

Michael Murray and Wellington Hsu

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## 38.1 Introduction

With modern surgical techniques and perioperative antibiotics, spinal infection after surgery is relatively uncommon. Certain patient and surgical treatments are however at higher risk, and should be treated with extra caution. This chapter will review the prevention, diagnosis, and treatment of postoperative spinal infection.

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## 38.2 Incidence and Risk Factors

The rate of infection following spine surgery varies considerably based on the procedure performed as well as patient factors. For example, the infection rate following a lumbar discectomy has been reported to be 0.7 %, increasing to 1.4 % with the use of a microscope [40]. In elective instrumented cases, the reported rate of infection increases to between 2.8 and 6 % [16, 20, 22, 29,

37]. In addition to the baseline risk of infection following surgery, additional risk is conferred to patients that have a history of smoking, obesity, diabetes, long-term steroid use, alcohol abuse, prior surgical site infection, prior spinal surgery, malnourishment, and a preoperative hospitalization of >1 week [9, 21, 29, 49]. Patient age as a risk factor has been a subject of debate. Some studies have shown a higher infection rate in older patients, while others show an equal infection rate among age groups [21].

The role of diabetes in the development of postoperative infections has been investigated thoroughly. Chen et al. demonstrated a relative risk of 4.10 (95 % CI: 1.37–12.32) for developing a postoperative surgical site infection (either deep or superficial) in diabetic patients [12]. A history of diabetes makes a patient susceptible to infection due to impaired tissue microvasculature, poor antibiotic penetration [18], and immunosuppression secondary to impaired granulocyte function [44]. In addition to these underlying impairments, elevated pre- (>125 mg/dL) and postoperative (>200 mg/dL) blood glucose levels have been identified as an independent risk factor for the development of a postoperative infection [33]. To this end, tight control of perioperative blood glucose levels is critical in preventing postoperative surgical site infections in this patient population.

A history of prior surgical site infection is another risk factor that has been described by multiple investigators [1, 36, 50]. It is proposed that the previous infectious organism may reside in small quantities in the scar tissue caused by

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M. Murray, M.D. (✉)  
Emory Spine Center,  
Department of Orthopaedic Surgery,  
59 Executive Park South, Suite 3000,  
Atlanta, GA 30329, USA  
e-mail: mmurra5@emory.edu

W. Hsu, M.D.  
Northwestern University,  
Department of Orthopaedic Surgery,  
676 N. St. Clair, Suite 1350, Chicago,  
IL 60611, USA  
e-mail: whsu@nmff.org

prior procedures. The patient may have no signs or symptoms of infection despite this bacterial colonization. Consequently, the antibiotic sensitivities of the prior infectious organism should be considered when choosing a perioperative prophylactic antibiotic in patients with prior surgical site infections [36].

Surgery for spine trauma is associated with an increased rate of infection compared with those for other diagnoses. The rate of a postoperative infection following trauma has been reported to be upward of 10 % [7, 37]. The explanation for this infection rate is believed to be multifactorial. These patients are more likely to have spent time in the ICU and have poor soft-tissue envelopes secondary to significant soft-tissue trauma. They often have a greater number of comorbidities and may be more nutritionally compromised due to their trauma-induced catabolic state [23]. Finally, trauma patients with complete neurologic deficit or cognitive impairment are at an even higher risk of a postoperative infection [37, 43]. Further risk factors specific to this population include multiple levels of surgical involvement and delayed surgical treatment of over 160 h [7].

In addition to patient-specific risk factors, the operative plan and surgical technique may also influence the chances of developing a postoperative infection. Multiple studies have suggested that the anterior surgical approach is associated with a lower rate of postoperative infections when compared to the posterior approach [26, 36]. This phenomenon is hypothesized to be a result of enhanced bacterial clearance due to superior venous and lymphatic drainage of the anterior spine. Therefore, consideration should be made for an anterior surgical approach if the same surgical goals can be accomplished equally through either approach.

