Review Article on Postoperative Spinal Implant Infection

Antibiotic treatment of postoperative spinal implant infections

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Abstract: Postoperative spinal implant infection (PSII) is a serious complication after spinal surgery. It is associated with increased morbidity and mortality for affected patients as well as significant costs for the healthcare system. Due to the formation of biofilm on foreign material, both diagnosis and treatment of PSII can pose a considerable challenge. Modern treatment protocols allow efficient eradication and good clinical outcomes in the majority of patients. In this article, we review the current antibiotic treatment concepts for PSII including the correct choice of antibiotics and their combination. In cases of late-onset PSII where the implants can be removed, two weeks of intravenous (IV) antibiotics followed by 4 weeks of oral antibiotics seem appropriate. If the implant needs to be retained, a 2-week IV antibiotic treatment should be followed by 10 weeks of oral antibiotic therapy with biofilm activity or, in case of problematic pathogens, a long-term suppression therapy. Initial empiric antibiotic therapy should cover staphylococci, streptococci, enterococci and Gram-negative bacilli as the most common pathogens. Antibiotic adjustments according to the type of pathogen and its antimicrobial susceptibility are essential for successful eradication of infection.

Keywords: Postoperative spinal implant infection (PSII); implant-associated infection; antibiotic treatment; spondylodesis

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Introduction

Orthopaedic implants are used in a variety of bone injuries and joint illnesses. In the spine, internal fixation devices have become a mainstay in the treatment of acute injuries such as fractures, as well as chronic degenerative changes such as osteochondrosis, or congenital deformities such as scoliosis. However, the incorporation of foreign materials into the human body is accompanied by the risk of infection. Implant-associated infections are among the most fearsome complications in the field of orthopaedic surgery. Despite ongoing advances regarding operation techniques and sterility levels, infection rates after spinal instrumentation are still reported up to 20% and are the most common reason for unplanned revision spine surgery within 30 days after index surgery (1-3). In revision spine surgery with removal of spinal implants postoperative spinal implant infection (PSII) has been reported to be found in up to 27% (4,5). One reason is related to the ability of bacteria to attach and form a biofilm on the surface of implants. The formation of such a biofilm dramatically reduces the bacteria's susceptibility to natural immune defence mechanisms as well as antibiotics. In addition, it also hinders the detection of bacteria, thereby creating a considerable medical challenge regarding both diagnostics and treatment. As a result, implant-associated infections may cause severe morbidity for affected patients as well as considerable costs for the health care system.

Table 1 Classification of postoperative spinal implant infections (source: PRO-IMPLANT Foundation, www.pro-implant-foundation.org)

Classification	Acute infection	Chronic infection
Pathogenesis		
Postinterventional	<6 weeks post-interventionally ("early-onset infection")	≥6 weeks post-interventionally ("late-onset infection")
Haematogenous or per continuitatem	<6 weeks symptom duration	≥6 weeks symptom duration
Clinical presentation	Acute pain, fever, prolonged wound secretion (>7–10 days), acute neurological deficits	Chronic pain, implant migration/loosening, fistula, neurological deficits
Typical pathogens	Highly virulent: *Staphylococcus aureus*, *Streptococcus* spp., Gram-negative bacteria	Low virulent: coagulase negative staphylococci, *Cutibacterium acnes*

Regarding the treatment of PSII, most cases require surgical revision (see article "Surgical Revision Strategies" in this issue). However, surgical interventions always have to be accompanied by an adequate antibiotic treatment protocol in order to achieve efficient pathogen eradication and good clinical results. Vertebral bodies are composed of highly vascularized bone, enabling easy penetration and diffusion to the infected site (6,7). In clinical practice choice of an adequate treatment strategy is challenged by the lack of published guidelines on the treatment of PSII and paucity of respective literature.

In this review, we aim to summarize the existing literature on antibiotic treatment of PSII and to provide guidance for treating doctors to make sensible decisions about antibiotic treatments for improved clinical outcomes.

