology related

It is well known that surgery for certain pathologies carries a higher risk of SSI. Spinal cord injury patients who undergo instrumentation and fusion are particularly at high risk and the rate of SSI is about 9.4% [21]. In paediatric spine surgery, neuromuscular scoliosis or meningocele patients have a higher risk for SSI [4].

With this short introduction, we present four case scenarios of surgical site infection following spine surgery and describe how they were treated.

4. Case examples

4.1. Case 1

A 52-year-old woman underwent a left-sided L5-S1 discectomy in a nursing home (Figure 1a). Seven days later she had wound discharge and fever. A wound wash and debridement were done at 11 days after the index surgery (TLC 12000, CRP 73, ESR 74). No organisms were isolated (Figure 1b). She was treated with intravenous meropenem and linezolid for 10 days.

At that point, the patient's back pain was better, the wound was clean (TLC 10000, CRP 28, ESR 128). The IV antibiotics were stopped and oral cefixime was started. She gradually started redeveloping back pain and one month following the wound debridement, she presented to us with severe back pain and was unable to turn in bed, sit or walk. Her wound had healed. She was afebrile and had no neurological complaints (TLC 8000, CRP 17 and ESR 128) (Figure). MRI showed florid discitis (Figure 1c).

ACT guided biopsy of the L5-S1 disc was performed following which empirical antibiotics (Cefoperazone + Sulbactam) in consultation with the infectious disease specialist were started. The biopsy sample grew *Burkholderia cepacia*. Blood cultures were negative. This is a hospital-acquired infection and this organism is not found in the environment. Typically, it resides and grows in contaminated water in hospitals. Hence, it was concluded that the infection was following a wound

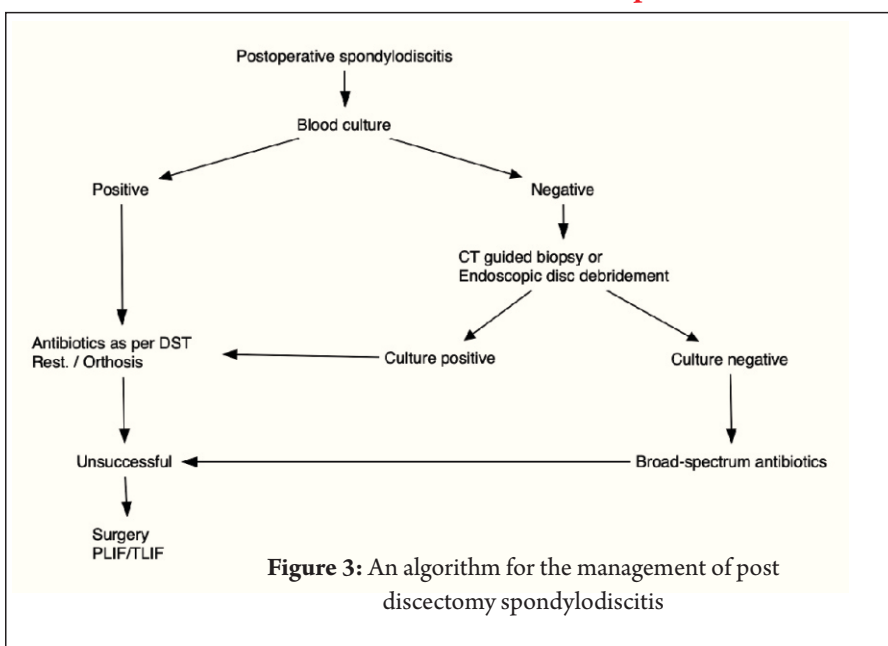


Figure 3: An algorithm for the management of post discectomy spondylodiscitis

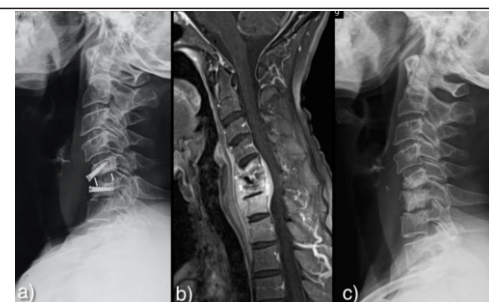


Figure 4: Case 2 - infected ACDF implant without esophageal injury



Figure 5: a) L3 pedicle screws appear loose with haloes, b) L2-3 spondylodiscitis c) left side psoas abscess at the level of the L2-3 disc