Intraoperative risk factors for infection have been reported to include prolonged surgical time and intraoperative blood loss in excess of 1 L [50]. Susceptibility to infection following blood loss has been hypothesized to be due to the association between significant blood loss and subsequent non-autologous blood transfusion. Non-autologous blood transfusions have been hypothesized to cause a relatively immunosuppressed state in the recipient (termed transfusion-associated immunomodulation (TRIM) [6]).

The patient may then become susceptible to an increased risk of infection [4, 31, 46]. Therefore, effort should be made to minimize blood loss whenever possible and, thus, the need for non-autologous transfusions.

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### 38.3 Etiology

Postoperative infections may arise from either direct inoculation of the wound or hematogenous seeding. Infections caused by low virulence organisms are thought to be due largely from direct inoculation. These organisms are highly susceptible to clearance by the body's immune system and rarely spread hematogenously. However, virulent organisms, such as *Staphylococcus aureus*, may cause infection by either route. *Staphylococcus aureus* has been demonstrated to be the most common causative organism in postoperative spine infections. A study by Massie [29] showed that *Staphylococcus aureus* was present in over 50 % of the 22 cases of postoperative spinal infections that were analyzed. Other isolated organisms included *Staphylococcus epidermidis* (coagulase-negative *Staphylococcus*), *Peptococcus*, *Enterobacter cloacae*, and *Bacteroides*. Gram-negative organisms are less commonly identified and are seen primarily in trauma patients, especially those with neurologic injury [37]. Polymicrobial infections may also occur but are felt to be due to direct inoculation of the wound as opposed to a hematogenous route.

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### 38.4 Prevention

Prophylactic antibiotics have been shown to decrease infection rates in all types of spine surgery [5]. Since *Staphylococcus aureus* is the most common offending organism in postoperative infections, a first-generation cephalosporin such as cefazolin is generally selected as the antibiotic of choice. Cefazolin is effective against both *Staphylococcus aureus* and *Staphylococcus epidermidis* and reaches peak serum concentrations quickly. For patients allergic to penicillin, clindamycin and vancomycin are viable alternatives. If a patient is at high risk for methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin is

frequently the prophylactic antibiotic of choice [40]. The perioperative antibiotic should be administered within 1 h of skin incision, and cefazolin should be re-dosed if the surgical duration exceeds 4 h. It is also recommended that antibiotics should also be re-dosed if the surgical blood loss exceeds 1,500 mL, based on findings of decreased tissue concentration of cefazolin in settings of blood loss that exceeds that volume [45]. Postoperatively, antibiotics should be given for 24 h after closure.

Perioperative skin preparation products are also utilized for infection prevention. For example, in foot/ankle and shoulder surgery, ChlorPrep (2 % chlorhexidine gluconate and 70 % isopropyl alcohol, Enturia, El Paso, TX, USA) was found to be superior to other cleansing products such as DuraPrep and povidone-iodine scrub in terms of reducing the cutaneous bacterial load [34]. Despite encouraging evidence for chlorhexidine as a superior operative skin preparation, there has not been any evidence to date, showing a decreased clinical infection rate when used in spine surgery.

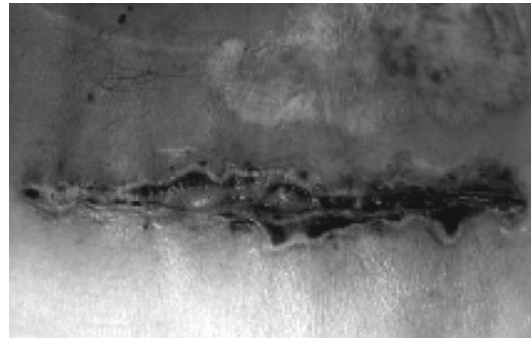
Other surgical techniques that have been postulated to reduce infection rates but not substantiated by results include release of retractors at least every 2 h, use of antibiotic irrigation, avoidance of drains, and debridement of necrotic tissue at the end of the case. The practice of shaving the surgical site prior to surgery has been examined and suggested to actually increase the rate of postoperative surgical site infection [10].

