REVIEW ARTICLE

Infections in spinal instrumentation

Antoine Gerometta • Juan Carlos Rodriguez Olaverri • Fabian Bitan

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Abstract Surgical-site infection (SSI) in the spine is a serious postoperative complication. Factors such as posterior surgical approach, arthrodesis, use of spinal instrumentation, age, obesity, diabetes, tobacco use, operating-room environment and estimated blood loss are well established in the literature to affect the risk of infection. Infection after spine surgery with instrumentation is becoming a common pathology. The reported infection rates range from 0.7% to 11.9%, depending on the diagnosis and complexity of the procedure. Besides operative factors, patient characteristics could also account for increased infection rates. These infections after instrumented spinal fusion are particularly difficult to manage due to the implanted, and possibly infected, instrumentation. Because the medical, economic and social costs of SSI after spinal instrumentation are enormous, any significant reduction in risks will pay dividends. The goal of this literature review was to analyse risk factors, causative

organisms, diagnostic elements (both clinical and biological), different treatment options and their efficiency and consequences and the means of SSI prevention.

Introduction

Over the last two decades, major advances in surgical instrumentation of the vertebral column have emerged for a number of spinal pathologies, including fractures and degenerative and neoplastic diseases. Infections may develop after any surgical procedure, and their management in the setting of spinal instrumentation is critical to provide appropriate postoperative care. The incidence of wound infection after spinal surgery without instrumentation is relatively low. However, using spinal instrumentation clearly increases the risk for postoperative soft tissue infections, and recent estimates from retrospective reviews range from 2.1% to 8.5%. A number of well-recognised risk factors for postoperative wound infection development are inextricably linked to the insertion of spinal instrumentation. Recognising such potential factors permits their reduction and, consequently, may diminish the incidence of wound infections. Given the wider application of spinal instrumentation in over the last 10 years, managing related postoperative infections has become increasingly important. The major objectives of this article were to review clinical characteristics, operative management and outcome of postoperative infections in patients with spinal instrumentation.

A. Gerometta
Orthopedic Resident Lenox Hill Hospital,
130 East, 77th street, 7TH floor,
New York, NY 10075, USA
e-mail: agerometta@wanadoo.fr

J. C. R. Olaverri (

Orthopedic Surgery, NYU School of Medicine,
Spine Orthopedic Attending Lenox Hill Hospital,
130 East, 77th street, 7TH floor,
New York, NY 10075, USA
e-mail: olaverri67@yahoo.com

F. Bitan

Orthopedic Surgery at Albert Einstein College of Medicine, Chief of Spine Services at Lenox Hill Hospital, 130 East, 77th street, 7TH floor, New York, NY 10075, USA e-mail: fabienbitan@gmail.com

Epidemiology

The most frequent causative organism for surgical-site infections (SSIs) following spine surgery is *Staphylococcus*



