Surgical site infection after pediatric spinal deformity surgery

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Abstract The incidence of surgical site infection (SSI) after spinal deformity surgery for adolescent idiopathic scoliosis ranges from 0.5–6.7%. The risk of infection following spinal fusion in patients with neuromuscular scoliosis is greater, with reported rates of 6.1–15.2% for cerebral palsy and 8–41.7% for myelodysplasia. SSIs result in increased patient morbidity, multiple operations, prolonged hospital stays, and significant financial costs. Recent literature has focused on elucidating the most common organisms involved in SSIs, as well as identifying modifiable risk factors and prevention strategies that may decrease the rates of infection. These include malnutrition, positive urine cultures, antibiotic prophylaxis, surgical site antisepsis, antibiotic-loaded allograft, local application of antibiotics, and irrigation solutions. Acute and delayed SSIs are managed differently. Removal of instrumentation is required for effective treatment of delayed SSIs. This review article examines the current literature on the prevention and management of SSIs after pediatric spinal deformity surgery.

Keywords Spinal fusion · Spinal surgery · Scoliosis · Spinal deformity · Infection · Complications · Surgical site infection · Spinal infection · Pediatric · Risk factors · Prevention · Instrumentation · Infected spinal instrumentation · Implant removal

Introduction

Surgical site infection (SSI) after pediatric spinal deformity surgery can have a substantial medical, social, and financial impact on patients. Prevention of SSI after spinal fusion is currently a major area of interest as new regulations may lead to altered reimbursements to hospitals for surgical complications. The Centers for Disease Control (CDC) has published specific definitions for SSIs [1]. The incidence of SSI varies depending on patient diagnosis. The risk of infection after surgery for adolescent idiopathic scoliosis (AIS) is much lower than the risk of infection after spinal fusion in patients with cerebral palsy or myelodysplasia.

Numerous authors have reported potential risk factors for SSI after pediatric spinal deformity surgery. Certain patient-related risk factors, such as underlying medical condition and previous surgery, are not modifiable. Recent literature has focused on identifying modifiable risk factors and prevention strategies that may decrease the rates of SSI after spinal fusion. Treatment and outcomes differ for superficial versus deep SSIs. Most authors advocate removal of instrumentation for delayed deep SSIs, but the timing of implant removal is controversial as the definition for a delayed SSI is inconsistent in the literature [2, 3, 4–6].

The purpose of this article is to examine the recent literature on the prevention and management of SSIs after

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pediatric spinal deformity surgery. This review will focus on deep SSIs, as these can result in much greater and long-lasting patient morbidity.

Definitions of SSIs

The CDC has published definitions for different types of SSIs. A superficial incisional SSI involves only the skin or subcutaneous tissues and occurs within 30 days after the procedure. Additional criteria include purulent drainage, positive cultures from fluid or tissue obtained sterilely from the superficial incision, pain or tenderness, localized swelling, erythema, and warmth. A stitch abscess is not considered a superficial SSI. A deep incisional SSI involves the fascia and muscle layers. It occurs within 30 days after the procedure if no implant is placed, or within 1 year if an implant is placed and the infection appears to be related to the procedure. Additional criteria include purulent drainage, deep wound dehiscence, fever, pain or tenderness, and presence of an abscess or other evidence of infection diagnosed by direct examination, radiographs, or cultures [1]. Deep SSI after spinal deformity surgery can be further classified into acute and delayed infection. The definition of a delayed SSI after spinal fusion is controversial and has been described as greater than 1 month [7], 2 months [8, 9], 3 months [3•, 10•], 6 months [6], and 1 year [2] after the initial procedure.

Incidence of SSI

The incidence of SSI after pediatric spinal deformity surgery varies depending on patient diagnosis. Reported rates of infection after surgery for AIS range from 0.5 to 6.7% [2, 4, 5, 10•, 11–17, 18•]. The risk of SSI after fusion for Scheuermann's kyphosis ranges from 3.8 to 4.3% [19]. The incidence of infection following surgery for neuromuscular scoliosis is greater, with an overall rate of 4.3–14.3% [8, 10•, 11, 16, 18•, 20, 21]. Rates of infection after spinal deformity surgery for myelodysplasia range from 8 to 41.7% [7, 10•, 22–27, 28•], whereas rates for cerebral palsy range from 6.1 to 15.2% [9, 10•, 29, 30•, 31–34].

