

Orthopaedic Implant Infections: Current Management Strategies

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Abstract

Orthopaedic joint replacement and spinal stabilisation surgery has provided pain relief and functional improvement for millions. Postoperative infections involving the implanted hardware can have devastating functional, emotional and financial consequences. Implementation of relevant preoperative patient preparation, utilisation of appropriate prophylactic antimicrobials and intraoperative infection control measures have combined to diminish orthopaedic implant infection frequency significantly. Our adeptness at establishing the presence of an implant-related infection with certainty is not as well advanced.

Similarly, treatment guidelines, while useful, remain somewhat anecdotal. The pathogenesis, risk factors, diagnosis, management and prevention of hip arthroplasty infection and spinal stabilisation with instrumentation infection are reviewed.

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“When a lot of remedies are suggested for a disease, that means it can’t be cured.” Anton Chekov, The Cherry Orchard

Introduction

Advances in biomedical engineering and surgical technique have increased the indications and rate at which orthopaedic implantation procedures are performed. Joint replacement and skeletal stabilisation provide dramatic symptom relief as well as rapid functional restoration to millions. However, medical progress comes at a price. With any type of foreign body implantation comes the not-insignificant risk of infection. As all orthopaedic surgeons will attest, infection associated with an implant can be catastrophic. Implant infection occurs in 1% to more than 8% of all cases (Table I). Orthopaedic implant infections are associated with significant morbidity (and occasionally death), at least temporary loss of function, skeletal defects, additional hospitalisation and significant financial cost. For example, management of an infected total hip arthroplasty increases the cost of that arthroplasty by 400% to 600%.²

The ensuing review addresses the pathogenesis, risk, diagnosis, management and prevention of infections associated with orthopaedic prostheses and implants. Emphasis will be placed on total hip arthroplasty infections and infections associated with spinal surgery involving instrumentation and fusion.

Pathogenesis

Infection occurs when the bacterial load exceeds that which the body is able to clear, local host defence is impaired, tissue trauma has occurred and / or a foreign

body is present. Many of these conditions exist when orthopaedic implants (and cement) are introduced. To compound the situation, prosthesis biomaterial and polymethylmethacrylate cement lower the bacterial inoculum threshold needed to establish an infection.³ Infection most commonly arises from bacteria introduced at the time of surgery. Less than fifty per cent of infections arise from haematogenous spread (from dental, gastrointestinal tract, genitourinary system, and integument sources> after implantation.

Once bacteria are present, the events which lead to implantation infection are bacterial adherence to the prosthesis, bacterial avoidance of host defense, bacterial

TABLE I: ORTHOPAEDIC INFECTION RATES

Event or procedure	Infection rate
Replacement arthroplasty	
Hip	<2%
Knee	2.9%
Elbow	5.3%'
Fracture	
Overall	3%
Open trauma	2.5% to 12.0%
Simple intervertebral disectomy	<1%
Spinal fusions	
Non-instrumented	1% to 4%
Instrumented	4% to 9.7%

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multiplication and, finally, bacterial invasion of local tissue and/or bone. Additional factors which may participate in infection establishment include production of extracellular slime as an adjunct to avoid host defenses and haematogenous bacterial dissemination with its associated morbidity.

Bacterial Adherence

Upon implant insertion, host protein coats the biomaterial surface. The variety and quantity of host protein present is a function of the biomaterial involved. Bacterial adhesion appears to be significantly dependent on the layer of host protein and the unique physico-chemical surface properties of prosthesis biomaterial. Bacterial adhesion to the protein/bioprostheses complex probably occurs through several mechanisms including ligand-receptor interaction, hydrophobicity, electrostatic interactions and van der Waals forces.

Bacterial Slime and Host Defence Avoidance

Many bacteria produce bacterial slime. This extracellular product is also known as glycocalyx. *Staphylococcus epidermidis*' glycocalyx has been studied most extensively. The presence of *S. epidermidis* slime likely explains why this otherwise low virulence organism is so frequently involved in foreign body-associated infections (e.g. orthopaedic prostheses, pacemakers and vascular catheters). Bacteria imbedded in glycocalyx evade leukocytes by chemically and mechanically interfering with opsonisation and phagocytosis. Bacteria may avoid eradication via glycocalyx preventing adequate antibiotic levels reaching bacteria imbedded in slime. Additionally, even when glycocalyx is not present, adherence to prosthetic biomaterials may phenotypically alter bacteria rendering them less intrinsically susceptible to specific antibiotics.⁴ Thus, eradication of established infections of implanted devices (particularly when glycocalyx is present) is unlikely to occur without implant removal.⁵

Risk of Orthopaedic Implant Infection

Tables II and III list the factors associated with increased risk of postoperative hip arthroplasty and spinal surgery, respectively.