Table 1 Characteristics of infection in several studies

Characteristics						
Articles	Number of infected patients	Population	Mono- or polymicrobial cultures	Most frequent organisms (%)	Time since initial surgery (number of patients)	No-growth cultures (%)
Pull ter Gunne et al. (2010) [6]	132	Adult	Monomicrobial 63 (52.1%)	. Staphylococcus aureus 65.1% . Enterococcus faecalis 14.5% . MRSA* 10.8 %	nd°	38 (31.4%)
			Polymicrobial 20 (16.5%)	. Escherichia coli 10.8%		
				. Klebsiella pneumoniae 7.2%		
				. Streptococcus spp. 4.8%		
Cahill et al. (2010) [7]	61	paediatric	Monomicrobial 29 (47.5%)	. S. aureus 38% . Staphylococcus coagulase negative 24%	\leq 3 months 32 (52.4%) \geq 3 months 29 (47.5%)	11 (18%)
				. MRSA 20%		
			Polymicrobial21 (34.4%)	. Pseudomonas spp. 20% . E. coli 18%		
				. E. faecalis 16%		
				. Enterobacter spp. 8%		
				. Proteus mirabilis 8%		
Mok et al. (2009) [1]	16	Adult paediatric	Monomicrobial 7 (43.7%)	. Staphylococcus coagulase negative 50% . Enterococcus spp.50%	≤ 3 months 12 (75%)	0%
				. S. aureus 31.2%		
			Polymicrobial9 (56.3%)	. P. acnes 18.7% . E. coli 18.7%	≥ 3 months 4 (25%)	
				. Enterobacter spp. 18.7%		
"Rihn"/>Rihn et al. (2008) [8]	7	paediatric	Monomicrobial 5 (83.3%)	. Staphylococcus coagulase negative 33.3% MRSA 16.6%	\leq 6 months 1 (14.3%) \geq 6 months 6 (85.7%)	14.3%
			Polymicrobial1 (16.6%)	. E. faecalis 16.6% . Streptococcus spp. 16.6%		
				. P. acnes 16.6%		
				. S. marscesens 16.6%		
"Ho"/>Ho et al. (2007) [9]	53	paediatric	Monomicrobial 45 (85%)	. Staphylococcusylococcus coagulase negative 47% . S. aureus 17%	≤ 6 months 31 (58.5%)	6%
				. Enterococcus spp. 6%		
			Polymicrobial 8 (15%)	. Pseudomonas spp. 6% . E. coli 4%	≥ 6 months 22 (41.6%)	
				. Enterobacter spp. 4%		
"Fang"/>Fang et al. (2005) [10]	48	Adult paediatric	nd°	. S. aureus 56.2% . Staphylococcus coagulase negative 37.5%	≤ 3 months 40 (83.4%)	0%
				. Enterococcus spp. 23% . E. coli 8.3%	\geq 3 months 8 (16.7%)	
				. Pseudomonas spp. 8.3%		
Collins et al. (2008) [11]48	74	Adult paedatric	Monomicrobial 46 (62.2%)	. Propionibacterium spp. 46% .Staphylococcus coagulase negative 24.3%	≤ 30 days 6 (8%)	0%
			Polymicrobial 28 (37.8%)	. S. aureus 12%		
				.Staphylococcusylococcus coagulase negative 24.3%	≥ 30 days 68 (92%)	

MRSA methicillin-resistant S. aureus, nd not documented

aureus [1–5]. Table 1 presents findings of studies by several authors following their analysis of SSIs.

Pull ter Gunne et al. [12] reported that of 132 cases, S. aureus was found in 72.6% of all deep SSI isolates,



with 17.8% demonstrating methicillin resistance. Either Escherichia coli or Enterococcus faecalis suggesting genitourinary or faecal wound contamination caused most other cases of deep SSI. In the superficial SSI group, S. aureus was present in 85.7%, with only 5.5% demonstrating methicillin resistance. However, in this series, patients had their incision primarily explored and cultured. Culture aspirations deep into the fascia were performed and local wound care initiated. The reliability of these cultures was not as favourable as deep cultures taken in the operating room. Only 51.2% of the wounds cultured were positive. As opposed to the literature, most SSIs were caused by a single species of bacteria. Low virulency skin organisms can lead to infection on spinal instrumentation. Propionibacterium is regarded as a low-virulence organism and has only been reported as a late cause of postoperative infection following posterior spinal fusion and instrumentation [13-15]. Bemer et al. [16] report a rate of 9.7% positive cultures to P. acnes in spinal instrumentation in a series of 68 patients. The incidence becomes obviously higher with improved culture technique, as P. acnes requires an extended incubation period before it can be identified. These authors recommend that the surgeon perform at least four deep samples to facilitate culture interpretation. Results of at least four deep-culture samples, histology and Creactive protein (CRP) must be compared with the preoperative macroscopic details.

Diagnosis

Clinical

Diagnosis may be difficult clinically, as signs appear a few months or even years after the surgery, making longterm follow-up necessary. In most cases, clinical signs and symptoms are fever, pain, erythema, swelling, warmth, tenderness to palpation or wound drainage. Pull ter Gunne et al. [12] found that wound drainage was the most frequently seen sign indicating SSI and was present in 67.9% of deep SSI and 64.6% of isolated superficial SSI. In the study of Collins et al. [11], the mean time for infection diagnosis was 14 months (days to years postoperatively); 24.3% were detected or more years after initial surgery. These demonstrate the need for longterm follow-up following instrumented spinal surgery in order to obtain a more accurate indication of the incidence of infection. Pull ter Gunne et al. [6] compared the time to diagnosis between deep and superficial SSIs and found the mean time to diagnosis was shorter in the deep SSI group (15 vs. 18 days).