Risk factors

Patient-related risk factors

Linam et al. retrospectively examined underlying health status as a potential risk factor for SSI after pediatric posterior spinal fusion (PSF). Obesity (body mass index >95th percentile) and American Society of Anesthesiologists (ASA) score greater than 2 were independent risk factors associated with SSI. Neuromuscular patients were more likely to have an ASA score greater than 2 [35]. Similarly, Aleissa et

al. found a significantly higher risk of infection in patients with non-idiopathic diagnoses who had undergone spinal fusion for scoliosis [11]. Sponseller et al. demonstrated that degree of cognitive impairment was a significant risk factor for deep SSI after scoliosis surgery in patients with cerebral palsy and myelodysplasia. In this multicenter retrospective case-control study, 32% of the patients who developed an infection had severe mental retardation, compared with only 2% of the patients who did not develop an infection [21]. Szoke et al. reported similar results when he reviewed 172 patients with cerebral palsy and scoliosis who had undergone fusion with unit rod instrumentation. All of the infections occurred in spastic quadriplegics with severe neurologic involvement, severe mental retardation, seizure disorders, and no ambulatory ability [9].

Both Jevsevar and Karlin, and Hatlen et al. have reported an increased risk of SSI after spinal fusion in neuromuscular patients with poor preoperative nutrition [28•, 36]. Jevsevar and Karlin found a lower rate of overall infection, including SSI, in patients with cerebral palsy with a preoperative albumin ≥ 3.5 mg/dL and total lymphocyte count ≥ 1500 cells/mm³ [36]. Similarly, Hatlen et al. demonstrated a significantly increased risk of SSI in patients with myelodysplasia with a hematocrit ≤ 33 g/L, which was their marker of malnutrition [28•]. Both authors recommended determining the preoperative nutritional status of neuromuscular patients and taking aggressive measures to improve nutrition prior to surgery [28•, 36]. However, Sponseller et al. did not find that preoperative malnutrition was significantly correlated with an increased risk of deep SSI [21].

Hatlen et al. demonstrated that a positive preoperative urine culture is a significant independent risk factor for SSI after spinal fusion in myelodysplasia patients. Eighty percent of patients with adequate preoperative nutrition and positive preoperative urine cultures developed SSIs. Two-thirds of these patients had a SSI caused by the same organism found in their preoperative urine cultures [28•]. Up to two-thirds of myelodysplasia patients have neurogenic bladders [37], leading to increased likelihood of chronic bacterial colonization and recurrent urinary tract infections. Hatlen et al. suggested checking preoperative urine cultures in myelodysplasia patients and treating positive cultures with antibiotics prior to surgery [28•].

Surgery-related risk factors

Surgical approach is associated with risk of SSI after spinal fusion. Infection after PSF is most common, whereas infection after anterior spinal fusion is rare. In patients who have undergone combined anterior and posterior spinal fusion, infection typically occurs only in the posterior wound [7, 11, 15].

Both Sponseller et al. and Aleissa et al. found that use of allograft bone was a significant risk factor for SSI after scoliosis surgery, particularly in neuromuscular patients [11, 21]. In Sponseller et al.'s series of cerebral palsy and myelodysplasia patients, 68% of patients in the infected group received allograft bone, compared with only 16% of patients in the uninfected group [21]. Aleissa et al. retrospectively reviewed 227 patients with a variety of diagnoses who had undergone spinal fusion for scoliosis. None of the neuromuscular patients who received autograft bone alone developed an infection, whereas 18% of the neuromuscular patients who received allograft bone developed an infection. The authors postulated that host defenses may be overwhelmed by the presence of a large amount of devitalized bone in patients who are already relatively immunocompromised [11]. In contrast, Master et al. did not find that use of allograft bone correlated with increased risk of deep SSI in the neuromuscular population [8].