## 38.5 Diagnosis

### 38.5.1 Clinical Presentation

Postoperative infections of the spine can be separated into superficial and deep infections. Superficial infections often present with pain, erythema, edema, warmth, and occasional drainage at the surgical site. These infections are primarily diagnosed by clinical evidence and are frequently managed medically with antibiotic therapy. However, it is essential that a deep infection is not overlooked.

Deep infections are often more challenging to diagnose. Patients often will have indolent



**Fig. 38.1** Clinical image of a deep infection of the lumbar spine associated with underlying myonecrosis (Reprinted with permission from Sasso et al. [40])

infections which do not manifest acutely after surgery. Fevers and other systemic symptoms are often not present, and white blood cell counts may not be elevated, particularly with indolent infections. A persistently painful, draining wound that does not respond to local wound care and conservative measures is one clue to a deep infection (Fig. 38.1). However, the superficial appearance of the wound may appear benign due to the deep nature of the infection and the multi-layered closure. Therefore, when pain at the surgical site is unexplained and does not decrease as expected postoperatively, an infection must be ruled out. Drainage from a seroma will be relatively clear, possibly blood tinged, and with low viscosity, whereas that from an infection will likely be more copious, viscous, and may appear purulent. Fluid aspiration and analysis has been deemed particularly useful for the detection of acute infections [43].

Patients suspected of infection must be evaluated for constitutional signs of infection as well such as a temperature reading over 39° centigrade. Furthermore, other signs such as chills, sweats, malaise, lethargy, and mental status changes should be noted. These signs warrant urgent intervention as sepsis may lead to multiple organ system failure and even death.

### 38.5.2 Laboratory Evaluation

As mentioned above, deep infections may exist in the setting of a completely benign appearing

surgical site. In these patients, the presence of unexplained increasing pain at the surgical site may be the only clinical finding. In the right clinical setting, leukocyte count, ESR, and CRP could aid the evaluation of these patients. An elevated ESR and CRP warrant increased suspicion for a postoperative infection; however, levels must be considered in respect to their expected postoperative values. ESR does not typically return to baseline values until 6 weeks after surgery, while CRP returns to baseline 2 weeks postoperatively [40].

### 38.5.3 Imaging

In addition to clinical and laboratory evaluation, imaging studies are often indicated and may be useful in the diagnosis of a postoperative infection. Plain radiographs should be evaluated carefully as the subtle finding of osteolysis around hardware may be suggestive of an infection. However, in the early stages of infection, the utility of plain radiographs is limited as radiographic findings lag considerably behind clinical symptoms.

A CT scan may be performed to examine the vertebral bodies as well as any possible dissolution of the end plates or disk space which may be seen with a diskitis or vertebral osteomyelitis. CT will also show increased detail of the instrumentation-bone interface. Evaluation of this interface is critical as lucency surrounding instrumentation may be indicative of a postoperative infection.

MRI is the most sensitive test for the detection of a postoperative infection; however, findings must be interpreted with caution because of the expected inflammatory response from the surgical insult. Gadolinium contrast can help to increase the sensitivity of the MRI by demonstrating rim enhancement of a large fluid collection, progressive marrow changes, and ascending epidural collections [24, 41].

While the diagnosis of a postoperative infection is challenging, adequate information can frequently be obtained through clinical evaluation, appropriate laboratory studies (including WBC,

ESR, CRP), and imaging studies. However, if the laboratory studies and imaging are equivocal, close observation with repeated studies as appropriate is recommended.