Classification and pathogens

Implant-associated infections can be divided into different categories according to time of symptom onset (acute or chronic) and origin of the pathogens (postinterventional, haematogenous, per continuitatem). These categories are typically characterized by distinct clinical presentations and causative microorganisms, and require specific treatment protocols. Major characteristics of acute and chronic infections are summarized in *Table 1*.

Acute implant-associated infections, occurring within 6 weeks after surgery or with a symptom onset <6 weeks ago, are usually caused by highly virulent pathogens. Patients with acute infections often present with acute pain, fever, prolonged wound drainage (>7–10 days) or acute neurologic deficits. The majority of early-onset infections has been reported to be caused by *Staphylococcus aureus*, followed by streptococci, enterococci, and Gram-negative bacilli (6,8). Treatment of early-onset infections is complicated by the fact that the operated spinal segments have not yet had enough time for fusion to take place. Therefore, the implant is often still necessary to maintain stability of the spine and cannot be removed, which also influences antibiotic treatment choices.

In contrast, chronic infections typically cause symptoms such as chronic pain and implant loosening, but may as well lead to neurologic deficits. Typical clinical signs of infection such as fever or pus are usually missing. In many cases, the diagnosis is made only based on the microbiological and histopathological examination of intraoperatively collected tissue samples and removed implants. Chronic infections are mostly caused by low-virulent pathogens or can be culture-negative. In the past, there has been some debate whether microbial agents are the cause of late-onset spinal implant infection or if late-onset drainage is rather a result of aseptic inflammation from metal corrosion, with cultures positive for low-virulent organisms being of no pathogenic significance (8,9). This thesis was supported by some studies reporting >80% of late-onset "infections" to be culture negative (10). In contrast, other authors have described pathogen detection rates of >90% using extended culture incubation times (11,12). Thus, the formerly reported high number of culture negative samples may be due to insufficient sensitivity or incubation times. It has also been suggested that the environment created by postoperative sterile inflammatory processes may be favourable for the growth of low-virulence organisms (1,13). Typical pathogens of chronic infections include coagulase-negative staphylococci and *Cutibacterium* (formerly *Propionibacterium*) spp. Late-onset infections are primarily caused by organisms that are able to produce biofilm on the implant. The presence of biofilm hinders pathogen detection and can make eradication difficult without implant removal, similar to other bone and joint infections involving prosthesis (8,14,15). In many patients

Table 2 Empiric therapy

Clinical situation	First choice	Alternative
First revision	Ampicillin/sulbactam IV 3×3 g plus vancomycin IV 2×1 g	Cefuroxime IV 3×1.5 g plus daptomycin IV 1×500 mg
Multiple previous surgeries	Piperacillin/tazobactam IV 3×4.5 g plus vancomycin IV 2×1 g	Fosfomycin IV 3×5 g plus daptomycin IV 1×500 mg

Alternative to vancomycin, fosfomycin IV 3x5 g can be administered. IV, intravenous.

with late-onset infections the operated segments have already fused allowing for removal of the implants.

Pathogenesis

One of the main reasons for the highly increased risk of infection after spinal instrumentation lies in the formation of biofilms. A biofilm is a structured aggregation of bacteria encased in a self-produced matrix of extracellular polysaccharides that adheres to a surface (16). The surface of materials commonly used for spinal implants such as titanium, stainless steel, various polymeric biomaterials, and polymethylmethacrylate (PMMA) cement are all susceptible to colonization by biofilm-forming bacteria. There are three different ways in which an implant can get colonized: (I) within the perioperative period, e.g., via intraoperative inoculation; (II) haematogenously by pathogens from other infected foci, e.g., respiratory or urinary tract infection; and (III) per continuitatem, e.g., due to infected surrounding soft tissues. The development of a biofilm on orthopaedic implants can be divided into the four stages cell adhesion, cell aggregation, biofilm maturation, and cellular detachment (17). Once a biofilm has formed, bacteria display a highly increased resistance against both endogenous immune defense and antibiotics. Even though the responsible mechanisms are not yet fully understood, the existence of slow or non-growing cells within the biofilm is thought to play an important role (18).