Laboratory

This literature review emphasises the importance of the different biological markers. All infection markers were elevated [white blood cells (WBC), erythrocyte sedimentation rate (ESR) and CRP)] at the time of diagnosis. First, it is important to determine what are considered the normal levels of these markers after surgery and when they should return to normal. CRP levels are increased and can take two weeks to normalise [17]. ESR will also be elevated, reaching a peak level at two weeks, and requires up to six weeks to return to normal levels. Most authors found that CRP value was more sensitive than ESR or WBC for different types of SSI. CRP level was a highly sensitive factor for infection [18-20], with 96.8% of deep and 100% of superficial SSIs having elevated levels. WBC count has been a less reliable indicator for infection. Takahashi et al. [21] found that the most sensitive parameter for enhanced inflammation elicited by implants appears to be CRP because its level increased sharply immediately after surgery, reaching a high peak level on day two. The authors concluded that hardware was the cause of the acute inflammatory response with a high CRP level after surgery. The increase in CRP seven days after surgery can be used to monitor a possible infection, because at that time, it should be lower than the level two days after surgery. WBC and renewed elevation of CRP, WBC and body temperature appear to be reliable signals [18]. Takahashi et al. [18] reported the utility of detecting a decreased lymphocyte count for early diagnosis of postoperative wound infection. In their prospective study, they measured WBC count and WBC differential in patients after spinal surgery with or without surgical-wound infection. Their results suggest that lymphopenia (no more than 10% or 1,000/µl) four days after surgery indicates possible surgicalwound infection.

Risk factors

It is important to know the risk factors for infection in order to use preventive measures and optimise surgical treatment [10]. Some factors cannot be modified, such as the patient's age. Cahill et al. [7] found that age has a significant inverse relationship with the risk of infection, with younger patients having a higher infection rate following scoliosis surgery. This suggests that the risk of infection has a bimodal age distribution, where the risk is lowest at some point in the teenage years. In a retrospective case control analysis of 48 cases of postoperative infection following spinal procedures, Fang et al. [10] found that for preoperative risk factors, age greater than 60 years, smoking, diabetes, previous infection, increased body mass index and alcohol abuse



were statistically significant preoperative risk factors. Previous surgery and steroid use did not appear to be predictive of infection. They found that for intraoperative risk factors, staged procedure, surgery time (over five hours) and the number of levels [22–27] were statistically significant intraoperative risk factors. They concluded that the most frequently infected procedure was likely to be a combined anterior/posterior spinal fusion performed in a staged manner under separate anaesthetics. In a multivariate analysis, Pull ter Gunne et al. [12] found that obesity was the only independent risk factor that increased the rate of superficial SSI. Approaches were associated with a 1.7% risk of SSI, and any surgery that included a posterior spinal approach was associated with a minimum of 4.4% risk of infection. The authors reported that improved vascularity and lymphatic drainage of the anterior spine may enhance the clearance of incidental bacterial contamination of an anterior spinal surgery. The rate of infection of the posterior approach depends on the nature of the procedure and after simple discectomy is approximately 1%, increases to 2-5% when there is a posterior spinal fusion and up to 9% with instrumentation.

Prevention

Antibiotic prophylaxis and precautions against risk factors are important to prevent infections after spinal surgery with instrumentation. Pull ter Gunne [12] found that an anterior approach was a protective factor. Isolated anterior surgical approaches were associated with a 1.7% risk of SSI, whereas any surgery that included a posterior spinal approach was associated with a minimum of 4.4% risk of infection. To address the complication of diabetes, Dubberke et al. [28] found that tight regulation of blood sugar pre-operatively may decrease the risk of SSI. To decrease the risk of infection when estimated blood loss (EBL) is greater than 1 l, some authors [12, 29] recommend minimising EBL in any surgical procedure, decreasing the need for nonautologous blood transfusions. They found that nonautologous blood transfusions produced immune suppression in the recipient. When the patient had a history of prior infection, Pull ter Gunne [12] attempted to determine the organism that caused the previous SSI and its antibiotic sensitivities in order to modify the antibiotic regimen. A meta-analysis was published by Barker [30] in which he evaluated six randomised controlled trials into which a total of 843 patients were enrolled. The difference between the raw pooled infection rate (2.2% in the antibiotic group and 5.9% in the no antibiotic group) was statistically significant. The results suggest that prophylaxis is beneficial in terms of reducing the incidence of operativesite infections following spinal surgery. Ho et al. [9] changed their antibiotic protocol from cefazolin to vancomycin and ceftazidime as prophylactic antibiotics for posterior spinal fusions in order to cover Staphylococcus epidermis (the most frequently found causative organism in their study). They use, in addition, jet lavage irrigation with detergent solution [31] before closure, with early results suggesting a much smaller infection rate. Another preventive measure was described by Cheng et al. [32] in a prospective, randomised study. They found that irrigation of the spinal wound with dilute Betadine solution completely prevented infection in a group of 208 patients compared with a 2.9% rate in 206 patients who did not have Betadine irrigation. Concerning obese patients, the Surgical Care Improvement Project (SCIP) advisory panel recommends a 2-g dose of cefazolin for prophylaxis in patients who weigh over 80 kg. Given the minimal side effects of cefazolin in nonallergic patients, it appears reasonable to give 2 g to all patients weighing more than 80 kg to decrease the risk of SSIs associated with obesity.