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## 38.6 Postoperative Wound Classification

Postoperative wounds may be classified by the Thalgott et al. [47] modification of the Cierny classification [13] for osteomyelitis. This staging system takes into consideration the characteristics of the infection (group 1: single organism, group 2: multiple organism, or group 3: multiple organism plus myonecrosis) as well as the clinical status of the patient (class A: healthy, normal immune system; class B: local or multiple systemic diseases, including smoking; or class C: immunocompromised or injury severity score >18). Thalgott evaluated 32 patients with postoperative infection and found 13 to be infected with a single organism (group 1), 16 with more than one organism (group 2), and two patients with multiple organism and extensive myonecrosis. The average time to diagnosis was 23 days (range 5–110 days). Most patients with single organism infections (group 1) were successfully managed with single irrigation and debridement with closure over suction drains. Patients with multiple organism infections (group 2) required an average of three irrigation and debridements. This study included two patients with group 3 infections. These were difficult to manage and had much worse outcomes than the other two groups. They required an average of six surgeries (range 4–8), and both patients required flaps for closure.

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## 38.7 Management

### 38.7.1 Non-Instrumented Infections

#### 38.7.1.1 Post-Procedural Diskitis

Percutaneous intradiskal procedures have become popular methods to both diagnose and treat disk pathology. The rate of infection

following these procedures has been reported to range between 0.2 and 2.75 % [35, 42]. Clinical manifestations of infection may not be obvious, and suspicion must be heightened in patients with unexplained increasing pain following the procedure. *Staphylococcus aureus* is the most common causative organism, but anaerobic organisms have been isolated as well [39]. The incision site is often benign appearing, and blood cultures are generally negative. Serum markers of inflammation including CRP and ESR may be helpful, but an MRI is frequently indicated. Findings on an MRI may be nonspecific, and a biopsy is often indicated for definitive diagnosis of an infection. After diagnosis, management with culture-directed intravenous antibiotics and bracing for comfort are generally effective [28]. Surgical intervention is primarily indicated only for cases of failure of medical management and development of neurologic deficit.

### 38.7.1.2 Post-Diskectomy Diskitis

Patients with a postoperative disk space infection may present with the isolated finding of increasing or non-dissipating low back pain in an otherwise uneventful postoperative course. Persistent elevation of inflammatory markers (ESR, CRP) may also assist in the diagnosis. Imaging using MRI with gadolinium is often performed and may show enhancement of the disk space and adjacent bone marrow on T2 images (Fig. 38.2) and decreased signal from the disk space on T1 images. Treatment of a postoperative disk space infection involves appropriate fluid or tissue culture followed by the administration of intravenous antibiotics. Often, treatment with antibiotics alone may resolve the infection and result in an autofusion of the infected disk space. Surgical indications include failure of medical management (evidenced by progression of infection on MRI), spread of the infection into the spinal canal with abscess formation, or neurologic deficit. Surgical intervention traditionally involves debridement of the disk space from either an anterior or posterior approach. Successful management has also been reported with a posterior interbody fusion with instrumentation [25].



**Fig. 38.2** A T2 weighted image of the lumbar spine demonstrating increased signal within the disk space and adjacent vertebral body. These findings are consistent with a diskitis (Reprinted with permission from Sasso et al. [40])

### 38.7.1.3 Postspinal Decompression

Postoperative infections following a spinal decompression often cause subfascial abscesses with or without associated disk involvement or vertebral osteomyelitis. These subfascial abscesses often do not respond adequately to antibiotics, and surgical intervention is generally indicated. Culture of infected material and debridement of infected necrotic material is recommended. Intravenous antibiotics based on culture results should be initiated. Intravenous antibiotics are maintained for at least 6 weeks, and consideration should be given to serial debridements, particularly those with multiple organism involvement, substantial myonecrosis, and in immunocompromised patients. Timely recognition and management of the infection is absolutely necessary in order to prevent spread of the infection to the disk and vertebral bodies.