Antibiotic treatment

An adequate management of PSII always involves a surgical intervention together with antibiotic treatment. For eradication of an implant-associated infection antibiotic treatment should be active against all causative pathogens in their biofilm form (19). In many cases, the exact pathogen(s) is/are unknown at the time of surgery. Therefore, empiric antibiotic regime is given initially that covers the most common and expected pathogens. Empiric treatment of PSII should cover staphylococci, streptococci, enterococci and Gram-negative bacilli. Polymicrobial infections are common in both, early-onset and late-onset PSII with rates of up to 50% (6,8,20,21). Antimicrobial resistant pathogens are rather uncommon, although their incidence is increasing (6,22,23). Antibiotic therapy should only be initiated after tissue samples for microbiological culture have been obtained (24).

Suggestions for empiric and targeted therapy according to the PRO-IMPLANT Foundation are presented in *Tables 2,3*. Antibiotic therapy is initiated by intravenous (IV) administration for the first 1–2 weeks to achieve sufficient tissue concentration in short time (6,25). When there is a clinical (no wound secretion) and laboratory response, which is usually after 1–2 weeks, treatment can be oral administration. The duration of the oral antibiotic treatment should be adapted depending on the causative pathogen and the clinical presentation of the patient.

In cases of late-onset PSII in which the implant can be removed without compromising the stability of the spine, treatment resembles therapeutic approaches of regular spondylodiscitis. Although there are no published guidelines, general consensus is to continue antibiotic treatment for around 6 weeks. Longer treatment durations may not bring additional benefit as similar outcomes have been reported (25-27). If the implants can be removed, oral biofilm-active antibiotics should be avoided in order to avoid resistancies.

In patients where implant removal is not feasible, antibiotic treatment is more demanding, especially because data on the optimal treatment duration is scarce (28,29). In the past, patients with PSII were usually administered antibiotic therapy for very long durations of more than 6 months, and in some cases up to 2 years on the basis of a few observational studies (6,8,29). However, current studies suggest good results for shorter antibiotic treatment with a total duration of 12 weeks (6,12). In some patients, particularly in those with problematic pathogens (i.e., pathogens resistant to biofilm-active antibiotics, e.g., rifampin-resistant staphylococci, ciprofloxacin-resistant Gram-negative bacteria and fungal infection), a long-term suppression therapy may be advisable until implant removal is possible. In general, if the implants are retained or exchanged an oral biofilm-active antibiotic treatment should only be started if an eradication is intended and not before the wound is dry. *Figure 1* depicts the different

Table 3 Targeted therapy (modified from and with permission of the Pocket Guide from PRO-IMPLANT Foundation)

