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Commentary

Antibiotic Prophylaxis for Prosthetic Joint Patients Undergoing Invasive Dental Procedures: Time for a Rethink?

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ABSTRACT

Background: In the United States, it has been common practice to recommend that dentists provide antibiotic prophylaxis (AP) before invasive dental procedures (IDPs) to prevent late periprosthetic joint infections (LPJIs) in patients who have prosthetic arthroplasties despite lack of evidence for a causal relationship between IDP and LPJI and a lack of evidence for AP efficacy.
Methods: A recent study quantified the IDP incidence over the 15-month period prior to LPJI hospital admissions in the United Kingdom for which dental records were available. A case-crossover analysis compared IDP incidence in the 3 months before LPJI admission with the preceding 12 months. The English population was used because guidelines do not recommend AP and any relationship between IDPs and LPJI should be fully exposed.
Results: No significant positive association was identified between IDPs and LPJI. Indeed, the incidence of IDPs was lower in the 3 months before LPJI hospital admission than that in the preceding 12 months.
Conclusion: In the absence of a significant positive association between IDPs and LPJI, there is no rationale to administer AP before IDPs in patients with prosthetic joints, particularly given the cost and inconvenience of AP, the risk of adverse drug reactions, and the potential for unnecessary AP use that promotes antibiotic resistance. These results should reassure orthopedic surgeons and their patients that dental care of patients who have prosthetic joints should focus on maintaining good oral hygiene rather than on recommending AP for IDPs. Moreover, it should also reassure those in other countries where AP is not recommended that such guidance is sufficient.

Replacing arthritic joints with prostheses is one of the great advances of modern medicine with 2.9 million joint arthroplasties performed annually worldwide [1,2]. Successful joint arthroplasties improve quality of life and provide pain relief, mobility, and independence for patients. There are already greater than 7 million people with prosthetic arthroplasties in the United States [3,4], and this number is increasing rapidly with approximately 4 million new hip and knee arthroplasties projected annually by 2030 [5].

Although a vast majority of joint arthroplasties are successful, periprosthetic joint infections (PJIs) remain one of the leading causes of arthroplasty failure. Early infections, defined as occurring within 3 months of joint arthroplasty, are likely due to wound contamination at the time of surgery. Early-infection rates in the 1950s were approximately 12%; since then, perioperative antibiotic prophylaxis (AP) administered before joint arthroplasty and laminar airflow operating rooms have reduced this to around 1%-2% [4,6–8] and refocused attention on late PJIs (LPJIs), which occur greater than 3 months after joint arthroplasty surgery. Although relatively uncommon, LPJIs are most likely due to hematogenous spread of infection from a distant site.

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The economic, societal, and personal costs of PJI are substantial. The cost of treating PJIs is 4 to 6 times that of the original arthroplasty [9–12] and was projected to reach $1.62 billion annually in the United States by 2020 [13] without accounting for personal and societal costs of long-term disability and impact on the patient quality of life [14]. PJI is, therefore, of major concern for the 28,000 orthopedic surgeons in the United States and the greater than 7 million individuals who have prosthetic arthroplasties [3,4]. Following the successful reduction in early PJI rates, there was a resultant desire to identify ways of reducing LPJI, particularly those due to hematogenous spread of infection from other anatomic sites. Not surprisingly, orthopedic surgeons recognized the efforts of the American Heart Association to reduce the risk of infective endocarditis (IE) following invasive dental procedures (IDPs) as a paradigm that could have applicability to PJI prevention.

The use of AP to prevent IE in susceptible individuals undergoing IDPs had become well-established following a series of guidelines first published by the American Heart Association in 1955 and supported by the American Dental Association [15]. By the 1970-80s, this led orthopedic surgeons to call for dentists to give AP to patients with prosthetic joints undergoing IDPs [16–19], a practice supported by greater than 90% of US orthopedic surgeons at the time [20,21]. However, unlike IE, where 30%-40% of cases are due to hematogenous spread of oral bacteria, mainly oral viridans group streptococci (OVGS) [22–26], these bacteria account for few cases of LPJI.