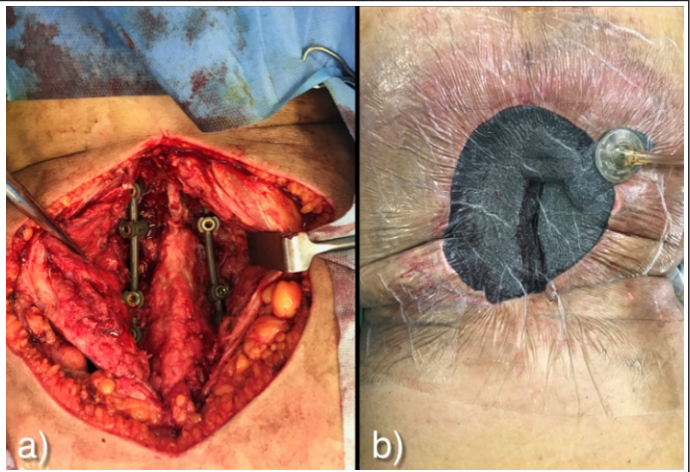


Figure 6: Negative pressure wound therapy (Wound VAC - vacuum assisted closure)

contamination at the time of the index surgery. Once drug sensitivity testing was available the antibiotic plan was changed to iv Ceftazidime and oral Ciprofloxacin for 6 weeks. PICC line was inserted for long term iv antibiotic care. 2 weeks later, her blood reports showed TLC 8000, CRP 5.4, ESR 96. Symptomatically she improved initially with the WBC counts dropping to 6700, CRP 4.7 and ESR 52 at the end of 6 weeks of IV antibiotics. Then oral Ciplox and Septran DS was given for additional 4 weeks. The WBC counts were 6500, CRP- 1.6 and ESR was 34. The back pain had reduced and she was ambulatory. After oral antibiotics were stopped, the back pain returned and the WBC and CRP started climbing up. Repeat MRI showed persisted discitis (Figure 1d). Hence, L5-S1 debridement

and fusion was performed and osteomyelitis was treated again with iv antibiotics for 6 weeks followed by orals for 6 weeks until the CRP normalized. This gave her relief of symptoms and she is now 4 years since the last surgery (Figure 2).

4.1.1. Take-home points

1. Empiric antibiotic treatment may not always work. Without knowing the organism, oral cefixime was the wrong choice in this patient. Alternatively, empiric long-term broad-spectrum antibiotics can lead to resistance.
2. Obtaining a microbiological diagnosis by biopsy is vital to the choice of the most appropriate antibiotic. The yield of the biopsy can be negatively affected by prior antibiotics [3]. It is always advisable to

avoid administering the antibiotics until a sample has been obtained for microbiological examination. Alternatively, disc debridement can be done using the endoscopic technique.

3. In patients with pyogenic infective spondylodiscitis, long-term antibiotics (8-12 weeks) via a PICC line are recommended [3]. Intravenous antibiotics are administered for the initial 4-6 weeks. Subsequently, oral antibiotics are administered for another 4-6 weeks. At that point, clinical and hematological improvement must be accompanied by a significant improvement in the bone marrow edema, reduction of hyper-intensity signal within the disc space and endplate erosions. Abscesses should have reduced in size or resolved. Only then should the antibiotics be stopped. The

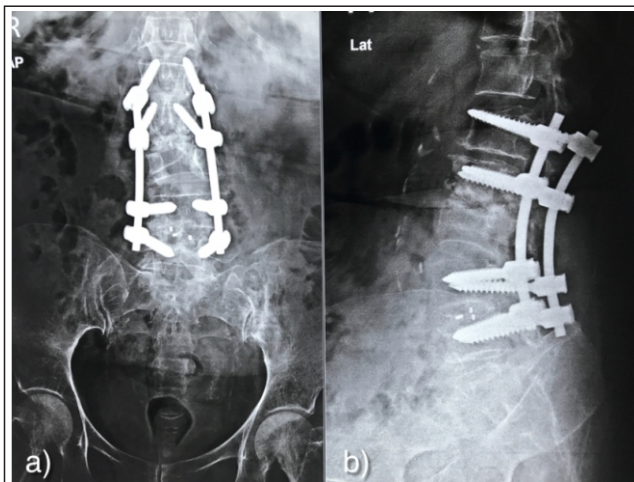


Figure 7: Healing after repeated debridements. Implants were retained and the patient went on to have a successful fusion.