Treatment

Infection types

The treatment for an SSI following spinal surgery may depend on whether or not the SSI is isolated superficial to the muscular fascia or includes the spine deep to the fascia. Many treatments have been described. The first is aggressive surgical treatment and antibiotic therapy [1, 33]; another is removal of the instrumentation, with delayed reimplantation [33, 34]; a third is retention of the stable implant [4, 35]. Concerning the wound, some authors primarily close the wound following debridement, whereas others suggest either a second-look operation or leaving the wound open and closing it in a second stage. Pull ter Gunne and al [6] use treatment that included aggressive wound and soft-tissue debridement (89.3%), stable hardware retention (73.3%) and primary replacement of instrumentation (14.7%) if fixation failure had occurred. This was followed by primary closure over multiple drains. With this method, 76% of deep SSIs could be treated with a single surgical debridement. Further debridement was performed if clinical evidence of uncontrolled infection continued. All patients received antibiotic therapy for a mean of 40.8 days. Intravenously administered antibiotics were used in 90% of cases and were often followed by a short course of oral antibiotics. The time when the infection is diagnosed is used to differentiate between late and early infection; 90 days is used as the cutoff time by some authors, with early being before 90 days and late after 90 days. For infections that develop within the 90 days, Mok [1] and Cahill et al. [7] recommend debridement without



hardware removal followed by suppressive antibiotics until adequate fusion is obtained. For late infections, they recommend inspecting the fusion before instrumentation removal is considered.

Instrumentation retention versus removal

With regard to the treatment of delayed infection after spinal instrumentation, most studies reported successful treatment through complete implant removal associated with appropriate antibiotherapy [36, 37]. In the retrospective case series by Hedequist et al. [38], 26 patients developed a delayed infection that could not be eradicated when implants were left in place. Further debridement was always needed, until eventual implant removal allowed wounds to be closed. There was no need for further operations after implant removal in most cases. In some, there was one additional operation needed, to apply vacuum-assisted closure (VAC) dressing for a period of two or three days to create a granulated bed for closure. Patients who underwent initial irrigation and debridement and whose wounds were closed with implants left in place always returned with infected implants, either at the same or a later hospital admission. Hedequist et al. [38] explains that debridement of a chronic infection without implant removal leaves areas underneath rods and spinal anchor points that are not thoroughly debrided, and pockets of infected tissue and that immediate implant replacement no doubt would probably lead to early reinfection because of a colonised bed. The risk of instrumentation removal is the loss of deformity correction. Even in the face of what appears to be a solid fusion at exploration, removal of spinal implants is sometimes associated with deformity progression [39]. In that study, some patients needed revision surgery at a later date because the deformity progressed. Six of the patients were revised at an average of 16 months after infected implant removal. In patients that were not revised, there was no deformity progression or significant complaints requiring revision. For patients with delayed spinal infection without a completely destabilised spine, Hedequist et al. [38] recommend immediate implant removal at the first operation, with thorough debridement, and revision surgery if necessary at a later date. Buchowski et al. [40] presented a study of 69 infections in 2,876 adult and paediatric patients in which 39.1% of patients required instrumentation removal to treat their infection. For Ho et al. [9], 18.9% of a study of 53 infected paediatric patients required instrumentation removal. Hahn et al. [41] eradicated 100% of infections by implant removal.