Diagnosis

Hip Arthroplasty

The onset of new pain in a prosthetic joint must add infection to the considered differential diagnosis. The pain may have an abrupt or indolent onset; pain may be present with or without limb movement. Pyrexia, local warmth and erythema are often not present. In fact, the history and physical examinations are rarely helpful in distinguishing pain due to sepsis versus aseptic prosthesis loosening. Despite the general limitations of the

TABLE II: INFECTION RISK: HIP ARTHROPLASTY

Prior surgery at site of implantation
Existing infection at time of implantation
Rheumatoid arthritis
Corticosteroid therapy
Diabetes mellitus
Poor nutritional status
Advanced age
Use of polymethylmethacrylate cement ⁴
? Presence of drains ⁶

TABLE III: INFECTION RISK: SPINAL SURGERY⁷

Preoperative
Prolonged preoperative hospitalisation
Obesity
Tobacco use
Poor nutritional status
Age if associated with chronic medical conditions
Immunosuppression
Diabetes mellitus
Infection at other sites
Previous radiation at surgical site
Operative
Surgeon experience
Duration of operation
Complexity of surgical procedure
Use of operating microscope
Failure to give pre-incision antibiotic
Instrumentation
Transpedicular fixation
Operating theatre traffic
Transfusion requirement
Presence of wound seroma
Use of morphine paste (D M Allen, unpublished data)

physical examination, the presence of a discharging sinus is diagnostic of an infected prosthesis.

An increasing erythrocyte sedimentation rate (ESR) is a sensitive but not specific measure of an infected prosthesis in the appropriate setting. Some suggest an elevated C-reactive protein may be more specific.⁸

Periosteal new bone formation on the anteroposterior hip X-ray is diagnostic of a septic process but is seen in a minority of patients with infected hip arthroplasties.⁹ Indium¹¹¹ white blood cell imaging is accurate for a septic process 80% to 94% of the time. Indium¹¹¹ polyclonal IgG imaging was found to be less specific for infection by Oyen et al;¹⁰ however, the source of the labelled immunoglobulin may affect results. Additional prospective studies utilising Indium¹¹¹ polyclonal IgG imaging are underway. Technetium-99m monoclonal antibody imaging investigations appear promising as well.

The detection of bacterial DNA in hip aspirates and biopsies by polymerase chain reaction technology is

being explored but is not currently pragmatic for routine clinical use.

The accepted means to diagnose a septic total hip arthroplasty remains joint aspiration with culture (sensitivity 87%, specificity 95% and accuracy 94%)¹¹ with the gold standard being hip biopsy with frozen section,¹² histology and culture.

Spinal Surgery¹³

Postoperative infections associated with vertebral fusions and vertebral instrumentation may be superficial to the fascia, below the fascia or both. Deep infections (below the fascia) can include diskitis, epidural abscesses, meningitis, vertebral osteomyelitis and psoas abscesses. Back pain, fever, chills, sweats, and wound tenderness are symptoms associated with superficial or deep wound infection following spine surgery.

While headache is concerning for a parameningeal or meningeal focus, it may be present with either superficial or deep infections.

Examination is also limited with regard to extent of infection depth. Superficial infections often appear red and boggy, while deep infections may be deceptively benign. New or progressive neurologic deficits suggest a deep process which requires emergent intervention.

Leukocytosis is not a consistent finding regardless of depth; however, assessment of the erythrocyte sedimentation rate is somewhat more reliable. Plain X-rays are useful if symptoms have been present for greater than 4 weeks by which time disk space narrowing, erosion of subchondral end plates, and eventual obliteration of the disk space has occurred. Osteosclerosis will be present, after 8 to 12 weeks of infection. Radionuclide studies are less useful particularly in the early postoperative period due to non-specificity. Magnetic resonance imaging (MRI) scans are not helpful in the presence of hardware due to hardware-induced signal artifact unless diskitis or an epidural abscess is present.

In summary, clues from the history and laboratory can support the diagnosis of a postoperative spine wound infection. However, the depth of an early infection with hardware in place is not confirmed without careful wound exploration. Long standing deep infections may be diagnosed by characteristic plain film spinal X-rays.

