Orthopaedics & Traumatology: Surgery & Research 102 (2016) S113–S119

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Review article

Patient-specific risk factors for infection in arthroplasty procedure

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ARTICLE INFO

Article history:
Received 14 January 2015
Accepted 25 May 2015

Keywords:
Infection
Arthroplasty
Risk factors
Prosthetic joint infection
Orthopedic surgery

ABSTRACT

All patients are not equally at risk when it comes to postoperative infections, whether the risks are related to the environment or the patient. Patient-specific infection risk factors for arthroplasty should be a focal point during the preoperative consultation as they impact the treatment decision. Eighty percent of patients have at least one modifiable infection risk factor. These risk factors must be corrected preoperatively whenever possible so that the patient is operated under the best possible conditions, with the lowest possible infection risk. The screenings and preoperative preparations are multidisciplinary but must also involve the patient. The information provided to the patient must match the patient's infectious risk profile. This lecture will review every infection risk factor, whether it is modifiable or not, and then suggest how the treatment decision should be adapted to each patient's infection risk.

1. Introduction

Periprosthetic joint infection (PJI) is the leading cause of failure in total knee arthroplasty (TKA) and the third most common cause of failure in total hip arthroplasty (THA) [1]. It extends hospitalization by 12–20 days, doubles the re-hospitalization rate, requires one or more reoperations and increases the cost of care by more than 300% [2,3]. This dreaded and devastating complication generally results in poor functional recovery and mediocre quality of life — outcomes that are far removed from the ones expected.

In France, 135,365 hip prostheses and 85,569 knee prostheses were implanted in 2012; it is estimated that 2000–2500 cases of PJI occur each year. The number of knee and hip arthroplasty procedures annually in the USA is projected to reach 4 million by 2030, which represents an increase of 673% for the knee and 174% for the hip in a 25-year span. In parallel, the number of revision procedures for infected prostheses could reach 35,000 cases per year [4].

Given the severity of PJIs, many studies have been performed to identify risk factors for infection. There are a tremendous number of risk factors; they can involve the patient (intrinsic factor) or the environment (extrinsic factor) and play a role in the pre-, intra- or postoperative period.

The goal of this lecture is to define the patient-specific risk factors for PJI and separate them into factors that can and cannot be modified. The end goal is to help the surgeon know how to screen for these risk factors and correct them preoperatively if at all possible, or abandon the surgery plans if the infection risk appears greater than the expected benefit from the surgery. In every case, the information provided to the patient must match the patient's infectious risk profile.

Although the impact of certain risk factors has been well documented in published studies and is the subject of recommendations, there are doubts surrounding many other factors because PJIs are relatively rare and few relevant prospective studies have been carried out. Given the large number of published studies and contradictory conclusions in some cases, we will focus on French and international recommendations; if these do not exist, we will use the conclusions of meta-analyses, literature reviews, joint registry reports and high-quality studies.

2. Role of the host: from contamination to infection

Infection is a clinical condition resulting from the host's reaction to the presence of pathogens. Although infection requires contamination by a micro-organism, the presence of bacteria does not by itself explain infection because contamination can have no clinical consequences, as observed in certain sites that are physiologically contaminated (e.g., skin, mucous). For a pathological process to be triggered, the balance between the host's defenses and the bacteria's pathological power must be altered. The progression from contamination to infection is a mutlifactorial event, governed by Altemeier's equation [5]:

$$\text{Infection} = \text{Degree of contamination} \times \text{Virulence of germs/host resistance}$$

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http://dx.doi.org/10.1016/j.otsr.2015.05.012
1877-0568/

The elements of this equation must be altered to reduce the infection rate:

- the battle against contamination is based on aseptic measures, skin preparation and control of air-borne contamination [6]. When standard preventative methods are used, this contamination is probably minor. As a consequence, it is nearly impossible to establish a link between a new preventative measure and the infection rate; the impact of this measure is now being evaluated by the presence of germs (contamination), not by the infection rate;
- host resistance, which is a difficult concept to quantify. It would require that the clinical infection rate be calculated after inoculating subjects with a given bacterium, which can only be done in experimental studies. Nevertheless, some host-related risk factors are known to increase the infection risk. We must compel ourselves to look for them and correct them as much as possible, in hopes of reinforcing the patient's defense capacities.

3. Modifiable risk factors

It is standard practice to take the patient's history, perform a clinical examination and then request additional preoperative tests to look for any comorbidities or on-going treatments that could increase the infection risk.

Eighty percent of patients who are candidates for an arthroplasty procedure have modifiable risk factors. The most common are obesity (46%), anemia (29%), malnutrition (26%) and diabetes (20%) [7]. The modifiable nature of these conditions makes it possible to more or less control them, reducing the risk of postoperative infection as much as possible. We can differentiate between risk factors that reduce the patient's immunity and those that increase the bacterial load.