Although, joint prostheses remain at infection risk throughout a patient's life, LPJI resulting from hematogenous seeding of bacteria from a remote site is rare. In the largest study that examined this scenario, a cohort of 6101 patients who underwent arthroplasty (4002 hip arthroplasties and 2099 knee arthroplasties) were followed for a mean period of 70 months [27]. During this time, 553 had distant infections, mainly cystitis episodes, pneumonia, skin and soft tissue infections, gastrointestinal infections, and so on, and there were also 3 dental abscesses. Although there were 71 PJIs in the cohort (incidence 71 of 6101 = 1.16%), only 7 (0.01%) of these were secondary to a remote infection, and none of these were dental in origin [27]. Therefore, the risk of hematogenous spread of infection from a distant site to a prosthetic joint was low and may have been responsible for only approximately 10% of all PJIs (7/71). Moreover, dental-related "seeding" appears uncommon.

Microbiological studies also suggest OVGS are an uncommon cause of LPJI. An analysis of 14 large studies of PJI microbiology, including >2400 patients who had hip or knee arthroplasty infections, found that 54% of all PJIs were attributable to *Staphylococci*, but only 8% to *Streptococci*, with other causes including *Enterococci* (3%), aerobic gram-negative bacteria (9%), anaerobes (4%), other (3%), polymicrobial infection (15%), and culture negative (14%) [27]. Despite accounting for less than 10% of PJIs, *Streptococcus* is a diverse genus with only a few species included as OVGS, and few studies have examined streptococcal species in sufficient detail to quantify the prevalence of OVGS. Two investigations with the largest cohorts published to date identified only 3% of 339 and 4.9% of 281 PJI cases due to OVGS [28,29].

Overall, recognizing that there are so few cases of PJI due to OVGS, any benefit of AP in preventing LPJI following IDPs is likely to be extremely limited. For this reason, many countries no longer recommend AP coverage of IDPs for those patients who have prosthetic arthroplasties, including Australia, Brazil, Canada, Denmark, France, the Netherlands, Norway, Portugal, and the United Kingdom including England, Scotland, Wales, and Northern Ireland [30].

For AP to be effective, a positive causal association must exist between IDPs and LPJI, and currently, supporting data are lacking [31]. Moreover, only five studies have previously evaluated whether such an association exists. In 1977, Waldman et al [32] performed a retrospective case review of 62 patients with late periprosthetic knee joint infection and identified 7 (11%) of them with a temporally associated IDP. In a related study, LaPorte et al [33] temporally associated 3 of 52 (6%) late periprosthetic hip joint infections with IDPs. However, neither study included a control group, making it impossible to draw conclusions regarding a possible association between IDPs and LPJI. In contrast, a case-control study by Kaandorp et al [34] reported that none of the 37 LPJI cases had undergone an IDP in the previous 3 months, but 10% of controls had. In a similar study of 42 Medicare patients with LPJI by Skaar et al [35], only 4 (9.5%) had undergone an IDP in the previous 3 months as compared with 15.9% of controls. However, differences were not statistically significant in either study. In the largest study, Berbari et al [28] found that 48% of 303 patients with PJI had undergone an IDP in the previous 2 years compared with 34% of 318 controls, but a high proportion had received AP. A subanalysis of those who had not received AP, however, identified 33 (11%) patients with PJI who had an IDP in the previous 2 years compared with 49 (14%) controls. None of the differences were statistically significant, and each study had a small sample size with a resultant lack of statistical power. The case-control studies also suffered from selection bias and risk factors confounding between cases and controls. Furthermore, there was confounding owing to the widespread use of AP in the populations studied. In addition, recall bias for IDPs was a limitation in some studies.

However, a recent study by our group has produced more conclusive evidence regarding the possible relationship between IDPs and subsequent LPJI [36]. This study included all 9427 LPJI hospital admissions in the United Kingdom between December 25th, 2011 and March 31st, 2017, for whom dental records were available. This cohort is more than 30 times larger than that in any previous study, and calculations showed that it had more than sufficient statistical power to detect any clinically significant association between IDPs and LPJI. Furthermore, confounders caused by AP use in previously investigated populations were avoided by using the English population, where use of AP to prevent LPJI has never been advocated [30]. Thus, any association between IDPs and LPJI should have been fully exposed. Recall bias was eliminated by inclusion of health records of all events and their timing. Additionally, a major advantage of the case-crossover design used in this study was the avoidance of selection bias since each individual served as their own control, and it also implicitly accounted for potential confounders (eg, differences in oral hygiene, comorbidities, age, gender, etc.) [37,38]. The study showed that there was no association between IDPs and subsequent LPJI. Indeed, there was a lower incidence of IDPs in the three months prior to LPJI (incidence rate ratio = 0.89, 95% confidence interval (CI) = 0.82-0.96, $P = .002$) than in the preceding 12 months [36]. Furthermore, a sensitivity analysis showed that when the exposure window for IDPs was extended to 4 or 5 months before LPJI hospital admission, there was still no significant association between IDPs and subsequent LPJI [36].