## 38.7.2 Instrumented Infections

### 38.7.2.1 Post-Instrumented Fusion

Deep infections that occur after instrumentation of the spine are best managed surgically. The goals of managing this type of infection are identification of the offending organism, eradication of the infection, wound healing, and maintenance of the structural integrity of the spine and viability of the bone graft. In the operating room, deep tissue gram stain and cultures should be obtained prior to administration of intravenous antibiotics. Cultures should be maintained for at least 10 days in order to detect less virulent organisms, including *Propionibacterium acnes* [38].

Meticulous debridement should be performed at all layers of the wound, and all devitalized material should be excised. Pulse lavage may be used but should not be considered a substitute for meticulous excision of necrotic tissue. In subacute cases (<6 months from the time of surgery) and in the absence of a virulent infection, an attempt is made to retain instrumentation and to revise or remove only instrumentation that is found to be loose [48]. Stable hardware should be left in place in order to prevent destabilization of the spine. If loose instrumentation is identified, titanium implants should be used for the revision in the setting of infection. Titanium has been demonstrated in an animal model to be associated with a lower infection rate following a bacterial challenge when compared to steel [2]. The efficacy of titanium in the setting of revision surgery following a spinal infection is also supported in multiple clinical studies [11, 17, 27]. The next step in management depends on the results of the intraoperative gram stain. If the gram stain reveals no organism or a single organism, closure over suction drains is a viable option. With a polymicrobial infection, consideration should be made for multiple debridements until the infection has been eradicated. With extensive myonecrosis, repeat debridement at 48 and 72 h is highly recommended with repeat gram stain and cultures taken at each debridement.

Wound closure in such cases of severe soft-tissue necrosis may be accomplished by granulation tissue from secondary intention, with/without

wound vacuum assistance, or coverage by local muscle rotational flap. The use of a wound vacuum has been successful in patients with exposed hardware and considerable tissue loss [3, 51]. Local muscle flaps have also been found to be effective, providing increased vascularity and soft-tissue coverage, thereby providing protection for underlying bone graft and instrumentation [15, 30]. Consideration for a plastic surgery consult should be made for wound management, particularly when substantial soft tissue is debrided [15, 30]. Broad-spectrum antibiotics should be started postoperatively until the intraoperative cultures and organism susceptibilities have returned. Culture-directed intravenous antibiotics are maintained for at least a 6-week period.

Consultation with infectious disease is often indicated for management of these difficult infections. In addition, it is important to consider the nutritional status of the patient. Laboratory markers such as transferrin and albumin can be monitored to assess the patient's nutritional state. For patients with substantial myonecrosis, intravenous hyperalimentation should be considered.

In the setting of extensive infection or in patients with late-onset of infection, antibiotics and surgical irrigation and debridement may not definitively eradicate the infection. Ho et al. demonstrated that hardware was able to be retained in 97 % of patients presenting with infection within 6 months of surgery but only 59 % of patients presenting >6 months of their index procedure [19]. Bose suggested routine removal of hardware in patients with a late presenting infection [8]. However, he further commented that this decision should be made on a case-by-case basis and that if the infection is not communicating with the hardware that an attempt can be made to retain the hardware. If retention of the hardware is attempted, but a patient fails serial debridements, one must be prepared to abandon this plan. The goal then becomes to delay removal of the instrumentation until successful spinal fusion has occurred. Intravenous antibiotic therapy may be used to suppress the infection until a fusion mass has solidified. Once the fusion mass is present, removal of instrumentation should be then

performed. However, postoperatively, the spine should be monitored carefully for the increased risk of spinal deformity following removal of instrumentation [14, 32].

### Conclusion

As with all spinal infections, diagnosis of a postoperative infection begins with a detailed history and physical exam, followed by appropriate blood tests and imaging. Spinal infections may be challenging as the definitive diagnosis often remains unclear after these diagnostic steps. In this case, a biopsy is indicated. Once the diagnosis has been established, treatment will vary depending on the characteristics of the infection. Variables that are considered in the treatment of all subtypes of infection are the duration of infection, organism, neurologic status, structural integrity, and maintenance of spinal alignment.