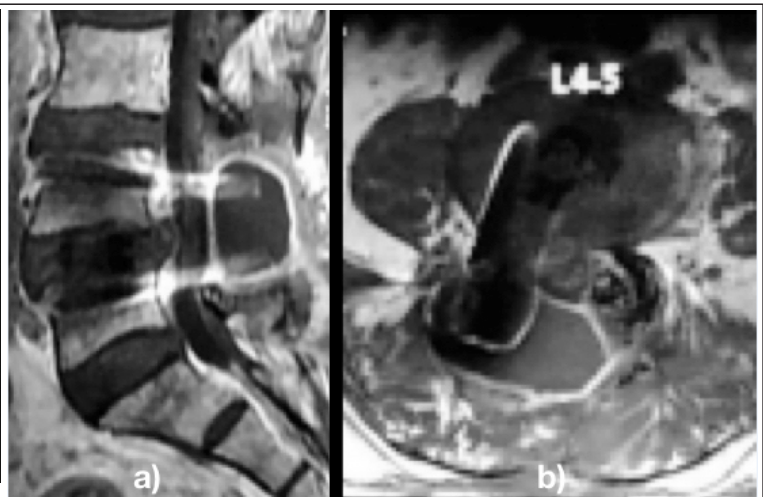


Figure 8: Pus collection around the implants on MRI

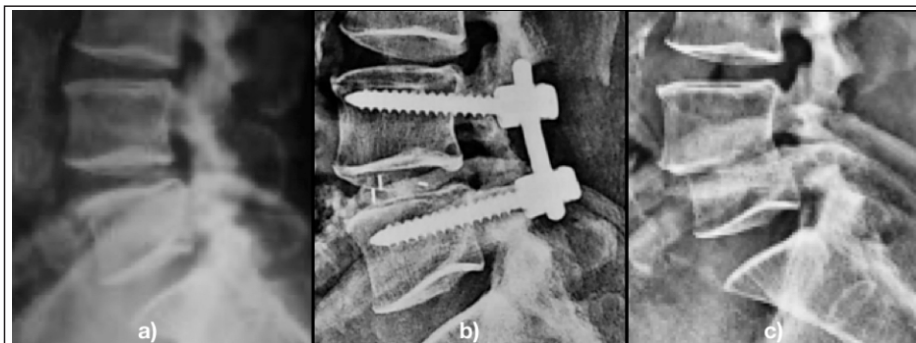


Figure 9: a) Preoperative X-ray, b) after a wound wash with implants in situ, c) collapse of the disc after implant removal

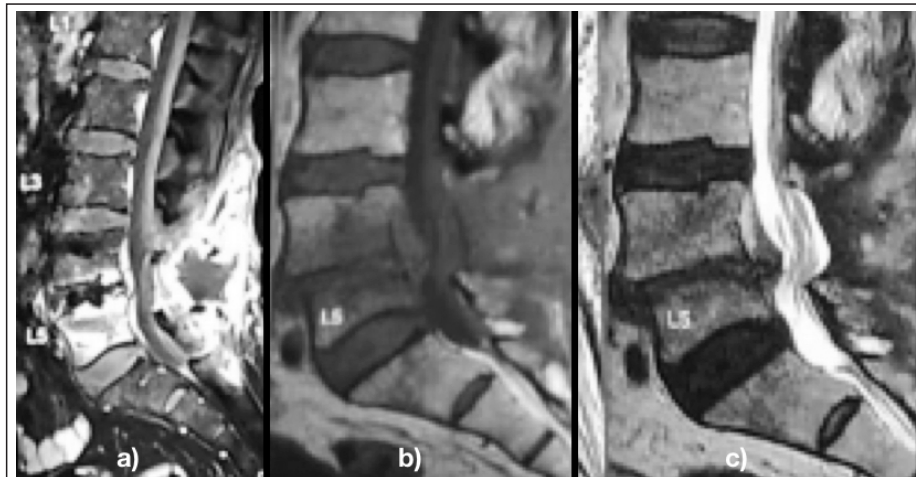


Figure 10: Sequential MRIs a) MRI 4 months from index surgery after repeated debridements now shows disc space infection with loosening of cage and vertebral body involvement b) MRI after removal of implant and cage

short duration of antibiotics often suppress the infection but do not completely eradicate it, resulting in a flare-up once the antibiotics are discontinued.