If there is no significant association between IDPs and subsequent LPJI, then how do we account for the very small proportion of PJI due to OVGS? The reality is that oral bacteria do not only enter the vascular circulation during IDPs, but also do so during common daily activities such as tooth brushing, flossing, and other oral hygiene procedures [39–41]. This may also occur during mastication, particularly if there is tooth mobility [40,42]. However, the frequency with which bacteremia occurs is influenced by an individual's oral hygiene status and periodontal health [29,40,43]. Those patients who have good oral hygiene and little or no gingival inflammation are less likely to experience bacteremia following daily activities than those who have poor oral hygiene. The

frequency of such bacteremia, particularly in those who have poor oral hygiene, is likely to pose a far more important overall risk for OVGS PJI than an occasional dental office procedure [28,41,44]. However, it is neither practical nor sensible to attempt to cover frequent daily events with AP—even in those patients who have poor oral hygiene. It does, however, seem reasonable to improve oral hygiene and eradicate disease around the teeth in all patients who have prosthetic joints to reduce episodes of OVGS bacteremia [29,40]. Indeed, the Berbari study found that patients with more than one dental hygiene visit were 30% less likely to develop a prosthetic hip or knee infection, although the study was not sufficiently large for this difference to be statistically significant [28].

It can be argued that just as obesity, diabetes mellitus, immunosuppression, and rheumatoid arthritis are considered risk factors associated with PJI, poor oral hygiene should also be considered as a risk factor [4].

In the absence of a positive association between IDPs and subsequent LPJI, there is no rationale for providing AP in those with prosthetic arthroplasties undergoing IDPs for LPJI prevention. This conclusion is also supported by the only study to evaluate AP efficacy in preventing LPJI, which demonstrated that AP had no effect in reducing the risk of subsequently developing total hip or knee infection (adjusted odds ratio, 0.9, 95% CI = 0.5-1.6, p = NS) [28].

The "downside" of administering AP before dental procedures for patients who have prosthetic arthroplasties must also be considered. AP is a major cost burden on patients and health care systems. The annual cost of providing AP in the United States is approximately $59,640,000 [3]. There is also a risk of adverse drug reactions due to AP [45,46]. Although amoxicillin AP is relatively safe in those who do not have a history of penicillin allergy, around 10% of the population report being allergic to penicillin [47]. Moreover, clindamycin, the antibiotic most frequently recommended as an AP alternative for those who have a history of penicillin allergy, has a much worse safety record, with 13 fatal and 149 nonfatal adverse reactions per million AP prescriptions—mainly due to *Clostridioides difficile* (previously known as *Clostridium difficile*) infections [45,46]. There is also widespread concern that unnecessary use of antibiotics for AP purposes leads to the development of antibiotic resistance among bacteria with the resultant loss of effectiveness of these agents [48,49].

It could be argued that all the focus on recommending AP for dental procedures to prevent OVGS PJI is detracting from other measures that are far more likely to be effective in reducing the risk of PJI, for example, improving oral hygiene and taking other actions to prevent the vast majority of LPJI caused by a panoply of other organisms [4]. In particular, *Staphylococci* account for more than half of all LPJIs and are common skin and nasal commensals [4,36]. Indeed, coagulase-negative *Staphylococci* are the predominant causes of PJI and are inherently able to adhere to prosthetic joint surfaces with subsequent biofilm formation. Other indwelling prosthetic devices, vascular catheters, percutaneous procedures, hemodialysis procedures, skin ulcers, injection drug usage, and so on are all associated with an increased risk of staphylococcal bacteremia [4,50–53].