3.1. Obesity

Obesity is defined as a body mass index (BMI) above 30 kg/m^2. Patients who are severely obese (35 < BMI < 40) or morbidly obese (BMI > 40) must be singled out. Although it would be preferable if BMI differentiated between fat mass and muscle mass, it is a clearly defined risk factor that can be explained by a chronic pro-inflammatory state related to adipose tissue degradation products: adipokines [8].

The increased infection risk in this population is also related to a metabolic syndrome that increases the prevalence of diabetes and cardiovascular diseases (increasing the use of anticoagulants and/or frequency of associated vascular diseases), malnourishment or even prophylactic antibiotics that are not adjusted to body weight.

Increased operative time also increases the infection risk: each 1 kg/m^2 increase in BMI increases the operative time by about 1 minute [9].

During a knee or hip arthroplasty, the infection rate is nearly 5% in obese patients and 10% in diabetic obese patients [10]. In cases of severe obesity (BMI > 35), the infection risk is 6.7 times greater for TKA and 4.2 times more for THA [11].

The American Association of Hip and Knee Surgeons recommends that arthroplasty be delayed in cases of morbid obesity (BMI > 40), especially in patients with associated comorbidities [12]. The procedure must be delayed until significant weight loss is achieved because moderate weight loss (5%) is not enough to reduce the infection risk [13]. In fact, patients generally do not lose weight after arthroplasty [14], which is an argument in favor of significant preoperative weight loss.

3.2. Diabetes

The mechanism by which diabetic patients are vulnerable to infection is not well understood, but it is very likely related to dysfunctional natural killer cells, which are responsible for infection control [15].

Patients with unrecognized diabetes or hyperglycemia induced by the hospitalization are also exposed to a larger number of postoperative complications. The stress of surgery and anesthesia are responsible for an antagonistic endocrine response to insulin and predispose patients to hyperglycemia [16]. Diabetics have a four times higher infection risk after arthroplasty, particularly if the diabetes is not well controlled [17].

During arthroplasty procedures in diabetics, the American Diabetes Association recommends stabilizing blood glucose parameters (HbA1c < 7.0%, fasting blood sugar between 90 and 130 mg/dL and postprandial blood sugar < 180 mg/dL), scheduling these patients early in the day, delaying the procedure if the glycemic balance is not satisfactory and treating known comorbidities or those discovered during the preoperative assessment [16].

3.3. Rheumatoid arthritis and its treatments

The vulnerability to infection of patients suffering from rheumatoid arthritis (RA) is due to the disease itself and its treatments. The postoperative infection rate is 3.7% [18], with the infection risk likely increasing as the disease becomes more chronic, if it is not controlled or if it is being treated with biologics [19].

Perioperative management of the medical treatments for RA must be evaluated in partnership with the rheumatologist and anesthesiologist.

Corticosteroids increase the infection risk in a dose-dependent manner. While a low dose of prednisone (less than 10 mg/day) moderately increases the infection risk [20], a dose above 10 mg/day increases by four to seven times. Thus it seems reasonable to wait for the effective corticosteroid dose to be as low as possible, and preferably under 10 mg/day [21], or even 5 mg, before contemplating performing arthroplasty.

Methotrexate does not increase the infection risk and does not need to be stopped perioperatively. There is limited data about leflunomide (ARAVA®), hydroxychloroquine (PLAQUENIL®), sulfasalazine (SALAZOPYRIN®) and azathioprine (IMUREL®) [22].

Treatments targeting tumor necrosis factor-alpha (anti-TNF drugs) have revolutionized the treatment of autoimmune and auto-inflammatory diseases. The French health authority (HAS) [23] and the Club rhumatisme inflammation (www.cri-net.com) have published recommendations on the management of TNF inhibitors. Given their respective half-lives, it is recommended that these treatments be stopped at least 15 days before scheduled surgery for etanercept (ENBREL®), and at least 4 weeks before for infliximab (REMICADE®), adalimumab (HUMIRA®), certolizumab (CIMZIA®) and golimumab (SIMPONI®). TNF inhibitor therapy can only be restarted once healing is complete and there is no definitive proof of infection. Particular care should be taken in cases of prosthesis revision.

3.4. Smoking

Tobacco contains more than 4000 chemical products that negatively affect bone union and healing. Each cigarette smoked is equal to 2–3 g of nicotine and 20–30 mL of carbon monoxide [24]. The consumption of tobacco alters hemostasis, inflammation and tissue oxygenation and induces hypoxia, necrosis and infection [25].

The infection risk is doubled in smokers. This risk can be reversed if the patient stops smoking and is reduced the longer the patient does not smoke: 6–8 weeks of not smoking before the