Managing postoperative wound infections after instrumented arthrodesis involves a protocol of aggressive debridement and irrigation with subsequent returns to the operating room until the wound is sufficiently clean for closure. After closure, the appropriate antibiotics are administered for two to six weeks [4, 42–46]. Collins et al. [11] reported that nine (40%) of 15 patients treated with antibiotic therapy and wound debridement with implant retention had an active infection at the time of implant removal despite antibiotic therapy. Infection could not always be eradicated with implant retention. Sponseller et al. [47] found similar results: 21% of deep wound infections after neuromuscular scoliosis surgery had persistent wound discharge and healed only after implant removal. Glassman et al. [42] reported 19 cases of deep wound infection following 858 instrumented fusions, which were diagnosed at an average of 16 days postoperatively. The patients underwent on average 4.7 procedures (two to ten) before closure. Instrumentation was retained in all cases with a one year follow-up, and no sign of infection was found. Cahill et al. [7] reported 61 infections in 1,571 paediatrics patients. The number of re-operations was similar between patients with early and late infections, and instrumentation was more likely to be retained in early infections (less than 90 days after operation). The average number of reoperations for early and late infections was 2.0 and 1.9, respectively. Instrumentation was retained in 24 (75%) of 32 early and in four (13.8%) of 29late infections. Instrumentation retention was associated with a slight decrease in reoperation rate. In 51% (31 of 61) of infected patients, instrumentation was completely removed to treat the infection. The average number of reoperations was 2.3 in the 31 patients who had complete instrumentation removal and 1.6 in the 30 patients who did not undergo complete instrumentation removal. In contrast, in their series of paediatric scoliosis patients, Ho et al. [9] found that implant retention was a significant predictor of further surgery (P < 0.05): Forty-three patients had implants retained from initial irrigation and debridement, 47% (20 of 43) of these patients required a second irrigation and debridement and 12% (five of 43) required a third irrigation and debridement. Of the ten patients who had implants removed at initial irrigation and debridement, only 20% required a second irrigation and debridement, and no patient required a third irrigation and debridement. For infections that develop within 90 days, Mok [1] and Cahill et al. [7] recommend debridement without removal followed by suppressive antibiotics until adequate fusion is obtained. For the later infections, they recommend inspection of the fusion before instrumentation removal is considered.

Many studies report loss of correction following instrumentation removal at the time the infection is treated. Hahn et al. [41] noted a loss of curve correction ranging from 10° to 26° in three of six patients; however, a solid fusion was achieved after instrumentation removal for delayed infection. Rihn et al. [8], with seven infected patients of 236 following surgery for idiopathic scoliosis, had three with a loss of curve correction: on average, 22° (36% of correction) of the thoracic curve and 8° (21% of correction) of the lumbar curve. This loss of correction was caused by



pseudarthrosis at the time of surgical debridement and instrumentation removal. For the three other patients with solid fusion, there was no loss of correction despite instrumentation removal. None of the patients with pseudarthrosis at the time of surgical treatment underwent revision spinal surgery after infection eradication. Patients in the infection group had similar outcomes to those in the noninfected group, despite pseudarthrosis and loss of correction. However, many studies show that there is no correlation between the amount of postoperative improvement in radiographic curve correction and outcomes [48, 49].

Antibiotic therapy

Reviewing the literature regarding antibiotic therapy revealed a varying range of treatments and recommendations. Rihn et al. [8], in a retrospective study of seven infected patients, used intravenously administered antibiotics for a minimum of six weeks, which cleared the infection in all cases. Clark and Shufflebarger [13] treated delayed infection with surgical treatment and 48-72 hours of antibiotics i.v., followed by ten days of culture-guided antibiotic treatment by mouth. All infections were eradicated with this approach. Richards and Emara [50] proposed giving patients less than three weeks of antibiotic therapy after surgical treatment for delayed infection, which resolved in all cases. These authors recommend a two to five day course of antibiotics i.v. followed by a seven to 14-day course of antibiotics orally. This shows that surgical treatment, irrigation, debridement and hardware removal may be combined with short-term antibiotic treatment for delayed infection.

Closure

Some authors recommend leaving the wound open at the time of irrigation, with debridement and closure done in a delayed-staged fashion. Szoke et al. [51], for early deepwound infections, recommend leaving the wound open after the initial irrigation and debridement while leaving spinal instrumentation and bone graft in place. Wenger et al. [52] feel that if the diagnosis was delayed and the wound grossly purulent, the wound should be left open to heal by secondary intention via dressing changes. Sponseller et al. [47] propose this method if patients have extensive purulence, failure of prior treatment or poor soft tissue coverage, but seven of 14 patients managed this way had recurrent infection. Rihn et al. [8] reported that all patients with delayed infection were successfully treated with a single irrigation and debridement, instrumentation removal and primary closure over a Hemovac drain that was maintained for 48 hours.