4. Patients with unrelenting pain, significant vertebral destruction (late presentation), progressive neurologic deficits and lack of adequate response to antibiotics may need surgery for debridement, fusion and instrumented stabilization⁶ (Figure 3).

4.2. Case 2

A 57-year-old woman was operated for radiculopathy with C5-6 anterior cervical decompression and fusion (ACDF). She presented to us 3 months later with increasing neck pain, dysphagia, and left arm C6 radiculopathy of 1-month duration. Recently, she had also developed fever with chills, and her dysphagia had worsened to a point where

she could not eat. On examination, there was redness and tenderness over the scar of the surgery (left-sided anterior cervical approach) and swelling and tightness in the neck. A sinus with a minimal discharge was noted. Range of motion was restricted. She had no focal neurological deficits. The Xray of the cervical spine revealed that a PEEK cage with an integrated screw system was used for ACDF (Figure 4a). Loosening of the screws with increase in the prevertebral soft tissue shadow was noted. MRI showed an increase in marrow signal at C5, C6 and C7 with an epidural abscess and fluid collection in the prevertebral area (Figure 4b).

Infection following anterior cervical spine surgery is rare. Hence, it was suspected that the infection was secondary to contamination of the surgical site due to esophageal injury from the screws that had backed out. An

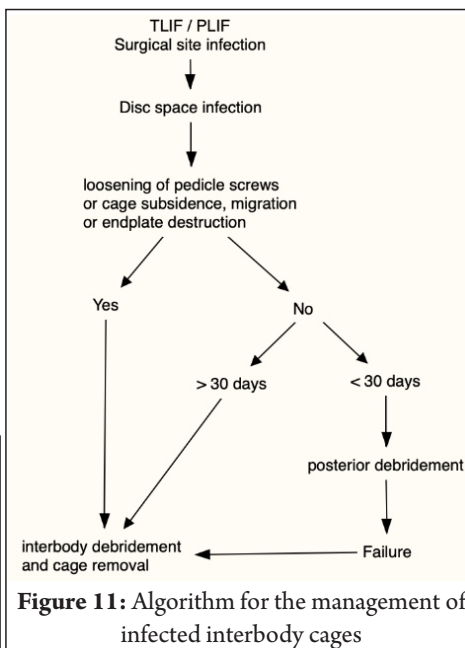


Figure 11: Algorithm for the management of infected interbody cages

esophagoscopy was performed which ruled out an esophageal injury. The patient's total WBC count was 12,000 with 88% neutrophils. ESR was 63. The patient underwent revision anterior cervical surgery via the same incision. Preoperative antibiotics were withheld to increase the yield of intraoperative cultures. An access surgeon was used to dissect the neck up to the implant. The sinus was traced all the way up to the implant. Intraoperatively, an endoscope was passed into the esophagus. The light within the esophagus helped the access surgeon to avoid injuring the hypopharynx. After reaching the implant the screws and the PEEK cage were removed. Fluid was sent for cultures. Following this, intravenous vancomycin was administered. Iliac crest structural bone graft was inserted in the disc space and no further implants were used. Post-operative course was uneventful. The cultures came back as pseudomonas aeruginosa which was pan-susceptible. Infectious disease was consulted and a 6 weeks course of ceftazidime was given via a PICC line followed by oral levofloxacin for another 4 weeks. The patient went on to heal well.

4.2.1. Take-home points

5. Infections following anterior cervical

approach are very rare because of the vascularity of the neck and for the fact that the tissue dissection in this approach follows surgical planes without any muscle or tissue damage. Hence, when wound infection is suspected following anterior cervical approach one should have a high degree of suspicion for esophageal injury [7].

6. Preoperative esophagoscopy can rule out esophageal injury. One should seek the help of access surgeon for surgical exposure as there is an increased risk of esophageal injury during the revision [8].

7. A loose implant within an infected wound must always be removed. However, maintenance of spinal stability is vital to alleviate pain, prevent spinal deformity and combat infection. Restoration of spinal stability may require insertion of a fresh implant.

4.3. Case 3

A 66-year-old woman was admitted to the hospital with presumed urosepsis (E Coli) complicated by acute kidney injury (AKI) and liver dysfunction. She had been operated for lumbar spine surgery (L3 to L5 fusion) 2 years ago and did well after the surgery. She complained of severe low back pain starting 8 days before the current hospital admission. She had been unable to walk or sit up in bed due to pain. Neurological examination revealed a focal neurological weakness in the left leg (left quadriceps- grade 3/5 and ankle dorsiflexors- 4/5).