Non-OVGS *Streptococci* are frequently associated with genitourinary tract, gastrointestinal tract, and skin colonization and have been associated with PJIs following gastrointestinal endoscopy [54,55], colorectal neoplasia [4], cystoscopy [55], cellulitis [56], urinary tract infection, and so on [57]. One study evaluating PJI risk following esophagogastroduodenoscopy found it was increased, particularly after esophagogastroduodenoscopy with biopsy (adjusted odds ratio = 4, 95% CI = 1.5-10), and the most common pathogens were *Staphylococci*, followed by gut-related *Streptococci*, *Enterococci*, gram-negative bacteria, and anaerobes [54].

Conclusions

These data suggest there is no rationale for patients who have prosthetic joints to receive AP before IDPs. Indeed, the risk of adverse drug reactions and contributions to the development of antibiotic resistance suggest that continuing this practice is likely to be harmful to individual patients and to society, in general. Thus, orthopedic surgeons in many countries have accepted that AP should not be recommended for prosthetic joint patients undergoing IDPs. Moreover, there is no evidence that the incidence of LPJI is any higher in the countries where AP is not advocated.

Therefore, it is time to consider recommending against the use of AP before IDPs to prevent LPJI in the United States and instead to focus on the importance of eradicating dental-related disease and establishing good oral hygiene in patients who have prosthetic joints. This is something that dentists and orthopedic surgeons should strongly support to benefit their patients.

References

[1] Colonna PC. An arthroplastic operation for congenital dislocation of the hip. Surg Gynecol Obstet 1936;63:777–81.
[2] Orthoworld. Orthopaedic industry annual report - focus on joint replacement. Chagrin Falls, OH: OrthoKnow; 2012.
[3] Little JW, Jacobson JJ, Lockhart PB, American Academy of Oral M. The dental treatment of patients with joint replacements: a position paper from the American Academy of Oral Medicine. J Am Dent Assoc 2010;141:667–71.
[4] Tande AJ, Patel R. Prosthetic joint infection. Clin Microbiol Rev 2014;27:302–5.
[5] Kurtz SM, Ong KL, Schmier J, Mowat F, Saleh K, Dybvik E, et al. Future clinical and economic impact of revision total hip and knee arthroplasty. J Bone Joint Surg Am 2007;89(Suppl 3):144–51.
[6] Kurtz SM, Lau E, Watson H, Schmier JK, Parvizi J. Economic burden of periprosthetic joint infection in the United States. J Arthroplasty 2012;27(8 Suppl):61–5.
[7] Kurtz SM, Ong KL, Lau E, Bozic KJ, Berry D, Parvizi J. Prosthetic joint infection risk after TKA in the Medicare population. Clin Orthop Relat Res 2010;468:52–6.
[8] Pulido L, Ghanem E, Joshi A, Purtill JJ, Parvizi J. Periprosthetic joint infection: the incidence, timing, and predisposing factors. Clin Orthop Relat Res 2008;466:1710–5.
[9] Bengtson S. Prosthetic osteomyelitis with special reference to the knee: risks, treatment and costs. Ann Med 1993;25:523–9.
[10] Klouche S, Sariali E, Mamoudy P. Total hip arthroplasty revision due to infection: a cost analysis approach. Orthop Traumatol Surg Res 2010;96:124–32.
[11] Peel TN, Cheng AC, Lorenzo YP, Kong DC, Buising KL, Choong PF. Factors influencing the cost of prosthetic joint infection treatment. J Hosp Infect 2013;85:213–9.
[12] Sculco TP. The economic impact of infected joint arthroplasty. Orthopedics 1995;18:871–3.
[13] Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. J Bone Joint Surg Am 2007;89:780–5.
[14] Cahill JL, Shadbolt B, Scarvell JM, Smith PN. Quality of life after infection in total joint replacement. J Orthop Surg (Hong Kong) 2008;16:58–65.
[15] Jones TD, Baumgartner L, Bellows MT, Breese BB, Kuttner AG, McCarty M, et al. Prevention of rheumatic fever and bacterial endocarditis through control of streptococcal infections. Circulation 1955;11:317–20.
[16] Ainscow DA, Denham RA. The risk of haematogenous infection in total joint replacements. J Bone Joint Surg Br 1984;66:580–2.
[17] Lattimer GL, Keblish PA, Dickson Jr TB, Vernick CG, Finnegan WJ. Hematogenous infection in total joint replacement. Recommendations for prophylactic antibiotics. JAMA 1979;242:2213–4.
[18] Norden CW. Prevention of bone and joint infections. Am J Med 1985;78:229–32.
[19] Pollard JP, Hughes SP, Scott JE, Evans MJ, Benson MK. Antibiotic prophylaxis in total hip replacement. Br Med J 1979;1:707–9.
[20] Howell RM, Green JG. Prophylactic antibiotic coverage in dentistry: a survey of need for prosthetic joints. Gen Dent 1985;33:320–3.
[21] Jaspers MT, Little JW. Prophylactic antibiotic coverage in patients with total arthroplasty: current practice. J Am Dent Assoc 1985;111:943–8.
[22] Griffin MR, Wilson WR, Edwards WD, O'Fallon WM, Kurland LT. Infective endocarditis. Olmsted County, Minnesota, 1950 through 1981. JAMA 1985;254:1199–202.
[23] Lacassin F, Hoen B, Leport C, Selton-Suty C, Delahaye F, Goulet V, et al. Procedures associated with infective endocarditis in adults. A case control study. Eur Heart J 1995;16:1968–74.