A decrease in wound complications, improved healing times and reduced overall morbidity rates have been reported. In the retrospective study by Canavese et al. [53], the efficacy of vacuum-assisted closure (VAC) in treating deep infection after extensive instrumentation and fusion for spinal deformity in children and adolescents was reported. A total of 14 patients with early deep spinal infection were treated using this technique. The mean follow-up was 44 (24–72) months. All wounds healed; two patients required plastic surgery to speed up the process. In no patient was the hardware removed nor did loss of correction or recurrent infection occur. In 2005, Mehbod et al. [46] reported a series of 20 patients with postoperative spinal infections treated with the wound VAC system. In all patients, wounds healed without implant removal.

Closed suction irrigation system (CSIS)

According to Rohmiller et al. [54], a closed suction irrigation system (CSIS) is effective for treating postoperative wound infections following instrumented spinal fusion, thus avoiding the need for secondary closure. In their retrospective record review of 500 posterior instrumented fusions between 1990 and 2002, 28 consecutive infections (5%) were diagnosed and treated by a standardised incision and drainage treatment protocol and CSIS:. Twenty-one patients with acute and seven with late (over six months) infections were followed up for 22.3 months post-CSIS treatment: 21 (75%) resolved without recurrence with one CSIS treatment; seven acute infections (25%) required a second course of treatment. In the seven reinfected patients, five required only placement of a second CSIS, and two were treated with antibiotics alone. No patient with acute infection required implant removal.

Use of allograft

Ho et al. [9] found no significant association between the use of allograft and infection eradication: 50% (26 of 53) of patients had an allograft in their initial fusion, and of those requiring a second irrigation and debridement for persistent infection, 46% (ten of 22) had an allograft.

Clinical outcome

Patients with infection can have good to excellent clinical outcome, with no long-term loss of function [55, 56], similar to noninfected patients [1]. In their retrospective case—control study on 32 patients with deep wound infection, at mean



follow-up of 62 months, Mok et al. [1] reported no significant difference in Physical Function, Role Physical, Bodily Pain and General Health domains between infection and control groups in the SF-36 Health Survey. There were 12 early and four later infections, of which treatment was irrigation and debridement, repeated if needed. Instrumentation was retained. Mean Physical Component Summary was 41.4 in the infection group and 44.3 in the control group (P=0.6). Collins et al. [11] found poorer outcomes, reporting that only 46% of patients had stable pain free spines when implants were removed in established fusions.

Infection and pseudarthrosis

Cahill et al. [7] reported 61 infections in 1,571 paediatric patients, with a 25% incidence of pseudarthrosis. To treat the pseudarthrosis, they needed an average of 1.2 procedures, and seven patients had persistent pseudarthrosis at the final follow-up. Rihn et al. [8] reported seven patients with infection after surgical treatment for adolescent idiopathic scoliosis, of which one was acute and the others delayed. Treatment was irrigation and debridement with spinal implant removal; 50% of late infections demonstrated evidence of pseudarthrosis at the time of irrigation and debridement, but no patient with pseudarthrosis underwent any additional procedures to obtain a solid arthrodesis.

Conclusion

In conclusion, knowledge of factors involved, understanding the severity of infections and preventing a delayed diagnosis can be key factors in preventing SSIs.

References

- Mok JM, Guillaume TJ, Talu U et al (2009) Clinical outcome of deep wound infection after instrumented posterior spinal fusion: a matched cohort analysis. Spine 34:578–583
- Blam OG, Vaccaro AR, Vanichkachorn JS et al (2003) Risk factors for surgical site infection in the patient with spinal injury. Spine 28:1475–1480
- Kanafani ZA, Dakdouki GK, El-Dbouni O et al (2006) Surgical site infections following spinal surgery at a tertiary care center in Lebanon: incidence, microbiology, and risk factors. Scand J Infect Dis 38:589–592
- Weinstein MA, McCabe JP, Cammisa FP Jr (2000) Postoperative spinal wound infection: a review of 2391 consecutive index procedures. J Spinal Disord 13:422–426
- Kowalski TJ, Berbari EF, Huddleston PM et al (2006) Do follow-up imaging ex- aminations provide useful prognostic information in patients with spine infection. Clin Infect Dis 43:172–179