On admission to the hospital for the urosepsis, she was started on IV meropenem and the general condition was stabilized. Persistent bacteremia, with a total WBC count of 21000 and complaints of severe back pain led to a search for an alternative source of infection. X-Rays of the lumbar spine showed loosening of screws and an MRI revealed abscess formation around the implant and within the psoas muscle with L2-3 discitis (adjacent segment to the

previous fusion). Patient was taken up for surgery to have source control as she was in sepsis. L2-3 disc debridement was done with evacuation of the abscess. The posterior stabilization was extended to L1 with the old implants being replaced with new screws of a larger diameter. Same organism (E. Coli) was isolated from the intraoperative samples. The iv meropenem was continued. Her back pain reduced and the WBC count reduced as she came out of sepsis with resolution of the kidney injury. She continued to have wound discharge for which an incisional VAC was applied, however, for 2 weeks the discharge continued and she was taken up again for wound debridement. This time a wound VAC was applied. Five days later the VAC was changed, however, the wound was still appearing unhealthy. Hence, mechanical debridement was done and the wound VAC was placed again. After 4 days, with plastic surgery help, the wound VAC was removed and wound closure was done. She was continued on iv meropenem and discharged home with a PICC line.

One month later she was readmitted with persistently high WBC counts and CRP in spite of two months of iv meropenem. The wound had healed completely, her back pain had resolved. However, the MRI still showed a large psoas abscess. A CT guided drainage of the abscess was done. The culture again grew E coli. Infectious disease consultant started injectable Ertapenem once daily. Over the next one month, she improved. The WBC count normalized and the CRP showed a reducing trend. Antibiotics were continued for another 6 weeks until the CRP normalized. Eventually, the infection was brought under control. At 2 years from these events, she is now symptom-free and doing well.

4.3.1. Take-home points

1. A late spinal infection is usually secondary to the spread of infection from another source [3]. In this patient, the

urinary tract infection resulted in bacteraemia with secondary involvement of the surgical site.

2. Since this patient was in sepsis with organ failure, source control of the infection was warranted. This involves local control of infection with radical debridement and restoration of spinal instability along with systemic control using intravenous antibiotics based on the drug sensitivity. Loose implants must be removed and replaced if possible to provide adequate stability. In wounds with implants or wounds that are not clean enough for closure and when there is a fulminant infection, rather than closing the wound after debridement, the author prefers to utilize a vacuum-assisted closure (VAC) system through a special sealed dressing to provide a negative pressure wound therapy. The vacuum draws out accumulated fluid from the wound and increases the blood flow to the area thereby helping in infection control and rapid healing of the wound [9]. Once the wound has granulated well and the systemic infection is controlled, secondary closure of the wound can be undertaken. With this strategy, implants can be successfully retained following wound infection [10].

3. Some patients may require repeated VAC dressings before the wound becomes healthy. Risk factors for repeated VAC dressing are patients with polymicrobial infection or MRSA or other resistant organisms [9].

4. Resolution of infection is assessed clinically with improvement in pain, spasm and movements along with adequate wound healing, hematologically with normalization of WBC count, CRP and on plain xrays with improved definition of endplates, sclerosis of bone, and spinal fusion. Reduction in bone marrow hyperintense signal on T2 weighted MRI and reduction in disc space hyperintensity along with fat replacement of the bone marrow and resolution/significant reduction of abscesses indicates healing of infection.

4.4. Case 4

80-year-old male, a known case of diabetes underwent L4-5 transforaminal lumbar interbody fusion (TLIF) for degenerative spondylolisthesis. Patient was reexplored on day 5 for a malpositioned right L4 screw that was causing leg pain. On day 10 patient had wound dehiscence with a watery discharge from the wound. The wound was again re-explored. The collection that was seropurulent was evacuated and the wound thoroughly debrided. The implants were found to be stable, so they were left in situ. A small dural puncture was detected in the shoulder of the right L4 root that was leaking CSF. Fat graft was used to seal the leak and the wound was closed tightly over a dependent drain. A PICC line was inserted for administering long term IV antibiotics. The culture grew *Klebsiella pneumoniae* that was resistant to all antibiotics except meropenem. IV meropenem and linezolid were started after consulting infectious disease physician.