Treatment

Hip Arthroplasty

Infected haematomas are managed with debridement and parenteral antibiotics. For infected prostheses, curative therapeutic options are limited. Prosthesis extirpation without reconstruction, extirpation with immediate implantation of a new prosthesis (one-stage procedure) and extirpation with delayed prosthesis replacement (two-stage procedure) are the recognised

definitive choices. One-stage procedures utilising antibiotic-impregnated cement and beads have a 77% to 94% success rate in highly selected populations. Success is a function of debridement and the infecting organism's virulence. *Staphylococcus aureus*, *Enterococcus* species, gram-negative bacilli, fungi and glycocalyx-producing coagulase negative *Staphylococcus* are considered virulent organisms. The two-stage procedure with 4 to 6 weeks of antibiotics followed by delayed re-implantation remains the standard-of-care as it has for two decades. Re-implantation should be delayed for several months for those with infections involving virulent organisms. The two-stage procedure delivers a reproducible 85% to 93% rate of success. Debridement with retention of the prosthesis and parenteral antibiotic therapy is a less desirable approach particularly when addressing late infections and those developing from haematogenous spread. The overall success rate with this approach is 15% to 60%.¹⁴

Antibiotic therapy without surgery (suppressive therapy) has a >50% failure rate in managing infected hip prostheses. Suppressive therapy has been suggested for those who are not surgical candidates due to infirmity. This technique is associated with progression of osteomyelitis into the femur and hemipelvis.^{15,16} However, recent studies utilising ofloxacin and rifampin as the suppressive antibiotic regimen have been more successful (70%) suppressing both chronically infected prosthetic hips and knees.¹⁷

Spinal Instrumentation

Treatment should be prompt, aggressive and surgical. Radical debridement with layer-by-layer desloughing is required. Meticulous desloughing with copious irrigation of superficial layers is essential prior to exploring tissue deep to the fascia. At each tissue layer, cultures should be taken and the level from which they were collected recorded. Removal of devitalised tissue and non-essential material (Gelfoam®) is imperative. Bone graft loosened during lavage and desloughing should be removed, otherwise fixed graft can remain. Implants may remain in place to promote union. Implants should be removed if infection cannot be controlled with the above measures. Wounds may be closed over suction drains or superficial wounds may be allowed to heal by secondary intention. Repeat debridement may be required. Symptom improvement, bone fusion by computed tomography or plain films and a declining ESR are the criteria of effective therapy. Once bone fusion is documented a further decision point is reached with regard to the removal of the implant. In selected circumstances, antimicrobials can be held and the ESR, clinical status and X-rays followed expectantly. Alternatively, the hardware can be removed and an additional course of antibiotic therapy is given. Excellent algorithms out-

lining a prudent approach to postoperative superficial and deep wound infections following spinal fusion with hardware in place is found in Abbey et al.¹⁸ Antibiotic duration may be from 10 days to several months and is dependant upon the infection depth, structures involved and presence of an implant. It is notable that there are conflicting opinions regarding the duration of antibiotic therapy and the role of hardware removal within the orthopaedic community itself and from infectious diseases consultants."

Prevention

General principles to prevent implant-related infections begin with optimising the patient's presurgical status. Nutritional repletion, limiting presurgical hospital stay, discontinuance of tobacco use, control of diabetes mellitus, eradication of existing genitourinary or other infections and correcting hypoxia are all worthy efforts prior to implantation. Additional presurgical measures include bathing with providine-iodine the evening prior to surgery, using clippers or depilatories in lieu of a razor to trim surgical site body hair and infusing prophylactic antibiotic in the one-half hour to one hour prior to the initial surgical incision. The prophylactic antibiotic selected should be one which addresses the common bacterial isolates associated with implant-related infections in your facility. Cefazolin is generally accepted as the first drug to consider.

Intraoperative measures to minimise infection involve limiting theatre traffic, double-gloving the surgeons, proper surgical site skin preparation, utilising a theatre with laminar air flow and 300 air exchanges per hour, meticulous surgical technique and wound irrigation (antibiotic addition to the irrigant is not advocated). Should the procedure be prolonged, additional intraoperative prophylactic antibiotic dosing is in order. With the introduction of ultra-clean rooms and prophylactic antibiotics, infection rates for hip implantation is now 0.5% to 0.8%.¹⁹ The role of prophylactic antibiotics incorporated into the cement is less clear.²⁰ The duration for antibiotics postoperatively in orthopaedic implant surgery has not been studied prospectively. A duration of 48 hours following surgery is generally suggested with others recommending antibiotic continuance until drain removal. From a more esoteric perspective, efforts to develop biomaterials which resist bacterial adhesion are ongoing.

Conclusion

The frontiers of orthopaedic surgery have expanded rapidly in the last two decades. While biomedical and surgical advances have been dramatic, postoperative infection remains a significant obstacle. Insight into infection predisposition and efforts to decrease infection rates have progressed nicely. Unfortunately, the data

regarding therapy, particularly with regard to infected instrumented spinal procedures, remain somewhat anecdotal as there are few prospective studies to guide us. Despite these limitations, the practical application of the principles of infection management provides appropriate guidance. The future holds the promise of improved biomaterials, a greater understanding of the impact of biomaterials on host defence and standardised therapeutic protocols.

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