[24] Mylonakis E, Calderwood SB. Infective endocarditis in adults. N Engl J Med 2001;345:1318–30.
[25] Strom BL, Abrutyn E, Berlin JA, Kinman JL, Feldman RS, Stolley PD, et al. Dental and cardiac risk factors for infective endocarditis. A population-based, case-control study. Ann Intern Med 1998;129:761–9.
[26] Tleyjeh IM, Steckelberg JM, Murad HS, Anavekar NS, Ghomrawi HM, Mirzoyev Z, et al. Temporal trends in infective endocarditis: a population-based study in Olmsted County, Minnesota. JAMA 2005;293:3022–8.
[27] Uckay I, Lubbeke A, Emonet S, Tovmirzaeva L, Stern R, Ferry T, et al. Low incidence of haematogenous seeding to total hip and knee prostheses in patients with remote infections. J Infect 2009;59:337–45.
[28] Berbari EF, Osmon DR, Carr A, Hanssen AD, Baddour LM, Greene D, et al. Dental procedures as risk factors for prosthetic hip or knee infection: a hospital-based prospective case-control study. Clin Infect Dis 2010;50:8–16.
[29] Pallasch TJ, Slots J. Antibiotic prophylaxis and the medically compromised patient. Periodontol 2000 1996;10:107–38.
[30] Simmons NA, Ball AP, Cawson RA, Eykyn SJ, Hughes SP, McGowan DA, et al. Case against antibiotic prophylaxis for dental treatment of patients with joint prostheses. Lancet 1992;339:301.
[31] Lockhart PB, Loven B, Brennan MT, Fox PC. The evidence base for the efficacy of antibiotic prophylaxis in dental practice. J Am Dent Assoc 2007;138:458–74.
[32] Waldman BJ, Mont MA, Hungerford DS. Total knee arthroplasty infections associated with dental procedures. Clin Orthop Relat Res 1997;(343):164–72.
[33] LaPorte DM, Waldman BJ, Mont MA, Hungerford DS. Infections associated with dental procedures in total hip arthroplasty. J Bone Joint Surg Br 1999;81:56–9.
[34] Kaandorp CJ, Van Schaardenburg D, Krijnen P, Habbema JD, van de Laar MA. Risk factors for septic arthritis in patients with joint disease. A prospective study. Arthritis Rheum 1995;38:1819–25.
[35] Skaar DD, O'Connor H, Hodges JS, Michalowicz BS. Dental procedures and subsequent prosthetic joint infections: findings from the Medicare Current Beneficiary Survey. J Am Dent Assoc 2011;142:1343–51.
[36] Thornhill MH, Crum A, Rex S, Stone T, Campbell R, Bradburn M, et al. Analysis of prosthetic joint infections following invasive dental procedures in England. JAMA Netw Open 2022;5:e2142987.
[37] Maclure M. The case-crossover design: a method for studying transient effects on the risk of acute events. Am J Epidemiol 1991;133:144–53.
[38] Maclure M, Mittleman MA. Should we use a case-crossover design? Annu Rev Public Health 2000;21:193–221.
[39] Lockhart PB, Brennan MT, Sasser HC, Fox PC, Paster BJ, Bahrani-Mougeot FK. Bacteremia associated with toothbrushing and dental extraction. Circulation 2008;117:3118–25.
[40] Lockhart PB, Brennan MT, Thornhill M, Michalowicz BS, Noll J, Bahrani-Mougeot FK, et al. Poor oral hygiene as a risk factor for infective endocarditis-related bacteremia. J Am Dent Assoc 2009;140:1238–44.