### References

1. Andreshak TG, An HS, Hall J, Stein B (1997) Lumbar spine surgery in the obese patient. *J Spinal Disord* 10(5):376–379
2. Arens S, Schlegel U, Printzen G, Ziegler WJ, Perren SM, Hansis M (1996) Influence of materials for fixation implants on local infection. An experimental study of steel versus titanium DCP in rabbits. *J Bone Joint Surg Br* 78(4):647–651
3. Argenta LC, Morykwas MJ (1997) Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. *Ann Plast Surg* 38(6):563–576;discussion 577
4. Banbury MK, Brizzio ME, Rajeswaran J, Lytle BW, Blackstone EH (2006) Transfusion increases the risk of postoperative infection after cardiovascular surgery. *J Am Coll Surg* 202(1):131–138. doi:10.1016/j.jamcollsurg.2005.08.028, pii:S1072-7515(05)01430-4
5. Barker FG 2nd (2002) Efficacy of prophylactic antibiotic therapy in spinal surgery: a meta-analysis. *Neurosurgery* 51(2):391–400; discussion 400–391
6. Blajchman MA (2002) Immunomodulation and blood transfusion. *Am J Ther* 9(5):389–395
7. Blam OG, Vaccaro AR, Vanichkachorn JS, Albert TJ, Hilibrand AS, Minnich JM, Murphey SA (2003) Risk factors for surgical site infection in the patient with spinal injury. *Spine (Phila Pa 1976)* 28(13):1475–1480. doi:10.1097/01.BRS.0000067109.23914.0A
8. Bose B (2003) Delayed infection after instrumented spine surgery: case reports and review of the literature. *Spine J* 3(5):394–399, pii:S1529943003000238
9. Capen DA, Calderone RR, Green A (1996) Perioperative risk factors for wound infections after lower back fusions. *Orthop Clin North Am* 27(1):83–86
10. Celik SE, Kara A (2007) Does shaving the incision site increase the infection rate after spinal surgery? *Spine (Phila Pa 1976)* 32(15):1575–1577. doi:10.1097/BRS.0b013e318074c39f, pii:00007632-200707010-00002
11. Chang CC, Merritt K (1994) Infection at the site of implanted materials with and without preadhered bacteria. *J Orthop Res* 12(4):526–531. doi:10.1002/jor.1100120409
12. Chen S, Anderson MV, Cheng WK, Wongworawat MD (2009) Diabetes associated with increased surgical site infections in spinal arthrodesis. *Clin Orthop Relat Res* 467(7):1670–1673. doi:10.1007/s11999-009-0740-y
13. Cierny G 3rd, Mader JT, Penninck JJ (2003) A clinical staging system for adult osteomyelitis. *Clin Orthop Relat Res* 414:7–24. doi:10.1097/01.blo.0000088564.81746.62
14. Deckey JE, Court C, Bradford DS (2000) Loss of sagittal plane correction after removal of spinal implants. *Spine (Phila Pa 1976)* 25(19):2453–2460
15. Dumanian GA, Ondra SL, Liu J, Schafer MF, Chao JD (2003) Muscle flap salvage of spine wounds with soft tissue defects or infection. *Spine (Phila Pa 1976)* 28(11):1203–1211. doi:10.1097/01.BRS.0000067260.22943.48
16. Esses SI, Sachs BL, Dreyzin V (1993) Complications associated with the technique of pedicle screw fixation. A selected survey of ABS members. *Spine (Phila Pa 1976)* 18(15):2231–2238; discussion 2238–2239
17. Fayazi AH, Ludwig SC, Dabbah M, Bryan Butler R, Gelb DE (2004) Preliminary results of staged anterior debridement and reconstruction using titanium mesh cages in the treatment of thoracolumbar vertebral osteomyelitis. *Spine J* 4(4):388–395. doi:10.1016/j.spinee.2004.01.004, pii:S1529943004000191
18. Goodson WH 3rd, Hung TK (1977) Studies of wound healing in experimental diabetes mellitus. *J Surg Res* 22(3):221–227
19. Ho C, Skaggs DL, Weiss JM, Tolo VT (2007) Management of infection after instrumented posterior spine fusion in pediatric scoliosis. *Spine (Phila Pa 1976)* 32(24):2739–2744. doi:10.1097/BRS.0b013e31815a5a86, pii:00007632-200711150-00018
20. Hodges SD, Humphreys SC, Eck JC, Covington LA, Kurzynske NG (1998) Low postoperative infection rates with instrumented lumbar fusion. *South Med J* 91(12):1132–1136
21. Khan IA, Vaccaro AR, Zlotolow DA (1999) Management of vertebral diskitis and osteomyelitis. *Orthopedics* 22(8):758–765
22. Klein JD, Garfin SR (1996) Nutritional status in the patient with spinal infection. *Orthop Clin North Am* 27(1):33–36
23. Klein JD, Hey LA, Yu CS, Klein BB, Coufal FJ, Young EP, Marshall LF, Garfin SR (1996) Perioperative

