ONE STAGE VERSUS TWO STAGES REVISION IN INFECTED TOTAL HIP ARTHROPLASTY

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By

Mohamed Khalaf Abdullah Mohamed
M.B., B.Ch

Supervised By

Prof. Dr. Ashraf Abdel Kader El-Nahal
Professor of Orthopedic Surgery
Faculty of Medicine
Cairo University

Dr. Omar Ahmed Soliman
Lecturer of Orthopedic Surgery
Faculty of Medicine
Cairo University

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ABSTRACT

Treatment options of infected THA is variable including suppressive antibiotic therapy for those patients not fit for operation, debridement for acute superficial infection, and exchange arthroplasty. Exchange arthroplasty can be done in one stage operation accompanied by meticulous debridement. Although this operation is simpler and less coasty, it is associated with higher rates of infection.

One stage arthroplasty indicated only if infection caused by known and sensitive organism to antibiotics, healthy host with few or none of the risk factors for infection such as rheumatoid arthritis, diabetes, chronic skin lesions and obesity and a wound in which there is adequate bone and soft tissue to support reconstruction of the hip. Two stages revision arthroplasty done in two stages the first is similar to the one stage change arthroplasty, the second stage arthroplasty can be done either cemented or cementless and both ways have better results when compared with one stage arthroplasty.

The current standard of care for late chronic infection is the two stages revision arthroplasty including removal of the prosthesis and cement, thorough debridement, placement of an antibiotic-impregnated cement spacer, a course of intravenous antibiotics, and a delayed second-stage revision arthroplasty.

Keywords: One Stage, Two stages, Staged revision, Infected THA

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LIST OF ABBREVIATIONS

Abbreviation	Meaning
^{111}In	Indium-111
ALAC	Antibiotic-Loaded Acrylic Cements
AP	Antero-Posterior
Clin Orthop	Clinical orthopedics and related research
CRP	C-Reactive Protein
CT	Computed Tomography
DNA	DeoxyriboNucleic Acid
ESR	Erythrocyte Sedimentation Rate
FDG	FIuoro-2Deoxy-2-D Glucose
Fig/s	Figure/s
IgG	Immunoglobulin G
IL-6	Interleukin-6
Instr Course lect	Instructional Course Lectures
J Arthroplasty	Journal of Arthroplasty
J Bone Joint Surg Am	Journal of Bone and Joint Surgery American edition
J Bone Joint Surg Br	Journal of Bone and Joint Surgery British edition
JAAOS	Journal of American academy of Orthopedic Surgeons
JAAPA	Journal of The American Academy of Physician Assistants
MRI	Magnetic Resonance Imaging
PCR	Polymerase Chain Reaction
PET	Positron Emission Tomography
PMMA	PolyMethylMethAcrylate
PROSTALAC	Prosthesis Of Antibiotic Loaded Acrylic Cement
rRNA	Ribosomal RiboNucleic Acid
S aureus	Staphylococcus aureus
S epidermidis	Staphylococcus epidermidis
Tc99m	Technetium 99 Meta-staple
THA	Total Hip Arthroplasty
WBCs	White Blood Cells

INTRODUCTION

One of the most dreaded complications of total hip arthroplasty is infection. Although the prevalence of infected total hip replacements is only one percent, the economic burden, as well as the likelihood of significant morbidity, and even mortality, make this complication potentially devastating. Fortunately, a number of diagnostic techniques are available to aid in determining the presence or absence of infection. Once an infection is confirmed, several treatment options are available to the treating surgeon. Knowledge of the proper indications for each technique ensures appropriate treatment and optimizes results. [(1)]

The pathogenesis of prosthetic joint infection is related to microorganisms growing in biofilms, rendering these infections difficult to diagnose and to eradicate. Low-grade infections in particular are difficult to distinguish from aseptic failure, often presenting only with early loosening and persisting pain, or no clinical signs of infection at all. A combination of preoperative and intraoperative tests is usually needed for an accurate diagnosis of infection of prosthetic joint infections. Successful treatment requires adequate surgical procedure combined with long-term antimicrobial therapy, ideally with an agent acting on adhering stationary phase microorganisms. [(2)]

Infection at the site of a total joint arthroplasty can be classified into four basic categories: Type I (early postoperative), Type II (late chronic), Type III (acute hematogenous), and Type IV (positive intraoperative cultures with clinically unapparent infection). [(3)]

The primary goals of the treatment of periprosthetic hip infections are the eradication of the offending pathogen and the restoration of function.

The treatment method of choice varies from case to case and depends on several variables including the following: [(1)]

I. The acuteness of the infection, virulence of the offending pathogen.
II. The quality of bone and surrounding soft tissues.
III. The stability of the implant.

IV. The patient's medical condition and willingness to undergo additional procedures.

The treatment of infected total hip arthroplasties consists of one or more of the following: [4]

I. Antibiotic therapy.

II. Incision and drainage of the hip.

III. Debridement and modified Girdlestone resection arthroplasty.

IV. One or two-stage revision to a total hip arthroplasty.

Aim of The Study

To illustrate the recent trends in diagnosis and treatment of infected total hip replacement emphasizing on comparing of the indications and results of one stage and two-stage protocols in surgical treatment of infected total hip replacement.