[41] Roberts GJ. Dentists are innocent! "Everyday" bacteremia is the real culprit: a review and assessment of the evidence that dental surgical procedures are a principal cause of bacterial endocarditis in children. Pediatr Cardiol 1999;20:317–25.
[42] Fine DH, Furgang D, McKiernan M, Tereski-Bischio D, Ricci-Nittel D, Zhang P, et al. An investigation of the effect of an essential oil mouthrinse on induced bacteraemia: a pilot study. J Clin Periodontol 2010;37:840–7.
[43] Tomas I, Diz P, Tobias A, Scully C, Donos N. Periodontal health status and bacteraemia from daily oral activities: systematic review/meta-analysis. J Clin Periodontol 2012;39:213–28.
[44] Guntheroth WG. How important are dental procedures as a cause of infective endocarditis? Am J Cardiol 1984;54:797–801.
[45] Thornhill MH, Dayer MJ, Durkin MJ, Lockhart PB, Baddour LM. Risk of adverse reactions to oral antibiotics prescribed by dentists. J Dent Res 2019;98:1081–7.
[46] Thornhill MH, Dayer MJ, Prendergast B, Baddour LM, Jones S, Lockhart PB. Incidence and nature of adverse reactions to antibiotics used as endocarditis prophylaxis. J Antimicrob Chemother 2015;70:2382–8.
[47] Drug, Therapeutics B. Penicillin allergy-getting the label right. BMJ 2017;358:j3402.
[48] American Dental Association Council on Scientific A. Combating antibiotic resistance. J Am Dent Assoc 2004;135:484–7.
[49] Sweeney LC, Dave J, Chambers PA, Heritage J. Antibiotic resistance in general dental practice–a cause for concern? J Antimicrob Chemother 2004;53:567–76.
[50] Bergin SP, Holland TL, Fowler Jr VG, Tong SYC. Bacteremia, sepsis, and infective endocarditis associated with Staphylococcus aureus. Curr Top Microbiol Immunol 2017;409:263–96.
[51] Tong SY, Davis JS, Eichenberger E, Holland TL, Fowler Jr VG. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev 2015;28:603–61.
[52] Fey PD, Olson ME. Current concepts in biofilm formation of Staphylococcus epidermidis. Future Microbiol 2010;5:917–33.
[53] Harris LG, El-Bouri K, Johnston S, Rees E, Frommelt L, Siemssen N, et al. Rapid identification of staphylococci from prosthetic joint infections using MALDI-TOF mass-spectrometry. Int J Artif Organs 2010;33:568–74.
[54] Coelho-Prabhu N, Oxentenko AS, Osmon DR, Baron TH, Hanssen AD, Wilson WR, et al. Increased risk of prosthetic joint infection associated with esophago-gastro-duodenoscopy with biopsy. Acta Orthop 2013;84:82–6.
[55] Zeller V, Lavigne M, Leclerc P, Lhotellier L, Graff W, Ziza JM, et al. Group B streptococcal prosthetic joint infections: a retrospective study of 30 cases. Presse Med 2009;38:1577–84.
[56] Everts RJ, Chambers ST, Murdoch DR, Rothwell AG, McKie J. Successful antimicrobial therapy and implant retention for streptococcal infection of prosthetic joints. ANZ J Surg 2004;74:210–4.
[57] Zeller V, Kerroumi Y, Meyssonnier V, Heym B, Metten MA, Desplaces N, et al. Analysis of postoperative and hematogenous prosthetic joint-infection microbiological patterns in a large cohort. J Infect 2018;76:328–34.