After the second re-exploration, the wound kept leaking CSF copiously. Lumbar CSF diversion drain was inserted to divert the CSF. Also, a commercially available wound sealing system called 'Zip-line' was applied to supplement the closure of the main wound. About 150cc of CSF was drained daily. On the 6th day of CSF diversion, when the main wound was clean and healing well, the CSF diversion drain was removed. The wound healed uneventfully and the sutures were removed at 2 weeks. IV antibiotics were continued for 6 weeks. Fortunately, the patient did not develop any meningitis symptoms.

Within two weeks of starting oral antibiotics, patient complained of increased back pain and fever. MRI showed fluid collection at the surgical site (Figure 8). The wound was re-explored. Thorough debridement of the wound was done. The implants were found to be stable and without a biofilm and hence were left in

situ. There was no CSF leak and hence now the wound was left open and a VAC system was applied. Over a period of two weeks, following sequential VAC dressings, the wound appeared to be granulating well and hence was closed secondarily over a drain. The same antibiotics were continued based on the culture report. He appeared to be doing well for the next 4 weeks with healing of the wound and reduction in back pain.

Another month later, he again started complaining of back pain. MRI showed no significant intradiscal infection or osteomyelitis. There was however a collection of fluid around the cage. It was decided to remove the implants. The wound was re-explored. The pedicle screws were still holding well. However, they were removed. The interbody cage was found to be loose and came out easily. The disc space was thoroughly irrigated and debrided. A VAC dressing was applied. Antibiotics were switched to imipenem and tigecycline. After sequential dressings, the wound was closed secondarily after 2 weeks. The wound healed uneventfully. The patient's clinical condition improved gradually with improvement in the blood parameters. The disc space at L4/5 went on to collapse and fuse. The patient is now 1 year following the last surgery, has excellent relief in back pain and has returned to normal activities.

4.4.1. Take-home points

1. CSF leak in the setting of a wound infection can result in catastrophic complication such as meningitis. In such a situation, wound infection has to be treated aggressively. Note that VAC dressing is contraindicated in the setting of a persistent CSF leak [11].
2. Resistant organisms are more difficult to eradicate and may require repeated debridements and VAC dressing [9].
3. Most early SSI (<3 months) can be managed with aggressive debridements, which leads to successful salvage of implants [12].

4. Delayed treatment (>3 months) or inadequate treatment, leads to progressive destruction and loosening of implants and reduces the likelihood of implants salvage [12].

5. If the pedicle screw fixation is secure, most surgeons agree that instrumentation can be salvaged and retained until fusion occurs [12].

6. Biofilm formation and bacterial adherence is least on tantalum cages, intermediate on titanium and worst on PEEK cages.

7. There is conflicting recommendations on the removal of an infected interbody cage. If there are signs of cage or pedicle screw loosening, posterior debridement alone could result in high failure rate. Such situations require radical debridement and removal of cage and potentially spinal reconstruction if unstable [12].

Conclusions

Surgical site infection in spine surgery is a devastating complication not only from a medical perspective but also from an economic standpoint. Poor operating conditions in ill-equipped hospitals that do not follow the standard of care put the patient at a higher risk. However, patient-related factors like diabetes, obesity, smoking etc may also contribute to this risk. Certain pathologies, for example, spinal cord injured patients, have a higher risk of postoperative wound infection. Early detection and debridement are necessary, especially if one has to retain the instrumentation. In late infection, if the fusion is confirmed, implant exit can be performed to better treat the infection. Patient with spondylodiscitis following microdiscectomy can be treated with a course of intravenous antibiotics followed by oral antibiotics provided the organism can be isolated and appropriate antibiotics are administered for a sufficient amount of time. If the organism cannot be isolated, or if the patient is severely painful or has a neurological deficit due to epidural abscess, it is better

to treat such patients with debridement and fusion. Patients with pedicle screws and cage loosening require removal of interbody cages and further spinal reconstruction to eradicate the infection.