Sponseller et al. found a significantly higher risk of SSI after scoliosis surgery in cerebral palsy patients who had undergone instrumentation with unit rods versus custom bent rods (15% vs 5%). Implant prominence, especially proximally, was significantly more common with unit rods. The authors hypothesized that unit rod instrumentation requires extensive soft tissue dissection to make the transition from the iliac wings to the spine. The resulting soft tissue trauma may lead to a higher risk of infection [38]. Mohamed Ali et al. retrospectively reviewed 236 cerebral palsy patients who had undergone unit rod instrumentation, and found that skin breakdown due to the instrumentation was a significant independent risk factor for a deep SSI. The proximal end of the unit rod was the most common location of breakdown [29].

Both Soultanis et al. and Di Silvestre et al. demonstrated a higher risk of late deep SSI after PSF in patients with AIS who received stainless steel implants versus titanium implants [13, 39]. In Soultanis et al.'s series, 12% of patients in the stainless steel group developed an infection, compared with 2% in the titanium group. There was significantly more inflammatory tissue and signs of metallosis with the stainless steel implants at the time of surgical debridement and implant removal [39]. Similarly, Di Silvestre et al. reported a 4.6% versus 1.3% rate of infection in their stainless steel and titanium groups, respectively [13]. Aleissa et al. found a significantly higher risk of infection in patients with a larger volume of instrumentation [11].

Ho et al. reported that receiving a blood transfusion and greater number of units of blood transfused were significant independent risk factors for delayed SSI after PSF for AIS [14]. This differs from Master et al. and Linam et al.'s findings, where transfusion requirement [8] and volume of blood products transfused [35] were not found to be significant risk factors. Ho et al. also demonstrated that failure to use a drain after surgery was a significant independent risk factor for delayed SSI. Patients who did not receive a drain were 3 times

more likely to develop an infection [14]. However, Diab et al. [40] did not find a difference in infection rate with drain use after surgery for AIS.

Most common infecting organisms

Gram-positive organisms, such as *Staphylococcus aureus* and *Staphylococcus epidermidis*, have traditionally been reported to be the most common organisms responsible for deep SSI after spinal deformity surgery [3•, 6–9, 10•, 11, 13, 15, 20, 34, 35, 41]. However, recent literature shows that gram-negative infections may be more common than previously thought, especially in the non-idiopathic population [7, 11, 18•, 30•, 35, 42]. *Pseudomonas* has been reported to be the most common isolate after *S. aureus* and *S. epidermidis* in several studies [7, 10•, 18•, 30•, 35]. Vitale et al. found that 46% of SSIs after scoliosis surgery involved at least one gram-negative organism, and patients with non-idiopathic scoliosis had a significantly higher rate of gram-negative SSIs compared to patients with idiopathic scoliosis (50.8% vs 7.7%) [18•]. Similarly, Sponseller et al. demonstrated that the number of gram-negative SSIs was equal or nearly equal to the number of gram-positive SSIs after scoliosis surgery in neuromuscular patients [21, 30•]. This is likely secondary to contamination from bowel and bladder incontinence. Brook et al. found that normal gastrointestinal flora were the most common organisms isolated from SSIs after scoliosis surgery. Only 11% of the patients who developed a SSI in their series had idiopathic scoliosis [42].

Low-virulent skin flora, such as *S. epidermidis* and *Propionibacterium acnes*, have become more recognized as a cause of delayed SSI after spinal deformity surgery [2, 4, 5]. Delayed SSI can occur from either hematogenous or intraoperative seeding. Intraoperative seeding occurs when low-virulent skin organisms are introduced into the wound at the time of surgery. This is followed by a subclinical quiescent period and activation at a later time [2, 4, 5]. Dietz et al. obtained positive wound cultures in greater than half of patients undergoing clean, elective orthopaedic procedures with no antibiotic prophylaxis. Coagulase-negative *Staphylococcus* was found in 58% of the cultures and *P. acnes* was found in 24%. The authors suggested that these normal skin flora were introduced into the field at the time of surgery [43]. Nandyala and Schwend reported a 23% rate of positive tissue cultures from pediatric patients undergoing PSF. Significant risk factors for intraoperative wound contamination were neuromuscular scoliosis with fusion to the pelvis, duration of surgery greater than 6 h, and patients over 11 years of age. *P. acnes* was the most common organism (69%), followed by *Staphylococcus* species. All of the cases of *P. acnes* involved adolescents 11 years and older, and back acne was a significant association. An acute SSI developed in 2.6%