Microorganism	Antibiotic (check susceptibility)	Dosage[b]	Administration
Staphylococcus spp.			
Oxacillin-/methicillin-susceptible	Flucloxacillin[a]	4×2 g#	IV
	+/– Fosfomycin	3×5 g#	IV
	For 2 weeks, followed by (depending on susceptibility):		
	Rifampin[c] +	2×450 mg	PO
	Levofloxacin or	2×500 mg#	PO
	Ciprofloxacin or	2×750 mg#	PO
	Cotrimoxazole or	3×960 mg#	PO
	Doxycycline or	2×100 mg	PO
	Fusidic acid	3×500 mg	PO
Oxacillin-/methicillin-resistant	Daptomycin or	1×8 mg/kg#	IV
	Vancomycin[d] or	2×1 g#	IV
	Teicoplanin	1×400 mg	IV
	+/– Fosfomycin	3×5 g#	IV
	For 2 weeks, followed by oral combinations of rifampin (see above)		
Rifampin-resistant*	Intravenous therapy for 2 weeks (as above), followed by long-term suppression for ≥1 year (e.g., doxycycline)		
Streptococcus spp.	Penicillin G or	4×5 million U#	IV
	Ceftriaxone	1×2 g#	IV
	For 2–3 weeks, followed by (where appropriate suppression for ≥1 year):		
	Amoxicillin or	3×1,000 mg#	PO
	Doxycycline	2×100 mg	PO
Enterococcus spp.			
Penicillin-susceptible	Ampicillin +	4×2 g#	IV
	Gentamicin[e]	1×240 mg#	IV
	+/– Fosfomycin	3×5 g#	IV
	For 2–3 weeks, followed by:		
	Amoxicillin	3×1,000 mg#	PO
Penicillin-resistant or allergy to penicillin*	Vancomycin[d] or	2×1 g#	IV
	Daptomycin	1×10 mg/kg#	IV
	+ Gentamicin[e] or	1×120 mg#	IV
	+ Fosfomycin	3×5 g#	IV
	For 2–4 weeks, followed by:		
	Linezolid (max. 4 weeks)	2×600 mg	PO
Vancomycin-resistant (VRE)*	Individual; implant removal or long-term suppression (e.g., with doxycycline, if susceptible)		

Table 3 *(continued)*

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Microorganism	Antibiotic (check susceptibility)	Dosage[b]	Administration
Gram-negative pathogens			
Enterobacteriaceae (*E. coli*, *Klebsiella*, *Enterobacter*, etc.)	Ciprofloxacin[f]	2×750 mg[#]	PO
Non-fermenting (*Pseudomonas aeruginosa*, *Acinetobacter spp.*)	Piperacillin/tazobactam or	3×4.5 g[#]	IV
	Meropenem or	3×1 g[#]	IV
	Ceftazidime	3×2 g[#]	IV
	+ (Tobramycin or	1×300 mg[#]	IV
	Gentamicin)	1×240 mg[#]	IV
	For 2–3 weeks, followed by:		
	Ciprofloxacin	2×750 mg[#]	PO
Multi-resistant	Depending on susceptibility: combination of meropenem 3×1 g[#], colistin 3×3 million U[#], fosfomycin 3×5 g IV[#], where appropriate oral suppression (if ciprofloxacin-resistant*)		
Anaerobes			
Gram-positive (e.g., *Cutibacterium*, *Peptostreptococcus*, *Finegoldia magna*)	Penicillin G[a] or	4×5 million U	IV
	Ceftriaxone	1×2 g	IV
	For 2–3 weeks, followed by:		
	Rifampin[c] +	2×450 mg	PO
	Levofloxacin or	2×500 mg[#]	PO
	Amoxicillin	3×1,000 mg[#]	PO
Gram-negative (e.g., *Bacteroides*)	Ampicillin/sulbactam[a]	3×3 g	IV
	For 2 weeks, followed by:		
	Metronidazole	3×500 mg	PO
Candida spp.			
Fluconazole-susceptible*	Caspofungin[g] or	1×70 mg	IV
	Anidulafungin	1×100 mg	IV
	For 2 weeks, followed by:		
	Fluconazole (suppression for ≥1 year)	1×400 mg[#]	PO
Fluconazole-resistant*	Individually (e.g., with voriconazole 2×200 mg PO); consider implant removal or long-term suppression		

Table 3 *(continued)*

Table 3 *(continued)*

Microorganism	Antibiotic (check susceptibility)	Dosage[b]	Administration
Culture-negative	Ampicillin/sulbactam[a]	3×3 g[#]	IV
	For 2 weeks, followed by:		
	Rifampin[c] +	2×450 mg	PO
	Levofloxacin	2×500 mg[#]	PO