References

- Horan TC, Gaynes RP, Martone WJ, et al. CDC Definitions of Nosocomial Surgical Site Infections, 1992: A Modification of CDC Definitions of Surgical Wound Infections. *Infect Control Hosp Epidemiology* 1992;13:606–8.
- Anderson DJ, Podgorny K, Berrios-Torres SI, et al. Strategies to Prevent Surgical Site Infections in Acute Care Hospitals: 2014 Update. *Infect Control Hosp Epidemiology* 2014;35:S66–88.
- Boody, B., Jenkins, T., Hashmi, S., Hsu, W., Patel, A., Savage, J. (2015). Surgical Site Infections in Spinal Surgery *Journal of Spinal Disorders and Techniques* 28(10), 352–362.
- Hikata T, Iwanami A, Hosogane N, et al. High preoperative hemoglobin A1c is a risk factor for surgical site infection after posterior thoracic and lumbar spinal instrumentation surgery. *J Orthop Sci* 2014;19:223–8.
- Mehta AI, Babu R, Sharma R, et al. Thickness of Subcutaneous Fat as a Risk Factor for Infection in Cervical Spine Fusion Surgery. *J Bone Jt Surg* 2013;95:323–8.
- Rihn JA, Radcliff K, Hilibrand AS, et al. Does Obesity Affect Outcomes of Treatment for Lumbar Stenosis and Degenerative Spondylolisthesis? Analysis of the Spine Patient Outcomes Research Trial (SPORT). *Spine* 2012;37:1933–46.
- Durand F, Berthelot P, Cazorla C, et al. Smoking is a risk factor of organ/space surgical site infection in orthopaedic surgery with implant materials. *Int Orthop* 2013;37:723–7.
- Klein JD, Hey LA, Yu C, et al. Perioperative Nutrition and Postoperative Complications in Patients Undergoing Spinal Surgery. *Spine* 1996;21:2676–82.
- Cross, M., Yi, P., Thomas, C., Garcia, J., Valle, C. (2014). Evaluation of malnutrition in orthopaedic surgery. *The Journal of the American Academy of Orthopaedic Surgeons* 22(3), 193–199.
- Kim DH, Spencer M, Davidson SM, et al. Institutional Prescreening for Detection and Eradication of Methicillin-Resistant *Staphylococcus aureus* in Patients Undergoing Elective Orthopaedic Surgery. *J Bone Jt Surg* 2010;92:1820–6.
- Radcliff, K., Neusner, A., Millhouse, P., Harrop, J., Kepler, C., Rasouli, M., Albert, T., Vaccaro, A. (2015). What is new in the diagnosis and prevention of spine surgical site infections *The Spine Journal* 15(2), 336–347.
- Blam OG, Vaccaro AR, Vanichkachorn JS, et al. Risk Factors for Surgical Site Infection in the Patient With Spinal Injury. *Spine* 2003;28:1475.
- Vitale, M., Riedel, M., Glotzbecker, M., et.al. (2013). Building consensus: development of a Best Practice Guideline (BPG) for surgical site infection (SSI) prevention in high-risk pediatric spine surgery. *Journal of pediatric orthopedics* 33(5), 471–478.
- McDermott, H., Bolger, C., Humphreys, H. (2012). Postprocedural discitis of the vertebral spine: challenges in diagnosis, treatment and prevention *Journal of Hospital Infection* 82(3), 152–157.
- Basu, S., Ghosh, J., Malik, F., Tikoo, A. (2012). Postoperative discitis following single-level lumbar discectomy: Our experience of 17 cases. *Indian Journal Of Orthopaedics* 46(4), 427–433.
- Lu, X., Guo, Q., Ni, B. (2012). Esophagus perforation complicating anterior cervical spine surgery *European Spine Journal* 21(1), 172–177.
- Rueth, N., Shaw, D., Groth, S. et al. (2010). Management of Cervical Esophageal Injury After Spinal Surgery *The Annals of Thoracic Surgery* 90(4), 1128–1133.
- Ploumis, A., Mehbod, A., Dressel, et al. (2008). Therapy of spinal wound infections using vacuum-assisted wound closure: risk factors leading to resistance to treatment. *Journal of Spinal Disorders and Techniques* 21(5), 320–323.
- Ousey, K., Atkinson, R., Williamson, J., et al (2013). Negative pressure wound therapy (NPWT) for spinal wounds: a systematic review. *The Spine Journal* 13(10), 1393–1405.
- Husted, D., Grauer, J., Hilibrand, A. (2004). The use of wound vacuums in the management of postoperative wound infections *Seminars in Spine Surgery* 16(3), 182–187.
- Chang, C., Fu, T., Chen, W., Chen, C., Lai, P., Chen, S. (2019). Management of Infected Transforaminal Lumbar Interbody Fusion Cage in Posterior Degenerative Lumbar Spine Surgery *World Neurosurgery* 126(), e330–e341.

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