- nutrition and postoperative complications in patients undergoing spinal surgery. *Spine (Phila Pa 1976)* 21(22):2676–2682
24. Kothari NA, Pelchovitz DJ, Meyer JS (2001) Imaging of musculoskeletal infections. *Radiol Clin North Am* 39(4):653–671
  25. Lee JS, Suh KT (2006) Posterior lumbar interbody fusion with an autogenous iliac crest bone graft in the treatment of pyogenic spondylodiscitis. *J Bone Joint Surg Br* 88(6):765–770. doi:10.1302/0301-620-X.88B6.17270, pii:88-B/6/765
  26. Levi AD, Dickman CA, Sonntag VK (1997) Management of postoperative infections after spinal instrumentation. *J Neurosurg* 86(6):975–980. doi:10.3171/jns.1997.86.6.0975
  27. Liljenqvist U, Lerner T, Bullmann V, Hackenberg L, Halm H, Winkelmann W (2003) Titanium cages in the surgical treatment of severe vertebral osteomyelitis. *Eur Spine J* 12(6):606–612. doi:10.1007/s00586-003-0614-z
  28. Lindholm TS, Pylkkanen P (1982) Discitis following removal of intervertebral disc. *Spine (Phila Pa 1976)* 7(6):618–622
  29. Massie JB, Heller JG, Abitbol JJ, McPherson D, Garfin SR (1992) Postoperative posterior spinal wound infections. *Clin Orthop Relat Res* 284:99–108
  30. Mitra A, Harlin S (2004) Treatment of massive thoracolumbar wounds and vertebral osteomyelitis following scoliosis surgery. *Plast Reconstr Surg* 113(1):206–213. doi:10.1097/01.PRS.0000097440.15013.5C
  31. Murphy GJ, Reeves BC, Rogers CA, Rizvi SI, Culliford L, Angelini GD (2007) Increased mortality, postoperative morbidity, and cost after red blood cell transfusion in patients having cardiac surgery. *Circulation* 116(22):2544–2552. doi:10.1161/CIRCULATIONAHA.107.698977, pii:CIRCULATIONAHA.107.698977
  32. Muschik M, Luck W, Schlenzka D (2004) Implant removal for late-developing infection after instrumented posterior spinal fusion for scoliosis: reinstrumentation reduces loss of correction. A retrospective analysis of 45 cases. *Eur Spine J* 13(7):645–651. doi:10.1007/s00586-004-0694-4
  33. Olsen MA, Nepple JJ, Riew KD, Lenke LG, Bridwell KH, Mayfield J, Fraser VJ (2008) Risk factors for surgical site infection following orthopaedic spinal operations. *J Bone Joint Surg Am* 90(1):62–69. doi:10.2106/JBJS.F.01515, pii:90/1/62
  34. Ostrander RV, Botte MJ, Brage ME (2005) Efficacy of surgical preparation solutions in foot and ankle surgery. *J Bone Joint Surg Am* 87(5):980–985. doi:10.2106/JBJS.D.01977, pii:87/5/980
  35. Petri A, Jensen IP (1996) Postoperative lumbar discitis. Types, diagnosis and treatment. *Ugeskr Laeger* 158(38):5281–5285