Note: in some countries, IV fosfomycin is available only as 4 or 8 g; in this case, the preferred dosing is 2×8 g instead of 3×5 g. Where ampicillin/sulbactam is not available, amoxicillin/clavulanic acid 3×1.2 g IV or cefuroxime 3×2 g IV can be used. [a], allergy to penicillin NOT type 1 (e.g., exanthema): cefazolin (3×2 g IV). In case of anaphylaxia (= type 1 allergy with Quincke's oedema, anaphylactic shock) or allergy to cephalosporin: vancomycin (2×1 g IV) or daptomycin (1×8 mg/kg IV). Ampicillin/sulbactam is equivalent to amoxicillin/clavulanic acid (3×1.2 g or 3×2.2 g IV). [b], laboratory checks: 2×/week: leukocyte count, serum CRP, creatinine/eGFR, liver enzymes (AST and ALT). Adaptation of the dosage according to renal function and body weight (<40 or >100 kg). [c], rifampin: do not administer before implantation of the new implant. In addition to intravenous therapy as soon as wound is dry, dosage reduction to 2×300 mg if age >75 years. [d], determination of the vancomycin trough serum levels at least 1×/week, target: 15–20 µg/mL. [e], gentamicin: use only if high-level (HL) gentamicin has been tested susceptible. In case of HL-gentamicin resistant *e. faecalis* or renal insufficiency: replace gentamicin with ceftriaxone 2×2 g IV (only in case of *E. faecalis*) or fosfomycin 3×5 g IV. [f], additional intravenous therapy (piperacillin/tazobactam 3×4.5 g or ceftriaxone 1×2 g or meropenem 3×1 g IV) for the first postoperative days (until wound is dry). [g], for patients <80 kg: loading dose of 70 mg on first day, then dose reduction to 50 mg from day 2 on. *, problematic pathogens; [#], adaptation to renal function necessary. IV, intravenous; PO, per oral; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase.

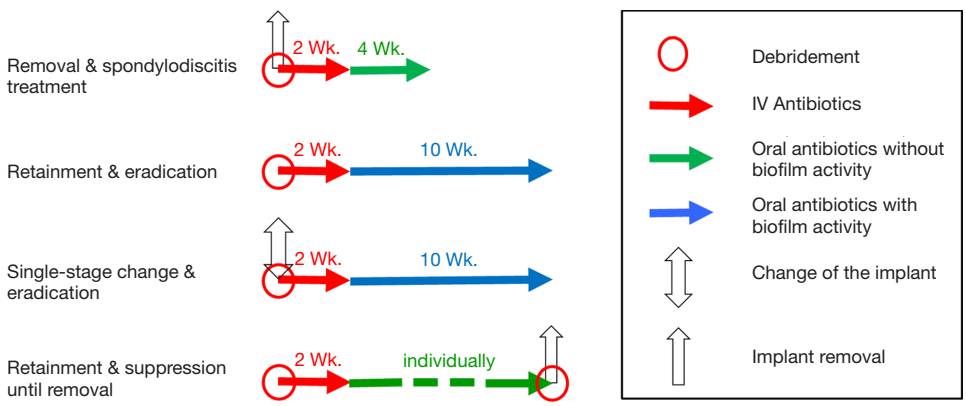

Figure 1 Treatment schemes for postoperative spinal implant infections (source: PRO-IMPLANT Foundation, www.pro-implant-foundation.org). IV, intravenous.

treatment strategies.

In cases of uncertainty it is recommended to consult an infectious disease doctor for co-management of the patient and to guide antibiotic therapy (24).

Conclusions

Adequate therapy of PSII is highly complex and challenging for physicians and patients alike. To reduce morbidity and mortality, continuous careful evaluation of treatment strategies is of uttermost importance. Data regarding optimal management are still scarce, but recent studies have shown good results for a total treatment duration of 12 weeks with IV followed by oral antibiotics for most patients. In patients with problematic pathogens, treatment duration has to be prolonged individually, possibly until

removal of the implant seems feasible.

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