- Pull ter Gunne A, Mohamed A, Skolasky R et al (2010) The presentation, incidence, etiology, and treatment of surgical site infections after spinal surgery. Spine 35:1323–1328
- Cahill PJ, Warnick DE, Lee MJ et al (2010) Infection after spinal fusion for pediatric spinal deformity: thirty years of experience at a single institution. Spine 35:1211–1217
- Rihn JA, Lee JY, Ward WT (2008) Infection after the surgical treatment of adolescent idiopathic scoliosis: evaluation of the diagnosis, treatment, and impact on clinical outcomes. Spine 33:289–294
- Ho C, Skaggs DL, Weiss JM, Tolo VT (2007) Management of infection after instrumented posterior spine fusion in pediatric scoliosis. Spine 32:2739–2744
- Fang A, Hu SS, Endres N, Bradford DS (2005) Risk factors for infection after spinal surgery. Spine 30:1460–1465
- Collins I, Wilson-MacDonald J, Chami G et al (2008) The diagnosis and management of infection following instrumented spinal fusion. Eur Spine J 17:445–450
- Pull ter Gunne A, Cohen D (2009) Incidence, prevalence, and analysis of risk factors for surgical site infection following adult spinal surgery. Spine 34:1422–1428
- Clark C, Shufflebarger H (1999) Late-developing infection in instrumented idiopathic scoliosis. Spine 24(18):1909–1916
- Richards BS (1995) Delayed infections following posterior spinal instrumentation for the treatment of idiopathic scoliosis. J Bone Joint Surg Am 77-A(4):524–529
- Schofferman L, Zucherman J, Schofferman J et al (1991) Diptheroids and associated infections as a cause of failed instrument stabilization procedures in the lumbar spine. Spine 16(3):356
 358
- Bémer P, Corvec S, Tariel S (2008) Significance of propionibacterium acnes-positive samples in spinal instrumentation. Spine 33: E971–E976
- Thelander U, Larsson S (1992) Quantitation of C-reactive protein levels and eryth- rocyte sedimentation rate after spinal surgery. Spine 17:400–404
- Takahashi J, Ebara S, Kamimura M et al (2001) Early-phase enhanced inflammatory reaction after spinal instrumentation surgery. Spine 26:1698–1704
- Yang MD, Jeng LB, Kao A et al (2003) C-reactive protein and gallium scintigraphy in patients after abdominal surgery. Hepatogastroenterology 50:354

 –356
- Bourguignat A, Ferard G, Jenny JY et al (1996) Diagnostic value of C-reactive protein and transthyretin in bone infections of the lower limb. Clin Chim Acta 255:27–38
- Takahashi J, Shono Y, Hirabayashi H et al (2006) Usefulness of white blood cell differential for early diagnosis of surgical wound infection following spinal instrumentation surgery. Spine 31:1020– 1025
- Abbey DM, Turner DM, Warson JS et al (1995) Treatment of postoperative wound infections following spinal fusion with instrumentation. J Spinal Dis- ord 8:278–283
- Calderone RR, Garland DE, Capen DA et al (1996) Cost of medical care for postoperative spinal infections. Orthop Clin North Am 27:171–182
- Wimmer C, Gluch H, Franzreb M et al (1998) Predisposing factors for infection in spine surgery: a survey of 850 spinal procedures. J Spinal Disord 11:124–128
- Perry JW, Montgomerie JZ, Swank S et al (1997) Wound infections following spinal fusion with posterior segmental spinal instrumentation. Clin Infect Dis 24:558–561
- Thalgott JS, Cotler HB, Sasso RC et al (1991) Postoperative infections in spinal implants: classification and analysis—a multicenter study. Spine 16:981–984
- Sculco TP (1995) The economic impact of infected joint arthroplasty. Orthopedics 18:871–873