  36. Pull ter Gunne AF, Cohen DB (2009) Incidence, prevalence, and analysis of risk factors for surgical site infection following adult spinal surgery. *Spine (Phila Pa 1976)* 34(13):1422–1428. doi:10.1097/BRS.0b013e3181a03013, pii:00007632-200906010-00016
  37. Rehtine GR, Bono PL, Cahill D, Bolesta MJ, Chrin AM (2001) Postoperative wound infection after instrumentation of thoracic and lumbar fractures. *J Orthop Trauma* 15(8):566–569
  38. Richards BR, Emara KM (2001) Delayed infections after posterior TSRH spinal instrumentation for idiopathic scoliosis: revisited. *Spine (Phila Pa 1976)* 26(18):1990–1996
  39. Saeed MU, Mariani P, Martin C, Smego RA Jr, Potti A, Tight R, Thiege D (2005) Anaerobic spondylodiscitis: case series and systematic review. *South Med J* 98(2):144–148
  40. Sasso RC, Garrido BJ (2008) Postoperative spinal wound infections. *J Am Acad Orthop Surg* 16(6):330–337, pii:16/6/330
  41. Seabold JE, Nepola JV (1999) Imaging techniques for evaluation of postoperative orthopedic infections. *Q J Nucl Med* 43(1):21–28
  42. Silber JS, Anderson DG, Vaccaro AR, Anderson PA, McCormick P (2002) Management of postprocedural discitis. *Spine J* 2(4):279–287, pii:S1529943002002036
  43. Sponseller PD, LaPorte DM, Hungerford MW, Eck K, Bridwell KH, Lenke LG (2000) Deep wound infections after neuromuscular scoliosis surgery: a multicenter study of risk factors and treatment outcomes. *Spine (Phila Pa 1976)* 25(19):2461–2466
  44. Subbaiah PV, Bagdade JD (1978) Demonstration of enzymatic conversion of lysolecithin to lecithin in normal human plasma. *Life Sci* 22(22):1971–1977
  45. Swoboda SM, Merz C, Kostuik J, Trentler B, Lipsett PA (1996) Does intraoperative blood loss affect antibiotic serum and tissue concentrations? *Arch Surg* 131(11):1165–1171; discussion 1171–1162
  46. Tartter PI (1988) Blood transfusion and infectious complications following colorectal cancer surgery. *Br J Surg* 75(8):789–792
  47. Thalgott JS, Cotler HB, Sasso RC, LaRocca H, Gardner V (1991) Postoperative infections in spinal implants. Classification and analysis—a multicenter study. *Spine (Phila Pa 1976)* 16(8):981–984
  48. Weiss LE, Vaccaro AR, Scuderi G, McGuire M, Garfin SR (1997) Pseudarthrosis after postoperative wound infection in the lumbar spine. *J Spinal Disord* 10(6):482–487
  49. Wimmer C, Gluch H (1996) Management of postoperative wound infection in posterior spinal fusion with instrumentation. *J Spinal Disord* 9(6):505–508
  50. Wimmer C, Gluch H, Franzreb M, Ogon M (1998) Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. *J Spinal Disord* 11(2):124–128
  51. Yuan-Innes MJ, Temple CL, Lacey MS (2001) Vacuum-assisted wound closure: a new approach to spinal wounds with exposed hardware. *Spine (Phila Pa 1976)* 26(3):E30–E33