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CORR Insights®: The Otto Aufranc Award: Modifiable versus Nonmodifiable Risk Factors for Infection After Hip Arthroplasty

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Where Are We Now?

Periprosthetic joint infection (PJI) is thought to be responsible for 15% of the primary THAs undergoing revision [1]. This major complication to THA is expected to increase [1, 18]. Infected THA causes a severe burden on patients, with substantial detrimental effects on quality of life, the health care system, and society, with annual costs estimated in the billions of dollars in the United States alone [8].

The incidence of deep PJI after THA varies, but the pooled incidence has been reported to be 0.9% and 1.1% in two systematic reviews [9, 18]. The incidence of PJI is much higher after revision THA (7%–12%) [17]. An increase in followup time results in a reported increase in prevalence: from 0.2% to 1.1% between discharge and 5 years postsurgery [18]; and from 1.6% to 2.2% between 2 and 10 years postsurgery [11].

Different clinical and laboratory criteria are used to diagnose PJI, with various sensitivity and specificity [5]. Some studies use clinical criteria alone [10], while others use both clinical and

laboratory criteria [17]. Diagnosis of PJI can be particularly difficult for low-grade infections that may present with nonspecific clinical signs and normal inflammatory markers. Prosthesis failure within 2 years of implantation has been shown to be highly predictive of infection even when the diagnosis is not obvious [15]. Recently, sonication fluid cultures were shown to be most accurate and sensitive in identifying the microorganism responsible of PJI [6]. This method was found particularly useful in detecting late infections [16].

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Where Do We Need To Go?

The rate of PJI after joint replacement has decreased from 9% in the 1960s to 1% to 2% in the 1990s [17]. Every effort needs to be made to continue the trend of decreasing the prevalence of PJI after THA. One important way to achieve this is to minimize, and where possible, eliminate, modifiable risk factors [7]. The study by Maoz et al. [10] investigated modifiable risk factors for infection after primary and revision

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THA in more than 4000 patients performed at the authors' institution during a 3-year period. The study used the New York State Hospital Infection Control database to ensure identification of PJI that were diagnosed using the criteria of the Centers for Disease Control and Prevention's (CDC) National Healthcare Safety Network (occurs within 30–90 days postoperatively, involves deep soft tissues of the incision, purulent drainage, dehiscence and fever, localised pain and tenderness). The authors recognize several limitations to their study including its retrospective nature and the fact that incidence of PJI reported does not include infected cases that were treated in states other than New York. In addition to the limitations acknowledged, the followup was short and the criteria used for diagnosing PJI might have omitted subtle infections. Despite its limitations, the study had, importantly, identified operating time, elevated BMI, tobacco use, and *Staphylococcus aureus* colonization as potentially modifiable risk factors of PJI after THA. If modified, these risk factors could potentially decrease PJI after THA.

How Do We Get There?

As previously acknowledged, the diagnosis of PJI can be challenging [3, 12]. I

would recommend a uniform, structured approach, such as the clinical practice guidelines established by the American Academy of Orthopaedic Surgeons (AAOS) [4], and updated by the International Consensus Meeting on PJI [13]. The criteria defined by the AAOS will need to be updated in time as new diagnostic techniques prove themselves for difficult and ambiguous cases. Potentially promising avenues include research on infection biomarkers [3] and implant sonication [6, 16].

The effect of altering modifiable risk factors for PJI after THA, like those identified by Maoz et al. [10], need to be tested and confirmed such that clinical recommendations and guidelines can be established, justified, and applied as a result of the current best evidence.

The need and difficulties to establish effective ways to prevent and treat PJI are well recognized and perhaps best illustrated by the efforts to develop a process for an international consensus on PJI [2]. The fact that 400 delegates representing more than 50 countries and 100 societies have reached a consensus on practices that lack high level of evidence [14] is an outstanding achievement and gives hope of good progress to come. The international consensus now needs to be put into practice.

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