of patients and all had positive cultures at the time of the index procedure. The authors proposed preoperative showering with an antimicrobial wash and a dermatology consultation for adolescents with back acne to lower the risk of intraoperative seeding of skin organisms [44•]. Richards and Emara recommended instituting barriers to skin flora during spinal surgery by performing a wide preparation, sealing the surgical field with an iodine-impregnated adherent plastic drape, avoiding extension of the incisions to the edges of the drape to prevent any violation of the barrier, and using frequent irrigation throughout the procedure. They also hypothesized that prominent instrumentation, such as cross-links, can lead to bursa formation, which may provide an environment that is conducive to growth of low-virulent organisms and subsequent development of a delayed SSI [4].

Both *S. epidermidis* and *P. acnes* may require prolonged incubation periods. *P. acnes* may necessitate an incubation period of 10–14 days. If final cultures are reported at 72 h, the organism may be missed [2, 4, 5]. Clark and Shufflebarger retrospectively reviewed 22 patients who developed a delayed SSI after PSF for AIS. These authors defined a delayed SSI as occurring greater than 1 year postoperatively. Cultures were continued for only 72 h in their first 10 patients and 90% of these were negative. Cultures were subsequently continued for 7 days in their last 12 patients and 92% of these were positive. *S. epidermidis* was found in 6 cultures and *P. acnes* was found in 3 cultures [2]. Richards and Emara recommended incubating cultures for up to 10 days, and not to attribute a delayed positive result for *S. epidermidis* or *P. acnes* to a contaminated specimen [4].

Antibiotic prophylaxis

Cefazolin or clindamycin in penicillin-allergic patients is the standard preoperative antibiotic prophylaxis for spinal deformity surgery. Linam et al. found that clindamycin as antibiotic prophylaxis was an independent risk factor associated with SSI after pediatric PSF. This was not related to inappropriate dosing, timing, or intraoperative redosing. The authors found a higher rate of gram-negative infections in their heterogeneous patient population compared with previous studies. They suggested that clindamycin alone may not be adequate prophylaxis for surgeries in which gram-negative organisms are common pathogens [35]. Some authors have proposed broadening coverage to include gram-negative organisms, although no studies to date have shown that additional antibiotic prophylaxis against gram-negative bacteria lowers the rate of SSI after pediatric spinal fusion [7, 11, 18•, 42]. Non-idiopathic patients, especially those who are nonambulatory and have bowel and bladder incontinence, may benefit from gram-negative antibiotic coverage, such as with gentamicin [7, 11, 18•]. In a survey of members of the Pediatric Orthopaedic

Society of North America and the Scoliosis Research Society, Glotzbecker et al. found that 48% of surgeons added gram-negative antibiotic prophylaxis for neuromuscular patients undergoing spinal deformity surgery.[unpublished data] It is unclear whether antibiotic prophylaxis should be broadened to cover *S. epidermidis* and *P. acnes*. Resistance of both *S. epidermidis* and *P. acnes* against cephalosporins, and *P. acnes* against clindamycin has been reported [45, 46].

Inappropriate timing of preoperative antibiotic prophylaxis has been found to be a significant independent risk factor for deep SSI after spinal fusion. Milstone et al. defined inappropriate timing as administration of antibiotics greater than 60 min prior to incision, given after incision, or not given at all. The authors reported a 3.5-fold greater risk of SSI when antibiotics were administered at an inappropriate time. No correlation was found between inappropriate preoperative antibiotic dosing or inadequate intraoperative redosing and risk of SSI [47]. The appropriate duration of postoperative antibiotics is debatable. Takemoto et al. showed that continuing postoperative antibiotics for the duration that a drain is in place does not decrease the risk of acute SSI after thoracolumbar spine surgery compared to continuing antibiotics for 24 h [48•].

