UPDATE ON INFECTIONS IN ARTICULAR PROSTHESIS

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ABSTRACT

The implantation of joint prostheses, especially for the hip and knee, is becoming increasingly common. This provides a significant reduction in discomfort and an immeasurable improvement in patient mobility. Reviews of the worldwide literature indicate that 1 to 5% of these prostheses become infected, although it is important to remember that as the number of operations performed to implant these prosthesis increases, so will the number of cases of this type of infection. Gram-positive bacteria predominate in contaminations of joint prostheses, in particular Staphylococcus aureus and Staphylococcus epidermidis. Infections caused by gram-negative bacilli and fungi such as Candida sp have been reported with increased frequency throughout the world. Infections of joint prostheses present characteristic signs that can be divided into acute manifestations (severe pain, high fever, toxemia, heat, redness and wound secretions) and chronic manifestations (progressive pain, cutaneous fistula formation and pus drainage, without fever). The definitive diagnosis of the infection should be made through cultures to isolate the microorganism, using material collected from joint fluid puncture, surgical wound secretions, and surgical debridement. It is essential to cover for methicillin-resistant Staphylococcus aureus, given the epidemiological importance of this agent in these infections. The total duration of antibiotic therapy ranges from six weeks to six months, and this treatment should be adjusted as needed, based on the results from culturing.

Keywords – Joint Prosthesis; Infection/diagnosis; Infection/therapy

INTRODUCTION

The implantation of joint prostheses, especially for the hip and knee, is becoming increasingly common. This provides a significant reduction in discomfort and an immeasurable improvement in patient mobility[1,2]. It has been estimated that, including both primary and revision surgery, around 800,000 operations to implant hip and knee prostheses are performed every year, in the USA alone[3] (Figure 1). Furthermore, although in small numbers, implantations of joint prostheses for the shoulder, elbow, wrist and temporomandibular joint are also becoming more common[2]. Reviews of the worldwide literature indicate that 1 to 5% of these prostheses become infected, although it is important to remember that as the number of operations performed to implant these prosthesis increases, so will the number of cases of this type of infection[4] (Figure 2). Even though infection occurs less frequently than mechanical loss of the prosthesis, it is considered to be the most devastating of

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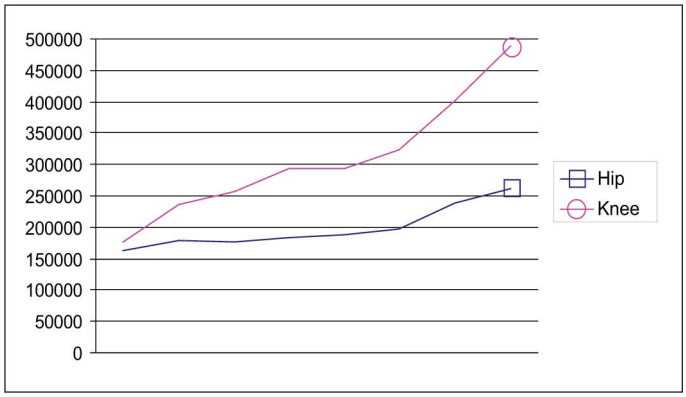
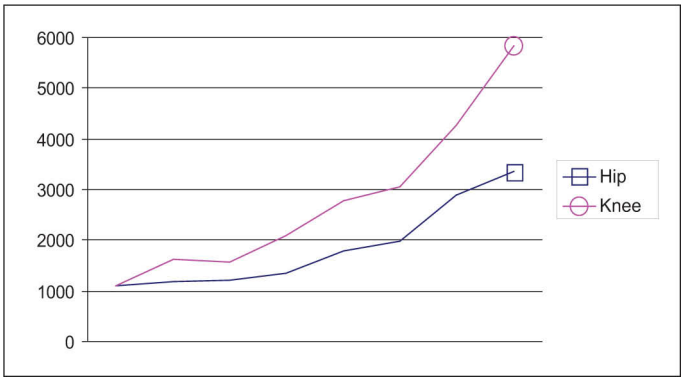


Adapted from: Kurtz *et al*[3].

Figure 1 – Evolution of the number of knee and hip prostheses implanted in the USA between 1990 and 2004.



Adapted from: Kurtz *et al*[3].

Figure 2 – Evolution of the number of infections in prostheses diagnosed in the USA between 1990 and 2004.

the complications, leading to prolonged hospitalization, repeated surgical interventions and even definitive loss of the implant, with shortening of the affected limb and significant permanent deformities[1,2].

Risk factors and physiopathogenesis

The main risk factors predisposing towards infections in joint prostheses that are cited in the literature are: advanced age, malnutrition, obesity, diabetes mellitus, HIV infection at an advanced stage, presence of a distant focus of infection and a history of arthroscopy or infection in a previous arthroplasty. Patients with rheumatoid or psoriatic arthritis are also at greater risk of postoperative infection, which has been estimated to be three to eight times greater than for other patients. Prolonged duration of surgery (more than 150 minutes), blood transfusion and carrying out bilateral arthroplasty in a single operation are other factors relating to greater occurrence of infection. Any factor that delays the healing of the surgical wound, such as ischemic necrosis, hematoma, cellulitis and/or wound abscess, increases the risk of infection, since the deep tissues contiguous with the prosthesis do not have any local defense barriers[1,2,4]. It is important to emphasize that the presence of the joint prosthesis leads to impairment of the function of the local granulocytes that accumulate around the implant: these become partially degranulated, with diminution of the production of dismutase superoxide and damage to the defense capacity against bacteria, particularly against Staphylococcus aureus. Thus, the presence of the implant diminishes the size of the bacterial inoculum needed for infection to take place, by more than 100,000-fold[5].

Joint prostheses can become infected through three different routes: direct implantation, hematogenesis and reactivation of latent infection[2].

Penetration of microorganisms into the wound during surgery can occur through either endogenous or exogenous sources. Examples of such sources include the patient's skin microbiota, the surgical team's limbs, the environment or even contaminated implants.

Bacteremia from distant foci may cause contamination of the prosthesis through a hematogenic route. The primary foci most frequently reported in the worldwide literature are the respiratory tract, skin, urinary tract, dentition and gastrointestinal tract[2,5].

Gram-positive bacteria predominate in contaminations of joint prostheses, especially Staphylococcus aureus and Staphylococcus epidermidis. However, infections caused by Gram-negative bacilli and fungi such as Candida sp are being reported with greater frequency all around the world[5].

Clinical presentations and diagnosis

Infections of joint prostheses present characteristic signs that can be divided into acute manifestations such as intense pain, high fever, toxemia, heat, redness and operative wound secretions, and chronic manifestations, namely progressive pain and formation of skin fistulas with drainage of purulent secretions, which in most cases are without fever. The clinical presentation depends on the virulence of the etiological agent involved, the nature of the infected tissue and the infection acquisition route. Several classifications have been put forward to define the moment at which the contamination occurred and, through this, to establish the likely etiological agent involved and the best therapeutic strategy[1,2,5].

Nonspecific laboratory tests such as leukogram, erythrocyte sedimentation rate, alpha-1-acid glycoprotein and