EPIDEMIOLOGY

The use of perioperative antimicrobial prophylaxis has substantially decreased the frequency of implant associated infections. In patients with primary joint replacement, the infection rate in the first two years is usually <1% in hip and shoulder prostheses, <2% in knee prostheses, and <9% in elbow prostheses. [(2)]

The reported infection rates are probably underestimated, since many cases of presumed aseptic failure may be due to unrecognized infection. In addition, infection rates after surgical revision are usually considerably higher (up to 40%) than after primary replacement. [(6)]

Importantly, prosthetic joints remain susceptible to haematogenous seeding during their entire lifetime and some perioperative infections may have a latency period longer than two years. Therefore, for accurate comparisons the frequency of infection should be reported as incidence rate (per prosthesis-years) rather than as risk (without specified denominator). In a study involving hip and knee prostheses, the incidence of infection was 5.9 per 1000 prosthesis-years during the first 2 years after implantation and 2.3 per 1000 prosthesis-years during the following 8 years. In the future, it is expected that the incidence of revision of prosthetic joint infections will further increase due to:

I. Better detection methods for microbial biofilms involved in prosthetic joint infections.
II. The growing number of implanted prostheses in the ageing population.
III. The increasing residency time of prostheses, which are at continuous risk for infection during their implanted lifetime. [(6)]

Infecting organisms

A variety of different bacterial species can cause deep periprosthetic infection Staphylococcus aureus and Staphylococcus epidermidis are the most common organisms, although a wide range of gram-positive and gram-negative organisms as well as anaerobic organisms have also been identified. Table (1) shows the most common organisms identified and incidence for each species. [(2)]

The timing of infection varies by species: S aureus predominate in early infections, and bacteria from normal skin flora such as S epidermidis, propionbacterium acnes and peptostreptococci present later in delayed infections. There have also been occasional reports of infection caused by rare organisms and fungus such as Candida albicans, and Actinomyces israelii. [(2)]

Microorganism	Frequency (%)
Coagulase-negative staphylococci	30-43
Staphylococcus aureus	12-23
Streptococci	9-10
Enterococci	3-7
Gram-negative bacilli	3-6
Anaerobes	2-4
Polymicrobial	10-12
Unknown	10-11

TABLE (1)

Commonly identified microorganisms causing prosthetic joint infection. [(2)]

PATHOGENESIS

Routes of Infection

There are four routes by which infecting organisms can reach periprostic space:

I) Contamination at the time of surgery:

It is a well-recognized factor. Lidwell et al, showed progressive decrease in the incidence of joint infection with reduction of air contamination. [7]

Salvati et al, investigated effect of ultraclean air laminar flow and found a statistically significant decrease in infection rates for hip replacement from 1.4% to 0.9%. [8]

II) Direct or contiguous spread:

It's more common in joints close to surface as knee and elbow than in hip arthroplasty. The organism may migrate to the hip from superficial infection.

Schmalzried et al, reported on 47 deep infections in a series of 3,051 THAs. Two of these patients had infection from direct or contiguous spread. One of them had urethral strictures that were complicated by a deep perineal abscess that drained into the hip. [9]

Surin et al, reviewed 34 deep infections in a consecutive series of 803 hip replacements. They found a 3.2-fold increase risk of deep infection (11 hips) in the 115 hips with postoperative wound drainage after a minimum follow up of 3 years.[10] However, this finding was not supported by the work of Gaine et al, who reported on 301 THAs at a mean follow up of 26 months. There was no increase in deep wound infection in the subgroup of 56 patients with a superficial infection, compared with those who had no wound complication. Four of the 301 arthroplasties (1.3%) resulted in early deep infections; the infection rate subsequently decreased with installation of a laminar airflow system. [11]

III) Haematogenous spread

Haematogenous seeding of bacteria that originated in a remote infection in the periprosthetic tissues. In the study of David and Vrahas on 67 infected THA, the most common source of infection was skin infections in 31 of 67 (46%), followed by dental infection or dental manipulations in 10 of 67 (15%) and also reported with urinary tact infections in 9 of 67 (13%).[12]

IV) Reactivation of infection in a previously infected hip

The fourth mode of infection is reactivation of infection in a previously infected hip. This was the mechanism of infection in 13 of 47 infected hips previously mentioned in the study of Schmalzried et al. [9]

Role of Microbial Biofilms

Implant-associated infections are typically caused by microorganisms growing in structures known as biofilms (figs. 1, 2). [13]

These microorganisms live clustered together in a highly hydrated extracellular matrix called slime attached to a surface. Depletion of metabolic substances or waste product accumulation in biofilms causes microbes to enter a slow or non-growing (stationary) state. Therefore, biofilm microorganisms are up to 1,000 times more resistant to growth-dependent antimicrobial agents than their free-living (planktonic) counterparts. [14]

Biofilms contain interstitial voids (water channels) in which nutrients can circulate between microbial cells. Within biofilms, bacterial cells develop into organized and complex communities with structural and functional heterogeneity resembling multicellular organisms in which water channels serve as a rudimentary circulatory system. [15]

Release of cell-to-cell signaling molecules (quorum sensing) induces bacteria in a population to respond in concert by changing patterns of gene expression involved in biofilm differentiation. [16]

Programmed cell death of damaged cells may play an important role in bacterial biofilms, similar to multicellular organisms. [17]

In summary, existence within a biofilm represents a basic survival mechanism by which microbes resist against external and internal environmental factors, such as antimicrobial agents and the host immune system. [18]

Role of Foreign Body

The pathogenesis of implant-associated infection involves interaction between the microorganisms, the implant and the host. [19]

Adherence of S epidermidis to the surface of the device involves rapid attachment to the surface of the implant mediated by nonspecific factors (such as surface tension, hydrophobia, and electrostatic forces), or by specific adhesions factors (specific protein called fibronectin-binding protein). This initial phase of adherence is followed by an accumulative phase during which S epidermidis bacterial cells adhere to each other and form a biofilms. [20]

Adherence of S aureus is more dependent on the presence of host-tissue ligands, such as fibronectin, fibrinogen, and collagen. The presence of a foreign body decreases the minimal infecting dose of S aureus more than 100,000-fold; this increased susceptibility is at least partially due to a locally acquired granulocyte defect induced by frustrated phagocytosis. [21]