Surgical site antisepsis

No study has examined preoperative surgical site antisepsis in pediatric spinal deformity surgery. Darouiche et al. conducted a multicenter, prospective, randomized clinical trial comparing chlorhexidine-alcohol and povidone-iodine in adults undergoing clean-contaminated gastrointestinal, thoracic, gynecologic, and urologic procedures. The rates of superficial and deep SSI were significantly lower in the chlorhexidine-alcohol group. The authors attributed their findings to chlorhexidine-alcohol's more rapid action, persistent activity despite exposure to bodily fluids, and residual effect [49•]. It is unclear whether the results of this study can be applied to pediatric spinal deformity surgery.

Local application of antibiotics

Addition of antibiotics to allograft bone and application of antibiotic powder to the wound have both been shown to reduce rates of SSI after spinal fusion. These techniques allow high concentrations of antibiotics to be delivered locally with limited systemic toxicity. Borkhuu et al. retrospectively reviewed 220 patients with cerebral palsy who had undergone PSF with a unit rod. All patients received plain freeze-dried corticocancellous allograft bone. One hundred fifty-four patients received an additional 30 to 60 cc of allograft bone soaked with liquid gentamicin at a dose of 8 to 10 mg/kg of body weight. The plain allograft group had a significantly greater rate of acute deep SSI

compared with the antibiotic-loaded allograft group (15.2% vs 3.9%). No adverse outcomes were attributed to the use of the gentamicin [34].

Several authors have demonstrated a significantly decreased risk of deep SSI after spinal fusion in adults with application of vancomycin powder to the wound [50, 51–53]. Sweet et al. retrospectively reviewed 1,732 adults who had undergone instrumented thoracolumbar fusion. All patients received standard preoperative prophylaxis with cefazolin. Half of the patients also received 1 g of vancomycin powder mixed in with the autogenous bone graft and 1 g of vancomycin powder spread throughout the wound. The rate of deep infection was significantly higher in the group that did not receive vancomycin (2.6% vs 0.2%). Postoperative serum vancomycin levels were drawn on 20% of the patients in the vancomycin group. Eighty percent of these patients had undetectable serum vancomycin levels, which suggests a low risk of systemic toxicity and development of resistant organisms at other body sites with local application of vancomycin. No complications resulted from the use of vancomycin powder [50]. Similarly, O'Neill et al. examined 110 adults who had undergone PSF for traumatic injury and found a 0% versus 9% rate of deep SSI (13% overall infection rate) in the vancomycin powder and standard antibiotic prophylaxis groups, respectively [51]. Rahman et al. also reported a significantly lower rate of acute deep SSI after fusion for adult spinal deformity when vancomycin powder was used (0.7% vs 5%) [53]. It is uncertain whether these findings are applicable to the pediatric population.

Irrigation solutions

Dilute povidone-iodine irrigation may be effective at reducing the rate of SSI. A recent meta-analysis suggested a lower rate of SSI after various types of surgery with the use of povidone-iodine irrigation [54]. Multiple authors have investigated the effect of dilute povidone-iodine irrigation solution on the risk of SSI after spinal surgery in adults [55, 56, 57]. Povidone-iodine has a bactericidal effect on a wide range of organisms, including methicillin-resistant *S. aureus* (MRSA). Maximum effectiveness against MRSA has been demonstrated with povidone-iodine concentrations of 0.05% to 0.4% [58]. Commercially available betadine solution contains 10% povidone-iodine. Betadine concentrations less than 5% appear to be non-toxic towards osteoblasts. Schmidlin et al. demonstrated that 10% betadine led to decreased viability and loss of differentiation of osteoblast-like cells [59]. Bhandari et al. also showed that betadine concentrations of 10% and higher had significant negative effects on mouse calvarial osteoblasts and osteoclasts [60]. However, Kaysinger et al. did not find any toxic effects on cultured chick tibiae and osteoblasts with betadine concentrations of less than 5% [61]. Cheng et al. performed