- Dubberke ER, Reske KA, Yan Y et al (2007) Clostridium difficile associated disease in a setting of endemicity: identification of novel risk factors. Clin Infect Dis 45:1543–1549
- Quintiliani L, Pescini A, Di Girolamo M et al (1997) Relationship of blood transfusion, post-operative infections and immunoreactivity in patients undergoing surgery for gastrointestinal cancer. Haematologica 82:318–323
- Barker FG II (2002) Efficacy of prophylactic antibiotic therapy in spinal surgery: a meta-analysis. Neurosurgery 51:391–401
- Bhandari M, Adili A, Schemitsch EH (2001) The efficacy of lowpressure lavage with different irrigating solutions to remove adherent bacteria from bone. J Bone Joint Surg Am 83:412–419
- Cheng MT, Chang MC, Wang ST et al (2005) Efficacy of dilute betadine solution irrigation in the prevention of postoperative infection of spinal surgery. Spine 30:1689–1693
- Tsiodras S, Falagas ME (2006) Clinical assessment and medical treatment of spine infections. Clin Orthop Relat Res 444:38–50
- 34. Bose B (2003) Delayed infection after instrumented spine surgery: case reports and review of the literature. Spine J 3:394–399
- Ido K, Shimizu K, Nakayama Y et al (1996) Suction/irrigation for deep wound infection after spinal instrumentation: a case study. Eur Spine J 5:345–349
- Lutz MF, Berthelot P, Fresard A et al (2005) Arthroplastic and osteosynthetic infections due to Propionibacterium acnes: a retrospective study of 52 cases, 1995–2002. Eur J Clin Microbiol Infect Dis 24:739–744
- Zeller V, Ghorbani A, Strady C et al (2007) Propionibacterium acnes: an agent of prosthetic joint infection and colonization. J Infect. doi: j.jinf.2007./j.jinf.2007.02.006
- 38. Hedequist D, Haugen A, Hresko T, Emans J (2008) Failure of attempted implant retention in spinal deformity delayed surgical site infections. Spine 34:60–64
- Potter BK, Kirk KL, Shah SA et al (2006) Loss of coronal correction following instrumentation removal in adolescent idiopathic scoliosis. Spine 31:67–72
- Buchowski JM, Lenke LG, Kuhns CA et al (2006) Infections following spinal deformity surgery. A twenty-year assessment of 2876 patients [paper #34]. Presented at the Scoliosis Research Society 41st Annual Meeting, Monterey, CA, September 13–16
- Hahn F, Zbinden R, Min K (2005) Late implant infections caused by propionibacterium acnes in scoliosis surgery. Eur Spine J 14 (8):783–788
- Glassman SD, Dimar JR, Puno RM et al (1996) Salvage of instrumental lumbar fusions complicated by surgical wound infection. Spine 21:2163–2169

- 43. Picada R, Winter RB, Lonstein JE et al (2000) Postoperative deep wound infection in adults after posterior lumbosacral spine fusion with instrumentation: incidence and management. J Spinal Disord 13:42–45
- 44. Stambough JL, Beringer D (1992) Postoperative wound infections complicating adult spine surgery. J Spinal Disord 5:277–285
- Wimmer C, Gluch H (1996) Management of postoperative wound infection in posterior spinal fusion with instrumentation. J Spinal Disord 9:505–508
- 46. Mehbod AA, Ogilvie JW, Pinto MR et al (2005) Postoperative deep infections in adults after spinal fusion: management with vacuum- assisted wound closure. J Spinal Disord Tech 18:14–17
- Sponseller P, LaPorte DM, Hungerford MW et al (2000) Deep wound infections after neuromuscular scoliosis surgery: a multicenter study of risk factors and treatment outcomes. Spine 25:2461–2466
- Merol AA, Haher TR, Brkaric M et al (2002) A multicenter study of the outcomes of the surgical treatment of adolescent idiopathic scoliosis using the Scoliosis Research Society (SRS) outcome instrument. Spine 27:2046–2051
- 49. Wilson PL, Newton PO, Wenger DR et al (2002) A multicenter study analyzing the relationship of a standardized radiographic scoring system of adolescent idiopathic scoliosis and the Scoliosis Research Society outcomes instrument. Spine 27:2036–2040
- Richards BR, Emara KM (2001) Delayed infections after posterior TSRH spinal instrumentation for idiopathic scoliosis: revisited. Spine 26:1990–1996
- Szoke G, Lipton G, Miller F et al (1998) Wound infection after spinal fusion in children with cerebral palsy. J Pediatr Orthop 18:727–733
- Wenger DR, Mubarak SJ, Leach J (1992) Managing complications of posterior spinal instrumentation and fusion. Clin Orthop Relat Res 284:24–33
- Canavese F, Gupta S, Krajbich JI, Emara KM (2008) Vacuumassisted closure for deep infection after spinal instrumentation for scoliosis. J Bone Joint Surg Br 90-B:377–381
- Rohmiller MT, Akbarnia BA, Raiszadeh K et al (2010) Closed suction irrigation for the treatment of postoperative wound infections following posterior spinal fusion and instrumentation. Spine 35:642–646
- 55. Beiner JM, Grauer J, Kwon BK et al (2003) Postoperative wound infections of the spine. Neurosurg Focus 15:E14
- Pappou IP, Papadopoulos EC, Sama AA et al (2006) Postoperative infections in interbody fusion for degenerative spinal disease. Clin Orthop 444:120–128