a prospective, randomized study on 414 adults undergoing spinal surgery. Half of the patients had their wounds soaked with 3.5% betadine solution for 3 min followed by irrigation with 2 l of normal saline prior to decortication and addition of autograft bone. The other half of the patients only had irrigation with 2 l of normal saline. No infections occurred in the treatment group, compared with a 2.9% rate of deep SSI in the control group. The authors did not find a significant effect of the betadine on time to fusion. No patients had an adverse or allergic reaction [56]. Chang et al. also conducted a prospective, randomized study on 244 adults undergoing PSF and found a 4.8% rate of deep SSI in the normal saline group, compared to no infections in the betadine group [55]. Similarly, Hardacker and Hardacker reported a significantly lower rate of deep SSI after adult PSF when dilute betadine irrigation was used (4% versus 0.7%) [57]. Again, it is unknown whether these results can be applied to pediatric patients undergoing spinal fusion.

Bhandari et al. compared different irrigation solutions at various concentrations, including povidone-iodine, chlorhexidine gluconate, and soap. They found that 1% soap solution resulted in the greatest preservation of osteoblast and osteoclast number, as well as osteoblast function. Low-pressure irrigation with 1% soap solution also led to complete removal of bacteria from bone. The authors explained soap's superior activity secondary to its ability to form micelles, which have hydrophobic and hydrophilic ends. The hydrophilic ends surround bacteria and prevent bacteria from adhering to bone [60]. This was an in vitro study and it is unclear whether the results can be reproduced in the clinical setting. The trauma literature has also demonstrated that low-pressure irrigation may be preferable to high-pressure irrigation. Several in vitro studies have demonstrated evidence of damage to bone [62–65], decreased new bone formation [64, 66], increased bacterial seeding in the intramedullary canal [62], and deeper bacterial penetration in soft tissues resulting in reduced bacterial clearance [67] with high-pressure pulsatile lavage.

Management of acute deep SSIs

Acute deep SSIs following spinal deformity surgery can be treated with aggressive irrigation and debridement, retention of instrumentation, and long-term parenteral and oral antibiotics [3, 20, 41, 68]. Implant removal is avoided during the acute postoperative period secondary to concern for deformity progression. Duration of antibiotic treatment varies, but parenteral antibiotics should be continued for a minimum of 4 to 6 weeks [3, 20, 41]. Subsequent treatment with oral antibiotics may be necessary for 2 to 6 months [20, 41].

Some authors recommend primary wound closure over drains after debridement [3]. Others have reported success

with use of the vacuum-assisted closure (VAC) system [20, 41]. The VAC promotes formation of granulation tissue, debrides necrotic tissue, and acts as a sterile dressing. Canavese et al. described application of a VAC sponge at the time of initial surgical debridement in 14 patients who developed an acute deep SSI after spinal fusion. Twelve of the wounds healed by secondary intention with the VAC. Removal of instrumentation was not necessary in any of the patients and there were no recurrent infections [41]. Van Rhee et al. published similar findings in 6 patients who developed an acute deep SSI after PSF. All patients in both studies received long-term parenteral and oral antibiotics [20, 41].

Rohmiller et al. described a closed suction irrigation system placed at the time of initial debridement for deep SSI. One proximal inflow catheter was placed deep to the fascia, and 2 to 3 distal outflow catheters were placed superficial and deep to the fascia. The wound was irrigated with normal saline for approximately 3 days. The catheters were removed sequentially as the drainage became clear and decreased in volume. Two-thirds of acute SSIs were treated successfully with this method. One third of patients developed a recurrent infection, which resolved after a second course of closed suction irrigation management. No patients required implant removal [68].

Management of delayed deep SSIs

Most authors agree that implant removal is necessary for the effective treatment of delayed deep SSIs after spinal deformity surgery [2, 3, 4–6, 13, 15]. Bacteria that infect metallic implants are surrounded by glycocalyx, a polysaccharide membrane that enables adherence to surfaces and provides resistance to macrophage attack and antibiotic penetration [69]. Once bacteria have formed this protective biofilm, complete removal of instrumentation is the only way to eliminate the infection. After all instrumentation is removed, the wound can be closed primarily over drains [2, 3, 4–6, 13, 15] and a short course of parenteral and oral antibiotics is sufficient [2, 4, 5, 15]. Both Clark and Shufflebarger, and Richards and Emara reported that deep SSIs after spinal fusion are soft tissue infections and not osteomyelitis. When they inspected the soft tissue and bone surrounding the implants, there was no sequestrum and the fusion mass was viable [2, 4]. This explains why short-term antibiotics are adequate after instrumentation removal. These authors recommended 2 to 5 days of parenteral antibiotics, followed by 7 to 14 days of oral antibiotics [4]. Several authors have reported success with this treatment protocol [2, 3, 6, 13, 15].

Hedequist et al. retrospectively reviewed 26 patients who developed a delayed SSI after spinal fusion. They found that patients who retained their instrumentation always returned with recurrent infection and required further debridements

until the implants were removed. In most cases, no repeat surgeries were necessary after instrumentation removal. Occasionally, patients with significant comorbidities underwent placement of a VAC at the initial debridement to promote formation of granulation tissue. These patients required one additional procedure for VAC removal and wound closure. The number of hospitalizations, number of hospital days, number of procedures, and cost of hospitalization correlated with the number of debridements before instrumentation removal. Average hospital charges if implants were removed during the first procedure were \$81,828, compared to \$101,590 if implants were removed within 3 procedures and \$674,292 if implants were removed after 4 or more procedures [3].

Ho et al. retrospectively examined 53 patients who developed a deep SSI after PSF for scoliosis. All patients underwent debridement, primary closure over drains, and antibiotics. Instrumentation was initially retained in 97% of acute SSIs and 59% of delayed SSIs. The authors found that retention of instrumentation was a significant predictor of further surgery. Of the 43 patients who retained their instrumentation after initial irrigation and debridement, 47% developed recurrent infection and had a second irrigation and debridement, and 12% required a third operation [6]. Ho et al.'s results suggest that perhaps removal of instrumentation should be considered in acute deep SSIs as well.

Implant removal prior to bony fusion can lead to progression of deformity [3, 4, 6, 10, 15]. Cahill et al. retrospectively reviewed 57 patients who developed a SSI after scoliosis surgery. Instrumentation was removed in 51% of patients. Forty-four percent of these patients developed greater than 10° of curve progression. The patients who underwent implant removal within 1 year of the initial surgery had an average of 30° of progression, compared with 20° for those who underwent removal greater than 1 year postoperatively [10]. Twenty-three percent of the patients in Hedequist et al.'s series underwent revision surgery for curve progression [3]. Ho et al. had limited follow-up on their patients who had implants removed, but 60% had greater than 10° of deformity progression in at least one plane [6]. Not all pseudarthroses are evident at the time of instrumentation removal and not all pseudarthroses result in progressive deformity. However, patients and families should be counseled about the possibility of curve progression, especially if implants are removed less than 1 year postoperatively. They should be advised that future revision surgery may be necessary once the infection has cleared.

Conclusion

SSIs after pediatric spinal deformity surgery can result in significant medical, social, and financial costs. Certain risk

factors are modifiable and different prevention strategies may decrease the rates of SSI. Efforts should be made to correct preoperative malnutrition and treat positive urine cultures, particularly in neuromuscular patients. Preoperative antibiotic prophylaxis regimens may need to be broadened to more adequately cover gram-negative organisms, *S. epidermidis*, and *P. acnes*. Surgical site antisepsis with chlorhexidine-alcohol may be superior to povidone-iodine. Gentamicin-loaded allograft may reduce the risk of SSI in the neuromuscular population. Application of vancomycin powder to the wound, and irrigation with dilute betadine and soap solutions may be beneficial. Acute and delayed SSIs are managed differently. Implant removal is required for effective treatment of delayed SSIs. This may lead to progressive deformity, and patients must be counseled appropriately as to this possibility.

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